ARTIFICIAL PNEUMOTHORAX IN PULMONARY TUBERCULOSIS

WITH SPECIAL REFERENCE TO

THE PREVENTION AND TREATMENT OF PLEURAL EFFUSION

Thesis submitted to the University of Glasgow

for the degree of Doctor of Medicine by

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TO MY PARENTS

C O N T E N T S

I N T R O D U C T I O N

It is unnecessary at the present time to prove that pulmonary collapse in one or other form is an essential weapon of the physician treating tuberculosis of the lung. Nevertheless, this fact is once again emphasised.

Collapse measures, first advocated more than a century and a half ago, were rendered safe and controllable to a great extent by the introduction of aseptic surgery and by the routine use of the X-Ray photograph. Artificial pneumothorax, the most frequently used of these collapse measures, has been in general use for the past thirty years and the treatment has become increasingly valuable with the clearer recognition of the type of case in which its use is likely to be successful.

Collapse of the lung has now become a highly individualised procedure. There is no single measure which can be applied wholesale in the expectation of a high proportion of successful results.

It is recognised today that the forms of collapse formerly thought to be less dangerous, namely, phrenic interruption and especially artificial pneumothorax, can carry considerable risk for the patient in the long run. The more drastic procedures, such as thoracoplasty, lobectomy and pneumonectomy, on the other hand, must now be thought of as initial procedures in certain cases rather than as a last resort.

With the advances in thoracic surgical technique in the past few years, more and more cases which would formerly have had artificial pneumothorax are now dealt with successfully by purely surgical measures supplemented, of course, by more or less long periods of sanatorium rest. It is now recognised also that the presence of stenotic or obstructive bronchial tuberculosis is a bar to the successful use of pneumothorax. Prolonged bed rest, supplemented in many cases by pneumoperitoneum, with or without associated temporary phrenic paralysis, obviates in many cases the need for further collapse treatment. Finally, Streptomycin is now being used with success in certain types of pulmonary tuberculosis, but it remains to be seen how many cases

in which it would previously have been considered necessary to use collapse may be spared its use and also how many cases who would previously have been considered unfit for collapse treatment can now have the benefit of this procedure.

Thus, artificial pneumothorax is not now used so frequently as formerly, although it has become a safer procedure through a better recognition of those cases in which it can safely be employed.

The object of the present investigation was primarily to record the short-term results of artificial pneumothorax in a series of one hundred cases personally treated. In addition, the high incidence of pleural effusion encountered made it seem desirable to determine, if possible, the prognostic significance of such effusions, including tuberculous empyema, and the correct handling of such complications should they arise. However, it was realised that the prognostic implications could not properly be assessed without a period of observation running into many years and, accordingly, attention was focussed on the twelve cases of empyema encountered in the series. Each case was studied in an effort to ascertain whether this complication could reasonably have been expected to occur and whether it could have been prevented and, finally, what effect its occurrence had on the outlook for the patient.

The work was carried out at the Transvaal Chamber of Mines (Springkell) Sanatorium during the years 1944 to 1949 and at the Johannesburg Municipal Tuberculosis Clinic during the same period.

I wish to express my thanks to Dr. M.A. Pringle, Medical Superintendent of the Sanatorium, for permission to use the hospital records and for his encouragement and advice with my work. I thank also the Acting Medical Officer of Health, Johannesburg, for permission to quote from the records of the Municipal Tuberculosis Clinic. My thanks also go to my wife who carried out the typing of the manuscript.

CHAPTER ONE

HISTORY OF ARTIFICIAL PNEUMOTHORAX

THE HISTORY OF ARTIFICIAL PNEUMOTHORAX

The original conception of therapeutic artificial pneumothorax in the treatment of Pulmonary Tuberculosis is said to be that of Dr. E. Claude Bourru, who published a book entitled "Utilité des voyages sur mer pour la cure des différentes maladies et notamment de la consumption" in 1770 (1).

He suggested that, if it was only the movement of the affected part that prevented healing of the ulcer, this could be overcome by making an opening in the chest wall to allow ingress of air as it was known, when this was done, air rushed in and the lung collapsed and took no further part in respiration. Bourru believed that the communication between the air inside and outside the chest wall should be kept open until the ulcer healed. He thus for the first time advocated pneumothorax and seemed to realise that it should be of some duration. Apparently he did not believe that it would take long for healing to take place.

No practical application of the above-mentioned conceptions was made until James Carson of Liverpool (2, 3, 4, 5) published his work on the elasticity of the lungs and kindred subjects.

Carson had noted that pulmonary cavities were prevented from healing by the elastic retraction of the surrounding lung tissue. He, therefore, advocated the introduction of air into the pleural space to overcome this and to allow the lung to collapse.

He performed artificial pneumothorax in rabbits by incision and, following his animal experiments, he suggested that it be used in human beings in the treatment of pulmonary abscess, in severe haemoptysis, and in unilateral tuberculosis. He thereafter attempted it in two cases of consumption. An incision was made between the sixth and seventh ribs calculated to admit air freely but both cases were unsuccessful owing to the presence of adhesions. He was an advocate of complete collapse of the lung, this to be brought about by admitting successive small amounts of air to the chest. Nothing more is heard of the method until in 1834 F.H. Ramadge (6) described two cases of consumption which he claimed to have cured by producing a pneumothorax with a trocar and cannula and subsequently keeping the opening from closing for as long as seemed to him necessary.

The first was induced in 1827 on a youth of 17 with clinically unilateral disease with cavitation. Amelioration of the symptoms was noted although the patient later died. The second case (1827 - 1834) was a man of 30 with a three-year history. He had signs of a cavity at one apex and was expectorating a quantity of purulent sputum. A trocar opening was made and kept open for ten days. He was reported to be in good health two years later. The third case was in 1831 when a similar technique was employed for a case of pulmonary abscess. Recovery followed.

Unfortunately, litigation in which he was involved caused Ramadge's methods to be disparaged by his contemporaries.

The first mention of improvement in pulmonary tuberculosis following spontaneous pneumothorax was made by James Houghton (7) in 1832. He described a case of phthisis in a man of 28 who developed a spontaneous pneumothorax. Following this occurrence his condition improved so much that he was able to resume his work as a bricklayer.

William Stokes (8) in his book written in 1837 also noted this fact. Under the heading "Phthisis complicated with empyema and pneumothorax from fistula", he states that it was his experience that many cases of phthisis showed an arrest and modification of the symptoms with the occurrence of spontaneous pneumothorax. He recognised that certain cases benefited greatly, losing their cough and their toxic symptoms, and he attributed this improvement to the compression of the diseased lung.

It is to Carlo Forlanini (9), however, that the world is indebted for the introduction of artificial pneumothorax treatment in a more modern sense. In 1882 he published a paper entitled "Contribuzione della terapia chirurgica della tisi: ablazione del polmone? pneumatorace artificiale?" He first quoted many cases in which the occurrence of a spontaneous pneumothorax had exerted a favourable influence on the course of phthisis. He then pointed out that the production of an artificially produced pneumothorax was a logical procedure and likewise capable of leading to a similar result. Theoretically, he suggested a complete and sufficiently long maintained immobilisation of the lung.

In 1888 he applied his proposition for the first time to a case of unilateral phthisis with effusion which he aspirated and replaced by filtered air. This was reported in 1890.

In 1892 he induced his first case having a relatively normal pleural space, ie. without a pre-existing effusion. This case and a second one were published in 1895 and thereafter nothing was published until 1906 when he published 25 cases.

Forlanini believed that the lung had to be flattened down, using considerable pressure. Using the puncture method, he introduced large amounts of nitrogen by means of a rubber hand-bulb, sometimes as often as once daily, through a hollow needle into the pleural space. As he had no manometer, he had no means of knowing that the gas was actually entering the pleural space except by the fact that it flowed easily, nor could he estimate the pressure in the pleural space. His technique was undoubtedly dangerous and probably gave rise to a good many cases of gas embolism. However, in 1910, he did eventually adopt the water manometer. At first he treated only the mildest cases; later he widened his indications.

In England in 1882 the gas replacement of a pleural effusion was undertaken by Mr. Hicks at the Brompton Hospital for a case of Sir Richard Douglas Powell.

In this year, also, R.W. Parker (10) described an apparatus used by him to replace the fluid in an empyema cavity by filtered carbolised air. He published a paper entitled "Suggestions for the treatment of special cases of empyema by thoracentesis and the simultaneous injection of purified air".

The next advance in pneumothorax therapy was in 1884 when Potain (11) operated on a case who had developed an hydropneumothorax. He devised an apparatus for the simultaneous withdrawal of fluid and its replacement by filtered air. For the first time mention is made of the use of a manometer to measure the pressure of air. Results were published in 1888 by which time three cases had been treated by the new method. As a result of his experience, he formulated the following conclusions:-

(a) That if, at the outset, a pneumothorax gave rise to no very marked dyspnoea, the operation should not be undertaken.

(b) That if a valvular pneumothorax gave rise to symptoms owing to the rise in intra-pleural pressure to a dangerous degree, then the operation should be resorted to and the intrapleural pressure brought down to atmospheric or below this.

(c) If, at a later stage in a case of pneumothorax, a sero-purulent effusion developed, the operation should be postponed unless the size of the effusion or the weight of the fluid caused distress.

(d) If there was a considerable amount of fluid in a hydropneumothorax, Potain advised removing this and replacing it with sterilised air, the final pressure in the pleural space being at or about the normal minus seven millimeters of water.

(e) That if a purulent focus communicating with a bronchus existed or if the effusion was purulent from the outset, he recommended gas replacement, provided that the contralateral lung could be expected to suffice for breathing purposes in spite of the displacement of the mediastinum.

(f) If respiration was seriously affected, he recommended closed drainage on the syphon principle.

Potain's actual work leading to the above conclusions was carried out in 1884 and published in 1888.

Artificial pneumothorax was next applied to the treatment of haemoptysis in 1885 by Dr. W. Cayley (12) of the Middlesex Hospital, who read a paper before the Clinical Society of London describing a case. The patient was a young man admitted after a sudden haemoptysis of about a pint. Haemoptysis recurred several times while in hospital and Dr. Cayley recommended that the left lung should be collapsed (the presumed side of the bleeding) in order that "the great diminution in the circulation through the collapsed lung, together with the pressure exercised by the air, would arrest the haemorrhage and supposing there was active development of tubercle in progress in the lung, this would probably for a time be checked". An incision was made in the left sixth space and tubing inserted. Air passed in and out freely: the lung collapsed, the apex beat was displaced to the middle line and the respiratory rate increased. He had only slight haemoptysis from the time of operation until he shortly afterwards succumbed to his disease. The post-mortem showed evidence of extensive pulmonary tuberculosis and adhesions were present.

It is unfortunate that this case was one of obviously hopeless phthisis, but it was evident that the pneumothorax had had a definitely favourable effect as far as the haemoptysis was concerned.

Meanwhile in America in 1898 J.B. Murphy (13) delivered an oration to the 49th Annual Meeting of the American Medical Association on "Surgery of the Lung". He described the method of collapse of the lung by intrapleural injections of air. He put forward his reasons for advocating collapse therapy, pointing out that tuberculous pulmonary cavities are exposed to physical conditions peculiar to the chest, namely, to the resistance of the chest wall to contraction and the effort at expansion of the cavity in each inspiratory act. He believed that the keynote of successful treatment was to allow the abscess to collapse and it would heal as other abscesses did. This was a stimulus to his researches in pulmonary surgery and led him to hope that, not only might pulmonary cavities be obliterated by permitting or forcing their collapse, but that primary tuberculosis might be encapsulated by immobilisation of the diseased portion by collapse and enforced rest of the lung. In other words, tuberculosis of the lungs treated in a similar manner to that of the joints, i.e. by immobilisation and enforced rest, should respond in a similar manner.

Methods of obtaining collapse were:-

(1) By permitting or forcing the collapse of the lung by separation of the parietal pleura and intrathoracic compression (extrapleural pneumothorax).

(2) By removing the bony resistance and allowing the collapse of the chest wall, thus favouring contraction and cicatrisation (thoracoplasty).

(3) Allowing the lung to collapse by intrapleural injections of gas or fluid.

As a collapse measure Murphy favoured intrapleural injection of gas (Nitrogen), having noted that a lung compressed by fluid for long periods may still regain its function when the fluid absorbs or is removed.

His technique was to incise the skin and then push in a trocar and cannula connected with a measuring bottle, a movable pressure bottle and the nitrogen

container. The gas was turned on before the parietal pleura was pierced by the trocar. When the parietal pleura was punctured, the trocar advanced rapidly and gas began to flow freely into the cavity unless adhesions existed. The amount of gas introduced varied between 70 and 200 cub.ins. given at one operation. He advised re-injection every six to ten weeks, depending on the rate of absorption.

At the time of his report, he gave details of 8 cases. In 5 the operation was successful and a marked pneumothorax was obtained. In 3, adhesions made it unsuccessful. Only one of these cases received a second injection. In the first case described in 1898 he introduced 120 cub.ins. of Nitrogen into the right pleural cavity of a young man with right apical disease. This was in April 1898. Re-examined in July, the pneumothorax was still present and the patient was still well.

Although Murphy's work was based on sound surgical principles, the technique was to some extent dangerous as large amounts of air were given without the safeguard of manometric control. The need for prolonged collapse in the sense used today was not apparently realised.

Murphy's work was continued by A.F. Lemke (14), who published his results on 53 cases in 1899. His technique was slightly different to that of Murphy, although he still had no manometer to guide him. He pushed an aspirating needle into the fifth or sixth interspace about the anterior axillary line and, to determine when the needle was in the pleural cavity, he caused the patient to take a series of deep breaths. If the needle was correctly placed, air could be heard rushing in. The tube from the Nitrogen bottle was then attached and the gas allowed to flow. This seems to be an advance on Murphy's method. He reports a case of Hemiplegia from gas embolism and another case with a severe haemorrhage following the introduction of the needle. He noted the accidents which may occur as gas embolism, wounding of intercostal vessels, pulmonary haemorrhage from puncture of the lung and wounding the pericardium when it is distended. He gave refills occasionally, but not as a routine and his cases were not followed up for long.

This work does not seem to have attracted the attention which it deserved in America but, although it was ignored there, Prof. L. Brauer (15) of Marburg became interested and his first case was reported in 1906. In 1908 another case was published. This was a woman having a cavity in the left lung. He used Nitrogen, the patient receiving four refills from 1000 to 1700 c.c. in four months. The treatment had a favourable influence on the symptoms; temperature, cough and expectoration diminished and the general condition improved after each injection.

His technique, a great advance on anything previously done, was to anaesthetise the skin and then incise down to the parietal pleura. Through this he pushed a blunt cannula with a side opening. The lung was considered free if air could be heard entering with inspiration. An elastic catheter was then introduced to ascertain whether or not there were adhesions in the neighbourhood. If the space was free, the tube was connected with a bottle containing Nitrogen. He used a manometer, by means of which the variations in intrapleural pressures could be measured accurately. Negative or only slightly positive pressures were used and after the lapse of some time a refill was given.

Forlanini had always considered that complete collapse of the lung using high pressures of Nitrogen was a prerequisite for healing, but in 1912 Ascoli (16) suggested that a complete collapse was not always necessary. In other words, a selective collapse of the diseased areas of the lung was proposed, keeping the intrapleural pressures at sub-atmospheric. He also proposed bilateral simultaneous pneumothorax.

With the introduction of the water manometer, pneumothorax became a safe procedure and the knowledge of its value spread throughout the world.

However, it is interesting to note that Lillingstone (17) found that artificial pneumothorax treatment was practically unknown in England in 1913. Rapid strides have been made since those days.

Although knowledge of artificial pneumothorax had become world-wide by about 1911, it was many years before its use became regular in sanatoria generally. Naturally it took many years before conclusions could be drawn from cases followed up for a sufficient length of time. There were many questions to be answered as to the indications for collapse therapy and as to the handling of each case and as to how long the lung had to be kept collapsed for healing to take place.

Beggs (18), writing in 1917 on "Induced Pneumothorax in Pulmonary Tuberculosis - Its present status", stated that some regarded the procedure as a last resort and others as the only treatment likely to benefit cases of progressive phthisis. There were at that time a good many opponents of the treatment. His conclusions were:-

(a) While the operation is not a dangerous one, the practice of collapse therapy calls for special skill.

(b) Collapse therapy restores to health many hopeless cases.

(c) It decreases the period of incapacity.

(d) It should be used in all cases of recurrent, severe and protracted haemorrhage.

(e) It should be invoked earlier.

(f) It should be considered in every case of advanced or moderately advanced slowly progressive pulmonary tuberculosis and in every case of actively progressive pulmonary tuberculosis, because all tend towards death.

(g) Obliteration of the space should be combatted by frequent fills and positive pressures.

(h) The procedure should be controlled by the use of X-Rays and screening.

In his paper Beggs gives no indication of the time thought necessary to maintain the collapse but in the main his conclusions were sound.

In the same journal Minor (19) drew his

deductions from 4½ years of artificial pneumothorax in the treatment of pulmonary tuberculosis. He started using the method in 1912 and reviewed a hundred cases. He did not use it in first-stage cases otherwise favourable, but used it mostly in bad, advanced third-stage cases. He got 12% well and working and in 20% life was prolonged and symptoms ameliorated. In his 100 cases no entrance was possible in 11. In 13 cases there was no favourable influence observed and the artificial pneumothorax was abandoned. 44 cases were classed as failures, usually due to the presence of adhesions, but 7 of these were due to contralateral spread. 21 showed excellent temporary success. He regarded adhesion formation as the commonest complication. Also noted were effusion, nervousness, pain and dyspepsia from pushing down the left hemi-diaphragm.

His opinion was that: "The method is chiefly applicable to moderately advanced or advanced chronic cases - not to florid acute cases nor to incipient cases unless showing signs of becoming active".

He used small refills and gave no definite guide to the length of treatment required. He considered one year to be long enough. Kendall (20) and Morris (21) published reports and drew somewhat similar conclusions in 1918.

About this time the presence of adhesions was a bar to adequate collapse in many cases. The introduction of thoracoscopy and adhesion section by Jacobaeus (22) was a distinct advance.

By 1927 Rist (23), reviewing the recent advances in the treatment of pulmonary tuberculosis, stated that collapse therapy became safe and controllable with the advent of aseptic surgery and roentgenology. The indications were becoming broader but unilaterality and a free space were necessary. He mentioned, however, that adhesions might stretch or could be cauterised. He said: "The artificial pneumothorax has lost its early dangers".

During this developmental period, it was noted that certain complications were found in association with artificial pneumothorax treatment. Wilson (24), for example, noted a case of mediastinal rupture which appears to have been caused by giving air at too high a pressure where it was known that gross adhesions were present. From this experience emerged a plea for fluoroscopy before and after refilling.

In the same year, Cooke (25) had a case of spontaneous pneumothorax superimposed upon an artificial pneumothorax. Here again there was no fluoroscopic control and reference in the article to "compression therapy" gives the impression that dangerous pressures were being used in the presence of adhesions. Reports of a number of other such cases had been published at the time of this article.

Heise (26), in the same journal, reports a case of haemo-pneumothorax superimposed on an artificial pneumothorax. Death followed.

Two cases of mixed tuberculous empyema were described by Waters (27) and their treatment with Gentian Violet was discussed.

In a symposium in the British Journal of Tuberculosis in 1927 Clive Riviere (28) stated that the use of artificial pneumothorax treatment was even then not very widespread in England. Several contributors still spoke of compression of the lung. Others recommended low pressures. Wynn (29) deprecated the tendency to widen the basis of selection and warned that artificial pneumothorax should not be the sole means of treatment. Morriston Davies (30) said that he had seen good results from partial collapse of the lung. He advocated the avoidance of high intrapleural pressures and mentioned bi-lateral collapse as a possibility. He also recommended the abandoning of a useless artificial pneumothorax and the substitution of thoracoplasty.

Bilateral artificial pneumothorax is discussed by Tobé et al (31) who sum up "everyone agrees in recognising that simultaneous bilateral pneumothorax is the only treatment for early and serious spread in cases of artificial pneumothorax".

As a result of numerous articles published between 1920 and 1930, the indications for the use of artificial pneumothorax were gradually becoming more clearly defined.

Young (32), in 1930, suggested the following:-

(a) Extensive disease, slowly progressive, clinically unilateral, the X-Ray showing little damage in the opposite lung. Cavity cases.

(b) Haemorrhagic cases. To be induced deliberately with repeated haemoptyses or as an emergency if life is threatened by blood loss.

(c) Where partial arrest has been obtained but where symptoms continue.

(d) In basal lesions where phrenic crush alone is ineffective.

(e) Spontaneous collapse with localised disease.

(f) In acute cases only if there is improvement with rest.

As contra-indications Young suggested extensive bilateral disease, extra-pulmonary lesions, dyspnoea, emphysema and age over 60.

Burrel and McNalty (33), in their report based on the personal experience of 16 physicians in the treatment of pulmonary tuberculosis by artificial pneumothorax, conclude as to the value of such treatment "that a favourable answer is returned by all with no uncertain voice". They hoped that the publication of their report might lead to the more general adoption of this valuable method of treatment.

Fourteen years later F.J. Bentley (34), from a perusal of the world literature on artificial pneumothorax from 1922 to 1936, found varying opinions but almost unanimous enthusiasm for artificial pneumothorax. He then compared the fate of 677 artificial pneumothorax cases with that of 3,329 conservatively treated cases and attempted to assess the value of pneumothorax therapy.

Firstly, he considered it impossible to obtain a perfect statistical control and rightly deprecated the tendency of several writers to compare a group treated by artificial pneumothorax with others who, although considered suitable for artificial pneumothorax, were in fact unable to have it because of extensive adhesions, or with those who were unsuccessfully treated or abandoned early.

He pointed out that all cases having artificial pneumothorax are not necessarily undergoing collapse. Complete collapse was seen to be more efficacious than only partial collapse. Of 267 incomplete collapse cases, only 50% survived a period of three years, whereas of

208 cases who had a complete collapse, 65.9% survived three years and, where the disease was strictly unilateral, the surviving percentage totalled 77.4.

He also noted that strict unilaterality was important in prognosis and as a single factor seemed to be more important than the degree of collapse obtained. He made a plea for collapse as complete as possible.

Another conclusion was that, having satisfied one-self that collapse is valuable it is necessary in incomplete cases and especially if the sputum still contains tubercle bacilli, to consider the possibility of proceeding to other collapse measures. Adhesion section, phrenic nerve operations and plastic operations on the chest wall were mentioned.

He considered that it was unlikely that a much greater proportion of patients would be found suitable for artificial pneumothorax, even if the treatment were to be carried out more frequently, because of the poor results where there was bilateral disease (over half the total number in the series) and where the collapse was incomplete (over half the remainder). He stated finally: "Artificial pneumothorax is not a specific cure..... it is indeed of only limited application. ... The present investigation has established that artificial pneumothorax is a power for the good, but only in carefully selected cases".

Bloch et al (35), in an important article written in 1941, attempted "to establish more firmly the basis on which it (artificial pneumothorax) can be applied and by what criteria its results can be judged". There was still much controversy on the indications for air collapse even on a pathological basis alone. The mass application of collapse therapy was rightly considered regrettable (in one sanatorium 96% of all cases had collapse of some sort attempted). They also thought that the manner of reporting results had caused over-optimism. Of 2,100 reports checked by these authors, only 99 dealt with results. On investigating these 99 reports from 18 countries, they classified them as having "acceptable standards, partially adequate standards, inadequate standards, or incomplete information". Patients in the first group had, among other criteria, been re-expanded for at least two years before being reported on as cured. Of this group of nearly 6,000 cases, 39.8% were "cured" and 38.8% were dead.

The conclusions were:-

(a) That the indications for artificial pneumothorax treatment required to be more sharply delineated.

(b) That rest treatment is a necessary adjunct to artificial pneumothorax.

(c) That surgical collapse should follow unsuccessful air collapse as soon as it is evident that the artificial pneumothorax has failed to obliterate cavitation.

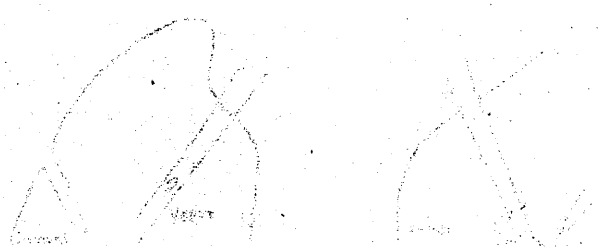
One of the most up-to-date accounts of collapse therapy with special reference to artificial pneumothorax is that of Rafferty (36). In discussing results, he summarises six artificial pneumothorax series. There were 966 cases with 105 deaths. He cites Bloch and concludes that artificial pneumothorax must be regarded as nothing more than an effective aid in the total therapy of pulmonary tuberculosis. "The most important single factor in insuring maximum results with collapse therapy is a proper choice of cases..... Many poor results in the past are due to the use of artificial pneumothorax in cases in which it is not suitable".

The advance in the treatment of pulmonary tuberculosis since the days of Forlanini has indeed been remarkable. The very numerous reports published since 1920 or so have served to emphasise that artificial pneumothorax is a procedure fraught with considerable danger. The advances in thoracic surgery, however, have made it possible to obviate many of these dangers by a careful choice of the collapse procedure to be employed in any given case, while the researches at present being pursued in connection with the use of streptomycin make it probable that collapse therapy will in future be applied to certain cases now considered unsuitable for it.

CHAPTER TWO

ANATOMICAL CONSIDERATIONS

PULMONARY PHYSIOLOGY



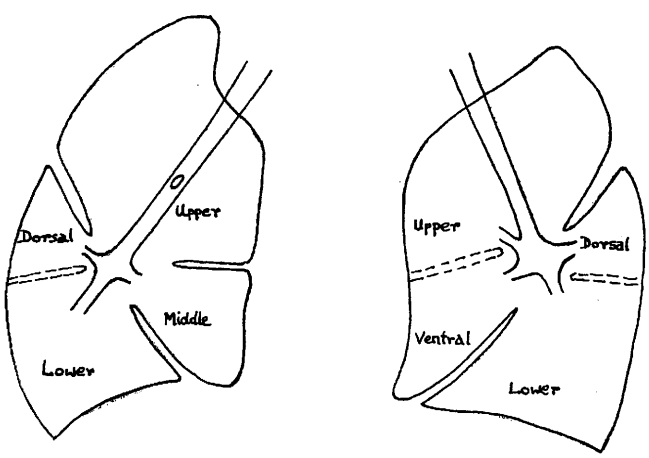
ANATOMICAL CONSIDERATIONS

The lungs are the essential organs of respiration. Irregularly conical in shape, they occupy the thoracic cavity, separated from each other by the mediastinal structures. The apices extend 3 - 4 cm. above the medial ends of the first ribs. The bases are concave, the concavity being greater on the right because of the position of the domelike right lobe of the liver (37).

The substance of the lung consists of innumerable lobules connected by areolar tissue rich in elastic fibres. Each lobule consists of the lobular bronchiole and its terminal air spaces, the pulmonary and bronchial vessels, lymphatics and nerves. This is the fundamental lung unit of Coope (38).

Lobes: As usually described, the left lung is divided into an upper and a lower lobe by the oblique fissure and the right lung is divided into upper, middle and lower lobes by the oblique and transverse fissures.

As pointed out by Nelson (39), however, the internal structures are similar on both sides and symmetrical. Therefore, one can describe the two lungs in terms of four principal areas, namely, upper, middle (ventral), dorsal and lower. The dorsal area on both sides is usually incorporated in the lower lobe but may be separated by a fissure and is anatomically and clinically independent of the rest of the lower lobe.



RIGHT LEFT

Trachea and Bronchi: The trachea is a membranous and cartilaginous tube continued downwards from the larynx as far as the upper border of the 5th thoracic vertebra where it divides into right and left main bronchi. The right main bronchus is shorter, wider and more vertical than the left. It gives off the eparterial bronchus to the upper lobe above the right pulmonary artery and then divides into branches for the middle and lower lobes. The left bronchus has no eparterial branch.

The trachea and extra-pulmonary bronchi consist of a framework of cartilaginous rings, incomplete posteriorly and joined together by fibrous and unstriped muscle tissue. The mucous membrane is made up of columnar ciliated epithelium on areolar and lymphoid tissue and this lies superficially to a layer of submucous tissue containing blood-vessels, nerves and mucous glands. The nerves are derived from the vagi, the recurrent laryngeals and the sympathetic trunks.

Intra-pulmonary Bronchi: These divide and subdivide, the smallest branches constituting the respiratory bronchioles. The larger branches consist typically of fibrous tissue enclosing hyaline cartilages, at first in the form of rings, as in the trachea, and, as the bronchi, diminish in diameter, in the form of irregular plates. When the diameter of the bronchi falls below 0.4 to 0.7 mm., the cartilage disappears from their walls (40).

Within this is the smooth muscle, arranged in a geodesic network (see diagram) and extending as far down as the atria. Inside this layer is the mucous membrane which also contains abundant elastic tissue. The cells are ciliated columnar epithelium, but in the smaller bronchioles and before the respiratory bronchioles are reached, this changes to cuboidal ciliated epithelium. This is then replaced by cuboidal cells without cilia in the respiratory bronchioles themselves. Thereafter, as the alveolar ducts are reached, these cuboidal cells are replaced by simple squamous epithelial cells.

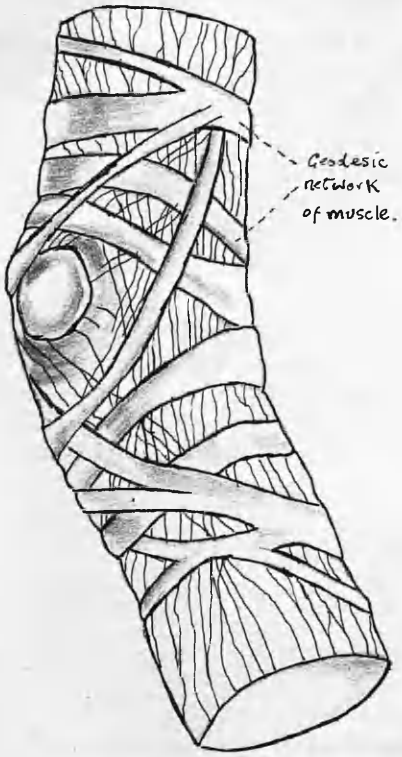


Diagram to show the myo-elastica (after Snow Miller).

Note: The muscle is arranged in a geodesic network.

The elastic fibres are mainly in a longitudinal direction.

At the exit of the branch bronchus, the myo-elastica would produce a sphincter-like effect by its contraction.

The respiratory bronchiole, so-called because of the alveoli which stud its walls, leads finally into the alveolar duct. From the distal end of this lead off several atria and from each of these several alveolar sacs from the walls of which project the alveoli or air cells. A network of abundant elastic tissue fibres encloses the atria, alveolar sacs and alveoli as the illustrations of Miller so beautifully show.

Alveolar Epithelium: A layer of thin, flattened squames closely applied to the alveolar wall has been described but doubt exists as to whether this appearance is not in fact an accompaniment of inflammation.

There is controversy as to whether pores of any kind exist in the alveolar walls which might allow communication between adjacent alveoli. If no communication existed, then blocking of a respiratory bronchiole with secretion might frequently lead to absorption of the air distally to the obstruction and a condition of atelectasis. The work of Adams and Livingstone (41) and van Allen and Lindskog (42) on obstructive atelectasis showed that block of lobar bronchi led to absorption of air in the lobe supplied and resulting

atelectasis. This did not occur when lobular bronchi were obstructed, indicating that air could still reach alveoli when their supplying bronchioles were cut off by way of adjacent alveoli. In other words, a collateral respiration exists between neighbouring lobules but not from lobe to lobe. This also presupposes a healthy state of the pulmonary epithelium.

Blood Supply: This is mainly from the pulmonary artery, the branches of which closely follow the divisions of the bronchi. Ultimately, a plexus of capillary vessels results covering the alveolar ducts and further air spaces. The bronchial arteries, branches of the aorta, supply the bronchi as far as the respiratory bronchioles. Most of the venous return from both pulmonary and bronchial arteries is, however, by way of the pulmonary veins.

Lymphatics: The lymphatics of the lung consist of a superficial set in the pleura and a deep set accompanying the bronchi and pulmonary vessels and also forming a network in the connective tissue septa.

Miller (40) states that there is only a single plexus in the pleura draining into the hilar lymph nodes. Connections exist with the deeper lymphatics of the lung along a narrow zone 2 - 3 mm. in depth just beneath the pleura. The valves in these connecting channels, however, all point outwards and drainage takes place towards the pleura and not in the reverse direction. He also states that no lymphatics are present in the walls of the atria or of the alveolar sacs.

Nerves: The anterior and posterior pulmonary plexuses situated respectively in front of and behind the lung roots, are made up of nerve fibres from the vagi and the second, third and fourth thoracic sympathetic ganglia. From these, two further plexuses arise, one accompanying the bronchi and the other the branches of the pulmonary artery. Efferent fibres run to the bronchial walls and afferents from the mucous membrane and the alveoli.

The Pleura: Each lung is invested by a serous membrane. Part of this covers the lung and lines the inter-lobar fissures. This is the visceral pleura. The remainder lines the hemi-thorax and covers most of the diaphragm and mediastinal structures. The layers are continuous around and below the lung root. The right and left pleural sacs are separate, coming into contact behind the upper half of the sternum and behind the

oesophagus in the mid-thoracic region. It is at these points that herniation may occur during artificial pneumothorax. The visceral pleura is inseparably connected with the lung and lines the interlobar fissures. The subserous areolar tissue invests the entire surface and extends inwards between the lobules. It contains a large proportion of elastic fibres. The free surface, a single layer of flattened nucleated cells, is smooth and moistened by serous fluid. Beneath this lie the elastic fibres and a good deal of unstriped muscle.

PULMONARY PHYSIOLOGY

Efficient respiration consists of adequate gaseous exchange between air and blood in the lung and between blood and cells at the capillary-tissue boundary. It presupposes efficient ventilation.

Ventilation: This is simply the rhythmic bellows-like action of the lung that ensures an adequate supply of air coming into contact with the respiratory epithelium.

According to Keith (43), the expansion of the thoracic cavity can be divided into two portions, namely, upper, where the movement of the upper five ribs causes expansion chiefly in an upward and forward direction, and lower, where the movement of the lower ribs and diaphragm cause expansion mainly laterally and downwards. These volume changes of the thorax cause approximately equal changes in the amount of air in the lungs.

Valuable information about the efficiency of ventilation can be obtained from spirometric studies.

Spirometric Data: A few of the more important practical data are:-

Vital Capacity - This is the maximum amount of air which can be exhaled following a maximum inspiration and varies in health from three to five litres or more, depending

upon physique and training. In pulmonary disease states it is frequently lowered. It is made up of:

Tidal air - the amount of air ventilated by one quiet respiration, approximately 500 c.c.;

Complementary air - the maximum amount of air which can be inhaled after a quiet inspiration and varying from 2.4 to 3.8 litres, and

Reserve air - which is the maximum amount of air which can be exhaled after a quiet expiration, about one litre.

In addition, there is Residual air, the amount of air left in the lungs following a maximal expiration in amount about one litre.

The minute volume of respiration is the amount of air ventilated under basal conditions, equalling, approximately, respiration rate times 500 c.c.

The maximum breathing capacity is the maximum amount of air that can be ventilated in one minute and varies from about 100 litres in the female to 150 litres in the male.

Ventilation Equivalent - This is the number of litres of air which must be ventilated to ensure the absorption of 100 c.c. of Oxygen. Normally, it is between two and three litres and, if it is high, the work of ventilation is increased. It is a measure of the efficiency of the pulmonary circulation.

Disturbances of Respiration in Pulmonary Tuberculosis: Respiratory insufficiency is produced by any condition preventing normal exchange of gases between the alveolar air and the blood. This may be due to -

(a) defective ventilatory function,

(b) inadequate composition of the alveolar air,

(c) any abnormal condition of the alveolar wall, such as thickening or thinning of the wall with diminished vascularity, the presence of exudate in the alveoli, or the presence of disease foci interfering with normal gaseous diffusion.

In far advanced tuberculosis, ventilatory failure may occur from widespread foci of tuberculous tissue, or bronchial occlusion with exudate or from bronchial tuberculosis, preventing an adequate supply of air from reaching the alveoli. Localised interference with ventilation is more frequent which, without leading to gross ventilatory failure, leads to decreased Oxygen tension in certain parts of the lung. The result is that blood coming from the affected areas is under-saturated with oxygen. Cyanosis may thus occur where blood is circulating through the walls of alveoli which are not receiving enough oxygen. When, however, these areas are collapsed so that blood circulation virtually ceases, the cyanosis disappears.

Ventilation may also be impaired by decreased movement of the thorax in pleurisy, in fibrosis of the pleura or lung, in diaphragmatic paralysis, in bronchial stenosis, in the presence of copious secretions and in the general atrophy of the muscles which occurs as part of the general wasting of advanced tuberculosis.

We are not here further concerned with respiration, except to note that it is under the control of three factors, namely, the respiratory centre and carotid sinus, the vagus nerves and the chemical condition of the blood.

The Bronchial Movements: A knowledge of the bronchial movements and an appreciation of their importance in pulmonary physiology and pathology is essential to an intelligent approach to the problems of treatment later to be discussed.

Macklin (44, 45, 46) deserves mention for having thrown much light on a previously obscure subject.

The bronchial movements may be divided into:-

A. Those not connected with the bronchial muscle, namely, pulsations transmitted from the heart and great vessels of the thorax, transmitted deglutition movements (47) and bendings of the bronchi in forced expiration in the presence of a high diaphragm. There is also the lung mass adjustment later to be described.

B. Movements associated with the bronchial musculature. These are length changes, calibre changes and peristalsis.

Length Changes:

By inspiratory lengthening and expiratory shortening, all the respiratory tissue is enabled to expand and contract. These movements can be seen directly through the bronchoscope (47). In addition, Jackson (47) brought forward X-Ray evidence. In the case of a child with a metallic foreign body in the right main bronchus, this could be seen to move through an excursion of two centimetres. In addition, the bronchi move apart, i.e. "fan out", as they lengthen (45).

Certain adjustments are necessary because the thorax does not expand equally in all directions. The space gained in inspiration is mostly caudally, from the movement of the diaphragm, and antero-laterally, from the movement upwards and outwards of the ribs and sternum, a little towards the mediastinum and none at all at the posterior apex and in the postero-medial region or the "supero-retro-radicular area" described by Macklin (46). The lung expands in these areas by the recession from the periphery of the basal ends of the bronchi, thus permitting their lengthening. Keith (43) described how the lung roots are drawn downwards and forwards by the action of the diaphragm and the movement of the lung root can be clearly seen if a metal rider is applied to the carina (48).

This lung mass shifting is essential to the proper expansion of areas of lung lying against inexpansible walls. Interference with lung root movements may thus explain the predisposition of the apical regions to tubercle as the apex first feels the effect of immobilisation of the root.

The bronchial muscle is passive during this lengthening process. Shortening is due to the recoil of the stretched elastic tissue aided possibly by active muscular contraction in forced expiration.

Calibre Changes:

Bronchoscopy shows that dilatation and lengthening go hand in hand, as do shortening and narrowing. This is so at least for the larger bronchi, which can be directly seen and so by

inference for the finer bronchioles. The cause of the dilatation is said to be the higher air pressure in the bronchi, as compared with that in the surrounding lung parenchyma during inspiration but the more likely explanation is that the bronchi are pulled open in all directions by the increased elastic tension of the lung which occurs during inspiratory enlargement of the chest wall (49). Expiration and narrowing of the bronchi are associated with the recovery of this stretched elastic tissue.

It will be seen that there is a capacity increase in inspiration which varies with the depth of respiration. The greatest variability in volume is towards the periphery of the bronchial system.

Expulsive and Protective Movements:

These are the cough reflex, ciliary action and peristalsis. The last has been described by Bullowa and Gottlieb (50, 51). It seems to be dependent on the bronchial muscle and is called into play under abnormal conditions, e.g. the ejection of thick exudate and foreign material. Reinberg (52) claims that the bronchial muscle structure, in resembling other tubes which show peristaltic movements, is adapted to this form of movement. It is a reflex movement depending on the integrity of the mucosa and nerve endings. It is more active in gangrene and abscess and less active in bronchiectasis.

Lewis (53) has demonstrated peristaltic movement in the respiratory tubes of ten- to sixteen-day old chick embryos before the nerve fibres show much development and has concluded that this type of contraction is a function of muscle itself.

The Mechanics of Respiration: The two layers of the pleura are everywhere in the closest functional contact. The conception that the pleural space is non-existent (54, 55) is not, however, strictly correct. There is a very small but definite space between the pleural layers containing fluid and this fluid serves the purpose of lubrication and the functionally more important one of providing a strong cohesive force between these layers. The movement of the outer layer is thus closely and immediately followed by that of the visceral layer (49).

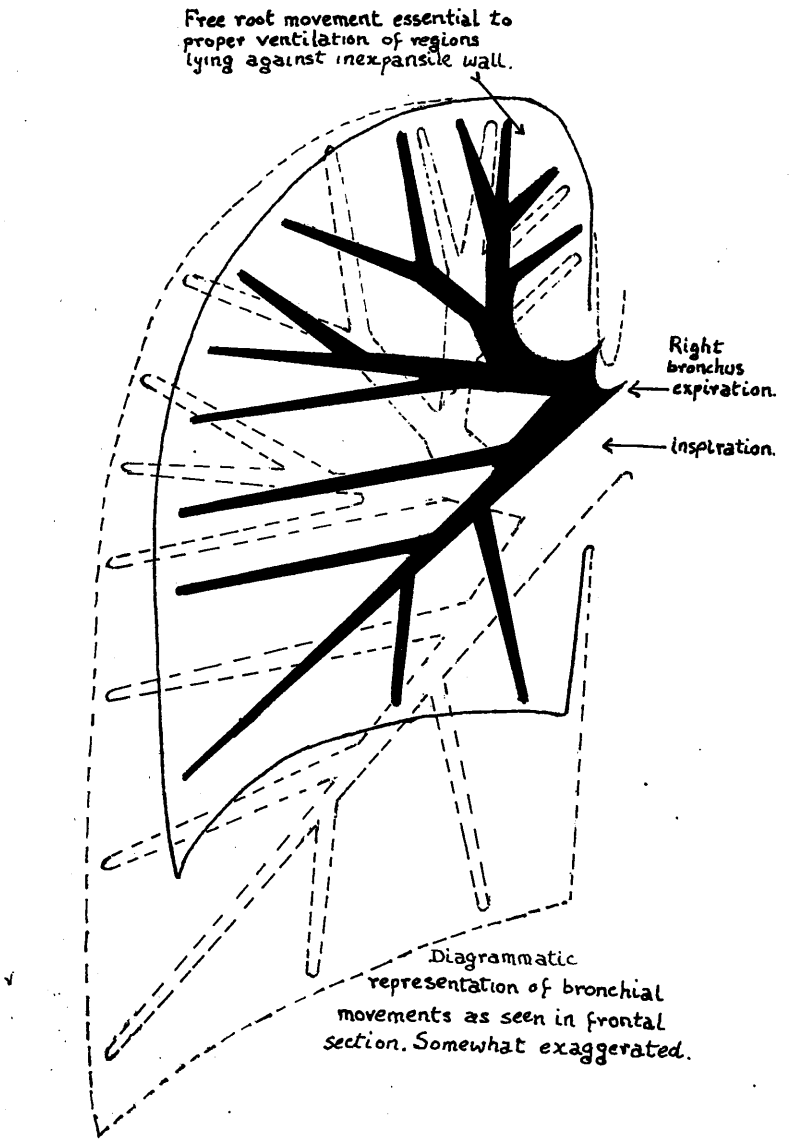


Diagram to show the bronchial movements and root movements. (After Macklin,C.C.) (45)

When the chest wall is opened after death, the lungs collapse and come to occupy much less space than they did during life, even in maximum expiration. It follows that the lungs, in all phases of the respiratory cycle, are stretched to some extent and, the more they are stretched, the greater the tendency to recoil.

The air pressure in the pulmonary air spaces at a hypothetical position of pulmonary rest is one atmosphere, i.e. 760 mm. of Mercury. Because of the elasticity of the pulmonary tissue and the tendency to recoil, the full pressure is not transmitted to the pleural space but is reduced by the amount of the elastic traction operating at the moment. The pressure in the pleural space is thus sub-atmospheric and varies from minus 5 mm. Hg. to minus 10 mm. Hg. in expiration and inspiration respectively.

The Pleural Stresses:

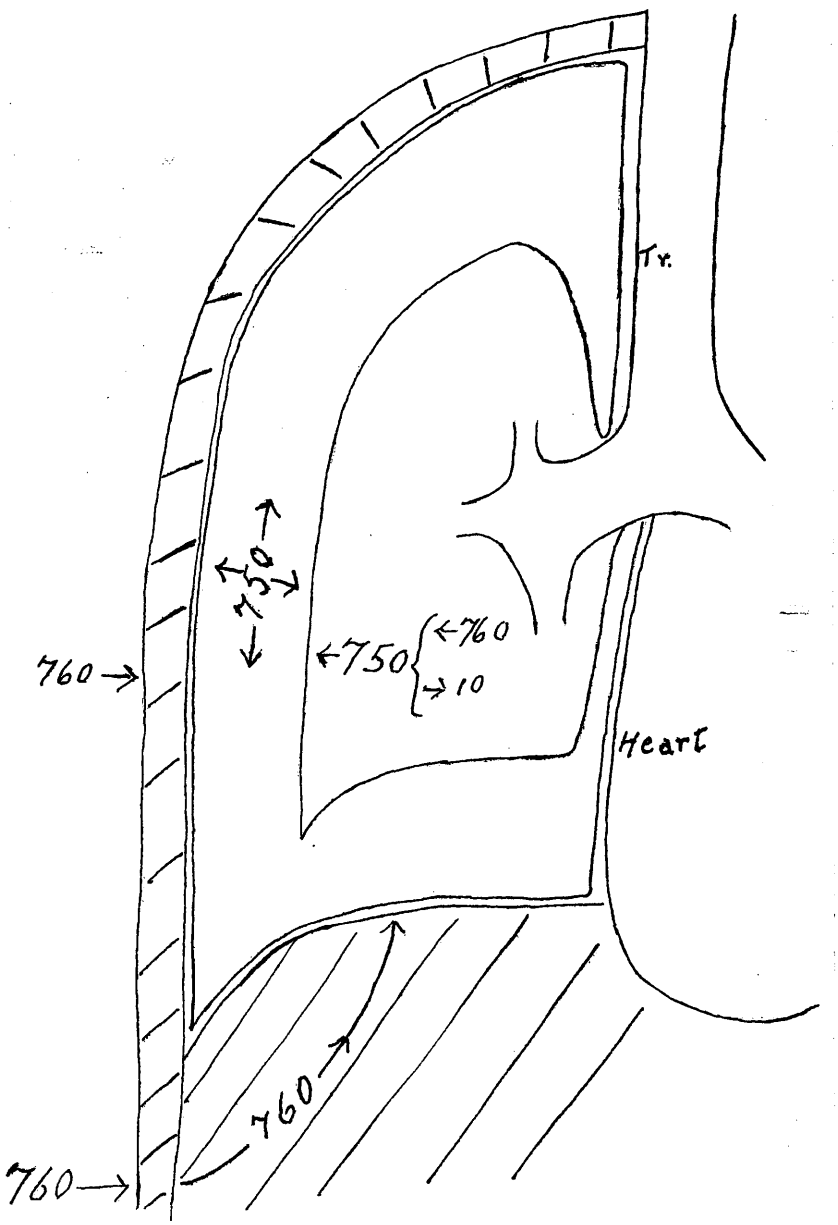
The intrapulmonary surface of the pleura is subjected to a stress of one atmosphere minus the retractive force of the lung. Thus, at the end of inspiration, 760 mm. minus 10 mm. equals 750 mm. Hg. and this is the pressure which the contents of the pleural space must assume. The visceral pleura is in a position of equilibrium. Although the intrapulmonary pressure is greater than that of the pleural space, the difference is precisely balanced by the elastic traction of the stretched lung (49).

It has been convincingly demonstrated by Andrus (49) that the outward movement of the visceral pleura is caused by a force provided primarily by the muscular energy of the thoracic walls. The outward movement of the parietal pleura is transferred to the visceral pleura through the inelastic intervening bridge of pleural fluid. The lung thus expands by being pulled out and not pushed out by the gas pressure within the lung. The normal movements of the visceral pleura are not caused by the negative pressure in the pleural space (which is the resultant of two forces and cannot "cause" movement) but are simply caused by the close adhesion of the two pleural layers.

This is not the case, however, where a pneumothorax is present. There is here no close adhesion of the pleural layers. The initial outward movement of the

parietal pleura is caused as before by the muscular inspiratory expansion of the thorax. The pressure of the gas in the pneumothorax space thus falls as the volume increases and the visceral pleura is pushed out by the excess of pressure of gas in the lung until the position of equilibrium of stresses is reached.

Diagram to illustrate pleural stresses (after Andrus, 12).



Mechanics of Inspiration:

Contrary to the usual teaching that the pulmonary air chambers are inflated by the inrush of air on inspiration, they are more probably expanded by traction from the outwardly-moving thoracic wall, transmitted along innumerable elastic tissue fibres throughout the lung. From the behaviour of elastic under stress, this means that all the air spaces throughout the lung are expanded simultaneously. The inrush of air is thus secondary to this expansion. The gas pressure in all the air spaces falls at the same time and air passes into them from the bronchi. The uniformity of pressure distribution depends on the variable elasticity of different parts of the lung and also upon the freedom of the lung to adapt itself to the varying shape of the thorax. As previously mentioned, fixation of the hilum from any cause may prevent proportionate expansion of the parts above and behind. Another function of the alveolar pores may also be to protect the delicate alveolar walls from damage by equalising the pressure of gas in adjacent alveoli.

It is likely that the gas pressure and mechanical stresses are much the same in different parts of the lung at a given time. Although the upper parts of the lungs are usually regarded as functionally less active than the bases, the downward displacement of the hilum during inspiration allows more space for the upper lobes to expand and it seems likely that the apices are as active in their ventilatory function as other parts of the lung. Walsh (56) goes further and states that the bases are functionally less active than the apices but his evidence is not convincing.

As previously noted, bronchial length and calibre changes are essential elements in lung expansion. This has been said to be due to excess gas pressure in the bronchi as compared with the surrounding lung but mechanical pull by the expanding lung is more important. Dilatation of bronchi is maintained in full inspiration with the breath held when no gas pressure difference exists any longer.

Mechanics of Expiration:

During inspiration a reservoir of energy is produced and when inspiration ceases, the increased tension of the elastic tissue of the lung tends to restore the status quo. Gravity also has an influence in restoring the ribs to their normal position.

The pleural space pressure has been shown to be sub-atmospheric in all phases of respiration. Thus, when the thoracic walls move inwards, the slack must be taken up simultaneously by the contraction of all the pulmonary elastic tissue. The air is actually expelled from the lung by the contraction of the elastic tissue in the walls of the air spaces. This is a passive act under normal conditions.

During abnormal respiration, e.g. cough, the intra-pleural pressure becomes positive and there is a localised lessening of elastic tension at the surface of the lung with a rise of gas pressure in the peripheral alveoli. Protracted severe cough may thus result in injury to these alveolar walls as witness the large emphysematous bullae often seen near the surface of the lung where there is a history of chronic cough. Partial bronchial obstruction in inflammatory states must, of course, be taken into account also when discussing the production of emphysema.

Physiology of Collapse Therapy: By collapse therapy is meant any form of treatment designed to produce a reduction of pulmonary volume. It includes artificial pneumothorax, operations on the phrenic nerve leading to paralysis of the diaphragm, oleo-thorax, extrapleural pneumo- and oleo-thorax, pneumoperitoneum, and thoracoplasty. Internal pneumolysis may also be mentioned as a measure used to improve the degree of collapse when the lung is held up by adhesions.

As a result of the reduction of lung volume, certain defects are produced and certain compensatory mechanisms come into play. Among the defects noted are:-

(a) Reduction in total pulmonary volume.

(b) Decrease in vital capacity.

(c) Increase in intrapleural pressure (57).

The total pulmonary volume can be reduced to the minimum vital capacity, i.e. the tidal air plus the residual air. The vital capacity can theoretically be reduced to less than the tidal air as an increase in the respiration rate ensures that the minute volume is

adequate. In this event, however, there would be no reserve to meet the needs of even mild exercise and such drastic reductions are not seen usually in collapse therapy.

Compensatory Mechanisms:

Muscular compensation. There is an increase in normal respiratory muscle function, the auxiliary muscles of respiration are called into play and there is also actual muscle hypertrophy.

Increase in respiratory rate, the object being to maintain the minute volume at an adequate level under varying conditions.

Increased oxygen absorption by an increase in the capillary bed available for gas exchange.

Increased cardiac output leading to increased pulmonary circulation.

A decrease in the circulation through the collapsed parenchyma.

An increase in the circulating haemoglobin giving the blood an increased oxygen-carrying capacity.

Hypertrophy of the right ventricle.

Emphysema of part of the lung to fill the vacant space. Perhaps actual hypertrophy of lung tissue.

Pneumonectomy:

This may be taken as an example of how the pulmonary volume can be diminished without seriously disturbing respiratory function. Following the operation on a dog, the following mechanisms can be observed:-

There is dyspnoea for a few hours. There is a gradual obliteration of the pleural cavity by dislocation of the mediastinum and possibly by actual hypertrophy of lung tissue. The alveolar CO_2 at first drops and then rises above normal, returning to the normal level by the 25th day. The alveolar oxygen rises at first then drops below normal and returns to normal about the same time as the CO_2. The haemoglobin shows a permanent rise. In dogs it seems that there is a true hypertrophy of lung tissue.

Physical and Physiological Aspects of Pneumothorax:

Ventilatory function can be studied by spirometry and bronchospirometry. Respiratory function is studied by gas analysis. The mechanical behaviour of the lung can be followed by X-Rays and by manometric observations.

Intrapleural Pressure:

In a normal person the intrapleural pressure is sub-atmospheric and is equal to the atmospheric pressure minus the retractive force of the lung. It is expressed as the difference between the two and oscillates during respiration between approximately minus 2 cm. of water in expiration and minus 6 cm. of water in inspiration.

The normal lung tissue has perfect elasticity. In other words, the stretch is proportionate to the stress. In diseased conditions, such as tuberculous infiltration, fibrosis and bronchial disease, there is an increase in the elastic modulus of the lung. This means that more force is needed to produce a given degree of distention. In such conditions, therefore, the intrapleural pressure is frequently lower than normal.

Effect of Pneumothorax:

When air is injected into the pleural space, several things happen. First of all, the pressure in the space rises, i.e. becomes less negative. That this pressure change is reflected in both pleural cavities was demonstrated by Graham and Bell (58) and Graham (59). Investigating the question of open drainage in empyema, they conducted experiments on the mechanics of respiration in dogs. Observing the effect on the pleural pressure on one side of injecting air into the opposite pleural cavity, they found that if the pressure of air was raised to 10 cm. of water, then the pressure rose to nearly the same level in the opposite space. They thus deduced that the thorax of the dog could be regarded as functionally one space. Graham later found that if greater pressure was used, a greater and greater difference existed between the two sides. However, the mediastinum in dogs is known to be very thin and permeable rather freely to gases and fluids. The elasticity and permeability of the mediastinum is variable in humans and, obviously, the conclusions of the above authors are not valid if the mediastinum is, for any reason, thickened and rigid. The amount of shifting of the mediastinum,

which is frequently observed in pneumothorax treatment, gives an idea of its elasticity. When pleural thickening occurs as, e.g. after an effusion, mediastinal shift is much less frequent.

The air injected expands to some extent as it rises to the temperature of the body. The volume of the lung is reduced, the diaphragm is pushed downwards, the thoracic cage is moved outwards and the mediastinum is shifted towards the opposite side. As all these things happen in varying degrees, it is impossible to predict how much pulmonary collapse will be caused by the injection of a given quantity of air.

A relationship does, however, exist between the amount of air introduced, the amount of lung collapse and the intrapleural pressure. This varies from patient to patient and, indeed, in the same patient from time to time. It can be said that, if the thorax is distended but little by the injection of a certain amount of air, then the lung will be collapsed relatively much and vice versa. It can further be said that, with a rigid or restricted pleural space, a given amount of air will raise the intrapleural pressure relatively much. This would occur, for example, where the pleura is thickened, where many adhesions are present or where the lung is maximally collapsed. On the other hand, a given amount of air will raise the intrapleural pressure relatively little if the lung is easily collapsed or the mediastinum easily shifted or the thorax easily expanded.

In a normal pleural space high intrapleural pressure indicates low pulmonary stress, i.e. marked collapse. Low pressure means little collapse. However, in diseased lungs, there is an increase in the elastic moduli and these conditions no longer apply. Stress and expansion are not then equivalent terms (60).

The pressure amplitudes indicate the discrepancy between the expansion of the thorax and that of the lung. It is everyday experience that large amplitudes indicate large thoracic excursions and small pulmonary ones. In other words, relatively large changes in volume are taking place, as for example in pleural fibrosis, bronchial stenosis, etc. Small amplitudes mean an easily distensible lung and only small volume changes in the pleural space.

Obviously, collapse of the lung means a decrease in the pulmonary air content but there is no definite relationship between the amount of pulmonary collapse and this decrease in air content because, with such a collapse, there is also a diminution in the blood content. The amount of air removed from the lung when air is injected intrapleurally can be measured by the bronchospirometer. In experiments, the injection of up to 400 cc. of air resulted in no change in intrapulmonary air (60).

When pneumothorax has been in existence for some time, thickening of the pleura occurs in greater or less degree, being much more marked where a pleural effusion has occurred. This reduces the distensibility of the lung. Obliterative pleuritis sometimes occurs leading to gradual loss of the pneumothorax space. Fibrosis may at the same time be occurring round the disease foci in the lung. These two factors lead to increased rigidity of the walls of the pleural space and to increase of the elastic modulus of the lung. Air refills thus have to be reduced as time goes on to keep the intrapleural pressures at a fairly constant level. Air is also absorbed more slowly through the thickened pleura. At the end of artificial pneumothorax treatment, when the lung is being allowed to expand, the pressures become "highly negative" and may be the cause of the terminal "ex vacuo" effusions so commonly seen.

Selective Collapse:

If artificial pneumothorax is induced in the presence of a normal lung, the lung collapses evenly, retaining more or less its normal shape. As previously noted, however, the presence of disease foci in the lung parenchyma, causes an increase in the elastic modulus of this diseased area. The result is that, when the pull of the chest wall is released and the lung is free to assume the shape dictated by its inner stresses, the diseased area collapses more than the healthy part of the lung. A further cause of this selective collapse is bronchial obstruction which, if complete, causes airlessness of the area involved. Further, it is seen if fibrosis exists. The development of selective collapse is not under the control of the operator except to a limited extent.

In such an area of selective collapse, if the distensibility of the lung is reduced to such an extent that the movement of the chest wall has no tendency to cause this part to expand, ventilation ceases, the

contained air is absorbed by the blood stream and selective collapse and rest becomes a fact. Meanwhile, respiration continues in the healthy portions of the lung, although perhaps to a reduced extent.

Adhesions:

In the presence of adhesions, lung collapse is irregular, the areas held up by the adhesions being prevented from collapsing and cavities are held open. Adhesions take various shapes and sizes and have varying degrees of importance. It is common knowledge that they are most frequently found holding up just that area which it is most important to collapse. Their elasticity varies with their age, shape and size. They act together as a force opposing the intrapleural pressure. The sum of the stresses of all the adhesions plus the atmospheric pressure equals the sum of the intrapleural and the pulmonary stresses (60).

Adhesions prevent the pleural space from enlarging when air is introduced in the same way as does pleural fibrosis. Thus, with given amounts of air, the intrapleural pressure tends to rise more than it would do if adhesions were absent.

Behaviour of the Pleural Space gases in Artificial Pneumothorax:

The gas introduced into the pleural space, usually air, is steadily absorbed into the bloodstream via the pleural vessels until it finally disappears. The pleura covering the lung is normally permeable to the constituent gases of air (O_2, CO_2 and N_2) and exchange goes on between the intrapleural gas and that in the venous blood in the pulmonary capillaries leading towards an equilibrium of pressures on both sides of the pleural membrane.

Wright (61) has described this process very clearly. The pressure of the pneumothorax gases is about atmospheric, (actually slightly less, as previously shown). The venous blood gas pressure is substantially less than the arterial gas pressure because of the fall of oxygen pressure and the small rise in CO_2 pressure. Nitrogen and CO_2 diffuse out of the blood and oxygen diffuses in, thus leading to a net loss of gas from the pleural space and a consequent drop in pressure. The

lung expands to balance this loss in pressure and the volume of the pneumothorax space is reduced. The pressures of the gases still remain higher than those in the venous blood and further diffusion, drop in pressure and expansion of the lung takes place. Thus the pleural space goes on diminishing in size until finally the gas has absorbed and the lung has re-expanded.

The mechanism described above also applies when the production of atelectasis beyond a bronchial obstruction is considered and also in the absorption of air from a pulmonary cavity when the draining bronchus has been completely obstructed (62). This mechanism will be referred to later.

Pleural Gas Analysis:

This procedure is mentioned here as it gives information about the existence or otherwise of a broncho-pleural fistula. In a relatively stable early artificial pneumothorax, the composition of the pleural gases is approximately:

Oxygen 1 to 5 per cent.

Carbon Dioxide 6 to 9 per cent.

Nitrogen approximately 90 per cent.

In the presence of a broncho-pleural fistula, the pneumothorax gases undergo constant mixing with alveolar air, hence the CO_2 percentage is lower and its O_2 percentage is higher than in closed pneumothorax. A concentration of CO_2 of less than 5% and of O_2 of more than 7% is diagnostic of broncho-pleural fistula according to Pinner (60). In practice the existence of a broncho-pleural fistula can usually be determined by simpler methods than this.

Effects of Collapse on Spirometric Findings:

When the lung is collapsed by artificial pneumothorax, the vital capacity at first becomes less than normal. When a certain quantity of air is injected, however, it does not cause a fall in the vital capacity of an equal amount. This may be explained as follows. In artificial pneumothorax, part of the lung collapse is at the expense of the residual air which is not taken into account in vital capacity estimations. Further, the collapse of diseased areas and cavities leads to no loss in vital capacity as the lung tissue concerned is,

in any case, relatively non-functioning. Again, the injected air may be chiefly expended in pushing out the chest wall, as mentioned previously. Compensatory increased function of the respiratory muscles comes into effect after a time and may thus lead to an actual increase in the vital capacity.

Fairly constant effects are: decrease in vital capacity, decrease in complementary air and a bigger decrease in supplementary air. The tidal air remains about the same. The ventilatory reserve is, of course, reduced.

Bronchospirometry:

The measurement of ventilation in the two lungs separately was first introduced by Jacobaeus et al (63) who used a double-barrelled bronchoscope. Gebauer (64) then introduced a rubber bronchial catheter and later Zavod (65) described his technique and special catheter.

These studies are obviously important for determining the relative function of the two lungs prior to any major collapse or extirpation procedure. Owing to the artificial conditions prevailing during the actual measurements, ordinary spirometry is used for determining the absolute amounts of the volumina and bronchospirometry then gives the percentages from each lung separately.

It has been shown by these methods that clinical observation and X-Ray studies may be misleading in that pleural obliteration which markedly reduces function may not be diagnosable. It is also often difficult to assess the loss of function of a lung from widespread fibrosis. In a series of observations, Pinner (66) found that localised parenchymatous changes, even extensive ones, impaired function only slightly, whereas pleural thickening from effusion or obliteration of the pleural space after artificial pneumothorax treatment led to marked degrees of functional damage. Diffusely scattered lesions with their accompanying areas of emphysema also led to functional damage as did also tuberculous bronchitis.

C H A P T E R T H R E E

PATHOLOGICAL CONSIDERATIONS

PATHOLOGICAL CONSIDERATIONS

Before proceeding to discuss any form of treatment, it is necessary to have a clear conception of certain aspects of the pathology of pulmonary tuberculosis.

At post-mortem examination, the lungs of a person dead of this disease may present a complex and perplexing picture indeed. The various stages of the evolution and healing of pulmonary tubercle are frequently present in the same specimen. In one part, old healed lesions can be seen; in another, actively evolving disease, caseation, cavitation and so on. The picture is further complicated by fibrosis, thickening of the pleura and, possibly effusion, purulent or otherwise.

From the point of view of treatment, some form of classification is obviously necessary. One method of classifying the lesions is based upon the method of spread of the disease in the lung. For example, chronic fibro-caseous tuberculosis, with or without cavitation, is said to result from spread by way of the lymphatics. Tuberculous broncho-pneumonia or caseating pneumonia are both forms of acute phthisis and spread here is via the bronchi. Acute miliary disease or localised metastatic tuberculosis is the result of spread by the bloodstream. This is the classification of the older pathologists and is the one followed by Muir (67).

Boyd (68), on the other hand, differentiates basically between the productive and the exudative reaction, as does Pinner (66) and such a classification into two basic groups gives us a clearer picture of the early pathological changes and, at the same time, makes clearer the explanation of the changes which occur later. The productive reaction occurs in its pure form in the non-immune animal, while the exudative reaction is characterised by an inflammatory exudate, the result of an allergic state of the tissues.

The Productive Reaction

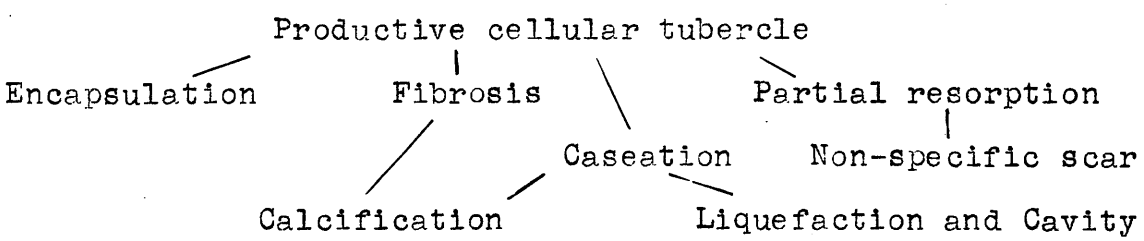
This results in the formation of a granulation tissue. It is characterised, firstly, by the proliferation of epithelioid cells. Giant cells then make their appearance and, later an outer zone of lymphocytes. This constitutes the classical tubercle and the lack of

vascularity is a noteworthy feature. Although caseation is not inevitable, it may appear as early as the second week (68). This is a coagulation necrosis which causes the details of tissue structure to be obliterated. As the tubercle grows, normal structures are pushed aside and a differentiating feature between this form and the exudative reaction is that in the caseated centres of productive lesions, no elastic fibres are to be found (66).

Caseation, as mentioned before, is not inevitable. It is absent in the hyperplastic form where virulence is low, dosage of bacilli small, or resistance high. What follows is variable.

Gardner (69) showed that, using a strain of tubercle bacilli of low virulence, many caseous lesions in animals disappeared completely without fibrosis or calcification. In other words, resolution occurred.

Pinner (66), on the other hand, states that such lesions are probably never entirely resorbed to leave normal tissue, although small, histologically non-specific scars may remain. Fibrous encapsulation or replacement (carnification) may occur and the deposition of calcium salts in the caseous material is seen frequently. Diagrammatically, the fate of the productive tubercle may be represented thus:



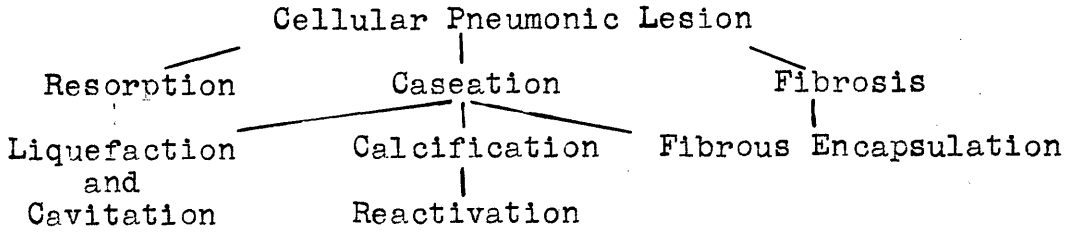
(After Pinner: Pulmonary Tuberculosis in the Adult)

The Exudative Reaction

This is the early cellular tuberculous pneumonia. Inflammatory phenomena appear within a few hours of the tubercle bacilli settling in the allergic tissues. Blood vessels become dilated and there is an outpouring of serum and leucocytes. These changes then subside and their place is taken by the formation of tubercles, but at a more rapid rate than in the productive reaction. Such lesions can resorb completely but pathological

resorption is not so frequently found as the radiological findings would suggest. If no resorption occurs, caseation follows and, in suitably stained sections, the normal lung structure can be seen in shadowy outline. Thereafter, fibrosis may occur or calcification. On the other hand, liquefaction leading to cavitation by connecting up with a bronchiole is a frequent occurrence.

Diagrammatically thus:



(After Pinner).

Resistance

The complexity of the pathological picture mentioned before has, as part of its explanation, the interplay of the factors, host resistance versus bacillary virulence and dosage. The resultant of these opposing influences varies from one individual to another and, in the same person, from time to time. A further classification can be made on the foregoing basis.

If the dose of bacilli is small, or their virulence low, while the resistance of the host is high, the result is commonly a quiet slowly progressive lesion, fibro-caseous tuberculosis or chronic phthisis. If the dose is large, the virulence high or the host resistance low, the result is tuberculous caseous pneumonia or acute phthisis. Massive repeated doses of tubercle bacilli into the blood stream leads to generalised miliary tuberculosis. Owing to the variability of the factors mentioned, as well as from the effect of still other influences, the more chronic forms of pulmonary tuberculosis may at any time become more acute.

Speaking generally, it seems that the European, especially the urbanised individual, possesses considerable resistance to the invasion of his tissues by the tubercle bacillus. This is shown by the numerous healed foci of tuberculosis found in the lungs of persons who have died from other causes and in whom there was no history of a recognisable tuberculous illness. That

resistance, in general, is low in the non-European in South Africa is well-known. Massive caseation, rapid spread of the disease and an acute fatal illness are commonly found in the Bantu miner (70).

Resistance also varies among the different members of the same racial stock. The infant and the young female have a notoriously low resistance. Such factors as inherited constitution, racial characteristics, endocrine constitution, environmental and hygienic conditions, all play their parts.

Allergy

Another important factor in determining how the individual reacts to the invasion of the tubercle bacilli is the allergic state of his tissues. On this, partly depends whether or not he can successfully overcome his disease.

First infection with the tubercle bacillus is a very different matter from that occurring later. The picture of the primary infection, the Ghon focus in the lung of the child, is well known. In spite of some writers' opinions (71), it seems clear that a high proportion of these lesions heal. The tissues, however, are thereafter left in a hypersensitive or allergic state to further infection with the tubercle bacillus. When this later infection does occur, caseation and cavitation are rather the rule than the exception.

Cavitation

It is common experience, unfortunately, that, by the time pulmonary tuberculosis is diagnosed, cavitation has, in the majority of cases, already occurred and the therapeutic efforts of the physician must be concentrated on the closure of the cavity. The era of collapse therapy has perhaps over-emphasised the importance of the cavity per se (72) but, with the introduction of Streptomycin, a new concept may take shape.

The fact remains that the cavity is a focus of danger in the lung. It is here that the tubercle bacilli find an ideal nidus for their multiplication and, being in communication with the bronchial tree, disemmination can occur to other parts of the lung and to the patient's outer environment. The cavity may also be the seat of haemorrhage, possibly fatal in itself or leading to the further involvement of healthy lung by aspiration spread of infected blood.

The cavity may be regarded in another light, namely, as a conservative measure on the part of the host. The body gets rid of the caseous contents and numerous bacilli in much the same way as occurs in the evolution of any pyogenic abscess. The cavity can thus be regarded as showing a certain resistance on the part of the individual.

Prognosis of Cavitation

In discussing the cavity from a prognostic point of view, it must first be emphasised that, prognosis being based upon so many different factors, the value of "cavity versus non-cavity" must not be given undue importance. An obvious example is that miliary tuberculosis without cavity may have a much worse prognosis than, say, the "good chronic" type of case with cavity. As will be mentioned later, cavities can be divided into several types, each connected with a predominant pathological process in the lung and each having its own prognostic implications.

When it comes to statistical evidence, and in spite of the limitations of the method, the evidence is overwhelmingly in favour of the definite worsening of the prognosis when a cavity can be demonstrated.

Barnes and Barnes (73) followed up 1,454 cases with cavity and found a mortality of 80% within one year. Within three years 85% had died and, where the cavities were from 1 to 2 cm. in diameter, no less than 88% were dead within three years.

Barnes (74) had previously shown that, of 122 patients with cavities less than 2 cm. diameter, only 31% were alive after five years.

Pinner (75) stated that cavities greater than 2 to 3 cm. in diameter not treated surgically, carried the gravest prognosis, although isolated spontaneous healing is not unknown.

Wessler (76) noted that, where a cavity existed, 63% died within four years and only 16% closed. Quoting from a series of 7,507 cases from different American sanatoria, 29% died within six months and 50% within two years.

Simpson (77), in a series of 1,601 cases with cavity, mostly less than 2 cm. in diameter, reported 60% dead within five years.

Thomson (78) reported on a series of 406 cases with cavity. Within one year, 42.3% were dead. At the end of five years 77.5% had died and 50% of those who were left had died before the end of the second five year period.

Table to show the foregoing results more clearly:-

Author	Cases	1	2	3	4	5	6	7	8	9	10 years
Barnes & Barnes	1454	80%		85%							
Barnes	122					69%					
Wessler	7507		50%		68%						
Simpson	1601					60%					
Thomson	406	42%				77%					89%

Further statistics showing the prognosis for the sputum-positive cases have a similar significance since it can be shown that, almost invariably, a positive sputum means the presence of a tuberculous cavity in the lung.

Thomson (78), in the article previously mentioned, quotes from another series of 655 sputum-positive cases in which only 27.8% remained alive after five years.

Tatersall (79), in a detailed investigation, followed up 1,192 sputum-positive cases seen at the Reading Tuberculosis Dispensary. He found that 32% survived five years and 22% survived ten years. After five years the survivors had a fifty-fifty chance of surviving a further fifteen years. After ten years survival, there was a two-to-one chance of surviving a further ten years. In addition, he found that the difference in survival rates of the patients treated with and without sanatorium care was transient only. The latter statement cannot be accepted as true unless "sanatorium care" be taken to mean bed rest and nothing more. This cannot be the case where treatment is undertaken in a modern chest hospital with all the means at its disposal. However, the statement of Trudeau (80) that the presence of a cavity nearly doubles the chance of dying within five years probably remains true today.

The foregoing figures rightly stress the danger to the individual of the presence of a cavity. Nevertheless, the mere presence of a cavity in the lung is not enough on which to base an accurate prognosis. The size of the cavity has been mentioned above as having a bearing on the outcome of the disease.

McMahon and Kerper (81) studied 296 cases with cavity. Of these, 115 became arrested, 65 by spontaneous closure and 50 following collapse treatment. The time taken in both groups was about 5 to 6 months. They further found that spontaneous healing occurred with less frequency as the size of the cavity increased. The figures were 40% in cavities 2 x 2 cm., 20% in those 3 x 3 cm., and only 6% where the cavity measured over 4 x 4 cm.

The position of the cavity in the lung is also a matter of some importance. The fact is that certain cavities tend to heal while others do not and this without reference to treatment at all.

Importance of the Type of Cavity Present:

Pinner (75) and Pinner and Parker (82), in a careful study of X-Ray findings, gross pathological appearance and of the histology in cases with tuberculous pulmonary cavitation, were able to distinguish three essential types of cavity, each with its own prognostic significance. These were:-

Type One. Small, usually multiple cavities appearing in densely infiltrated tissue and having the radiological appearance of "moth-eaten" areas. Histologically, such cavities are each found to represent simply a focalised loss of substance. They are found in areas of homogeneous caseation and the elastic tissue is fairly well preserved, as would be expected in a predominantly exudative reaction.

Such a process may lead to large areas of excavation even involving a whole lobe and the presence of such cavities indicates an almost complete lack of resistance on the part of the tissues to the tubercle bacillus. Fibrosis, however, may still develop.

Type Two. Such cavities show on the X-Ray as sharply defined, translucencies of varying size, almost always round or oval in shape, with clear borders or heavy perifocal infiltration. Anatomically, they have smooth walls and the histology is that of type one cavities. That is to say, there is a caseous lining but there is also a clear-cut ring of young fibrous tissue present. This type would indicate an early productive response to an excavating caseous broncho-pneumonia. Such cavities also behave in a peculiar manner in that they may vary rapidly in size, appear rapidly and sometimes disappear just as quickly. They may develop into the following type.

Type Three. These are the heavy-walled, irregularly shaped cavities commonly seen in or near the apex. A feature is the marked pleural thickening which is present. Histologically, they show an inner necrotic layer without elastic tissue (hence, according to Pinner, not exudative in origin). Outside this layer is a mantle of connective tissue and granulation tissue. Pathologically, such cavities show slow progress and marked tissue resistance. Productive processes are dominant.

A prognosis based on a type-classification, such as the foregoing, depends to an extent on the type of treatment adopted and this in turn is based on our knowledge of the behaviour of these various forms of cavity. It now remains to trace the development of the cavity from its inception.

Formation and Evolution of Tuberculous Cavities.

The initial stages in cavity formation are well-known. The essential preceding occurrence is liquefaction and the discharge of this liquefied caseous material through a bronchus. The cavity then occupies the place of acini and bronchioles which disappear. It increases in size by further caseation of its wall and the involvement of more and more pulmonary tissue. At first the cavity is in contact with a small bronchiole but, as it enlarges by this process of destruction larger bronchi become involved. Ultimately the draining bronchus may be a lobular or even a lobar bronchus (83).

Further small cavities develop in a similar manner in the neighbourhood and these coalesce leading to a still larger cavity. The progression in the size of the cavity is thus a process of slow ulceration.

According to standard text-books of Pathology, cavities in the lung are formed as described above (67, 68). Calmette (106) also supports this view.

That such a process does occur to account for the formation and evolution of certain cavities is not denied. However, the anomalous behaviour of certain cavities cannot wholly be explained on such a basis and considerable research has been undertaken to elucidate the real explanation (72, 84, 85, 86, 87, 88, 89, 90, 91). The result has been a revolution in our ideas of the pathogenesis of cavitation.

It is now apparent that there are two main groups of factors responsible for cavity formation.

(A) The pathological processes leading to destruction of lung tissue.

(B) The mechanical factors inherent in the pulmonary dynamics.

(A) The Pathological Processes leading to cavity formation have already been described. Cavities do not always begin as small holes in the lung and gradually enlarge as might be expected if a slow process of ulceration were responsible. Nor do they close slowly as would happen if fibrosis were alone responsible for their closure. Neither is the shape of most cavities accounted for by coalescence of other small cavities to form one larger one. If this were the case, irregular outlines would be common, whereas, in the type two cavities described previously, the shape is round or oval. Further, the fluctuations in size of certain cavities require another explanation and this is to be found in a consideration of various mechanical factors now to be discussed.

(B) Mechanical Factors.

(a) Elastic Tension. An important factor in the genesis of the cavity is that of the elastic tension of the surrounding lung parenchyma. The lung, as previously des-

cribed, is an organ exceptionally well endowed with elastic tissue and, in the unopened thorax, is in a constant state of elastic tension. That this tension factor is important in determining the circular shape of certain cavities has been pointed out by several investigators (84, 92).

Moolten (92), using calf and human lungs, introduced bronchoscopic punch forceps to varying depths down the bronchi and removed pieces of the pulmonary parenchyma. This represents to some extent what happens in the initial stages of cavity formation at the time of the evacuation of the caseous matter. Thereafter, he inflated the lungs by introducing air into the bronchi under pressure. In all cases, spherical cavities were produced as shown on X-Ray and a definite "wall" formed round the cavity, composed of collapsed lung tissue. If the lungs were examined without inflating them, tears in the bronchial walls and loss of parenchyma were seen, corresponding to the actual damage done by the punch forceps. Interruption of the lung tissue can thus be said to cause a lowered resistance to inflation. The centrifugal pull of the elastic tissue in all directions may also be a factor in the production of the circular shape of the cavity. This is by no means proved by such experiments, however.

Increased Elastic Tension of the Diseased Lung.

Van Allen and Wu (93), in investigations on experimental pneumonia in dogs, showed that there was a definite increase in elastic tension in the lung tissue even before consolidation took place and that this increased tension persisted for some time after re-aeration had occurred. This was considered to be due to thickening of the alveolar septa by early inflammatory phenomena.

That an increased tension exists in lung tissue infiltrated with tuberculosis is known and is probably due, as above, to the thickening of the septa from dilatation of the blood vessels and interstitial deposits of inflammatory fluids and cells. This has also been pointed out by Dumarest and Lefevre (94).

Clinically, it has been noted by Andrus (84) that, when atelectasis occurs in a part of the lung, as shown by the displaced trachea and mediastinum, the raised diaphragm and the crowded ribs, a cavity previously seen to be small may suddenly undergo marked enlargement.

Parodi (95), in discussing static and dynamic tension, maintained that the reason for the increased tension in any part of the lung which is the site of lesions is that the weight of such a lung section is increased and its distensibility is reduced, i.e. there is less "give". Whether this is so or not is doubtful.

Undoubtedly, elastic tension and especially increased elastic tension in diseased states has much to do with the spherical shape of some cavities and may also explain the rapid increase in size of a cavity in an area of atelectasis and its diminution in size when the atelectasis clears up. If the elastic tension were the only factor of importance in the formation of cavities, one would expect immediate reduction in size and prompt closure of the cavity on the induction of artificial pneumothorax, provided, of course, the cavity was not held open by adhesions. This does, in fact, frequently happen but the fact that it does not, in a fair proportion of cases (88), leads us to look for further factors at work.

(b) The role of the draining bronchus. Hall (120), in a lecture on "Some Rare and Obscure Pulmonary Conditions", pointed out that a valvular bronchial block could give rise to ballooning of a cavity. He described such a case in which the valvular bronchial exit was demonstrated. Clinically, the condition had simulated pneumothorax.

Vere Pearson (72) also described this factor of bronchial block to which little attention had hitherto been drawn. In a case with a large apical cavity, a needle was introduced through the chest wall into the cavity and the pressure of the gas was measured manometrically. This was plus 16 cm. of water. Air was then removed and the pressure fell, but not below a certain limit because of an apparently obstructed bronchial outlet. Two months later, on needling the same cavity, the pressure was found to be about that of the atmosphere, namely, that of the bronchi generally and the inference was drawn that the bronchial opening had become unblocked during the interval.

Pearson felt that the positive pressure within the cavity was a definite factor in causing excavation. In support of his theory, he pointed out how frequently a fluid level was found in a cavity and suggested that viscid secretions might act as a valve preventing the air from leaving the cavity. The spherical shape of some

cavities also seemed to be explained by the presence within them of air under a pressure greater than that of the atmosphere. Further, there was the analogy with valvular pneumothorax. The rapid appearance and disappearance of certain cavities could also be explained on this theory.

Morland (85), in supporting the above explanation, suggested a further analogy with emphysematous bullae in connection with which it was found that the bronchi were also blocked.

It is to Coryllos (55, 83, 87, 96), however, that the chief credit must go for demonstrating the role of the draining bronchus.

He considered, firstly, that the spherical shape of certain cavities is due to the pressure differences inside and outside the cavity, He saw that this pressure difference was largely due to the valve-like action caused by disease of the draining bronchus or at the broncho-cavitary junction. Three things may happen to the draining bronchus:-

It may remain open. The air thus continues to flow into and out of the cavity with respiration, the tubercle bacilli can multiply freely, drainage from the cavity is free and there can be no pressure difference between the inside and the outside of the cavity to account for any particular shape.

It may become closed. If the block is complete, any air within the cavity is absorbed into the blood stream as it is from any other closed space in the body (62).

It may become narrowed. This may result from disease of the bronchus or from pressure outside the bronchial wall. A check-valve may easily result. Here the air can get past the obstruction on inspiration but not out again on expiration and, hence, the pressure of gas in the cavity rises higher than that in the surrounding lung and ballooning of the cavity occurs, leading to the formation of the so-called tension cavity.

The further development of the cavity depends upon what happens to the draining bronchus. Should a partially or intermittently obstructed bronchus become fully obstructed, the air will be absorbed and the cavity will close. If it reopens so that air passes freely in and out at all phases of respiration, the tension factor will disappear.

(See diagram on next page).

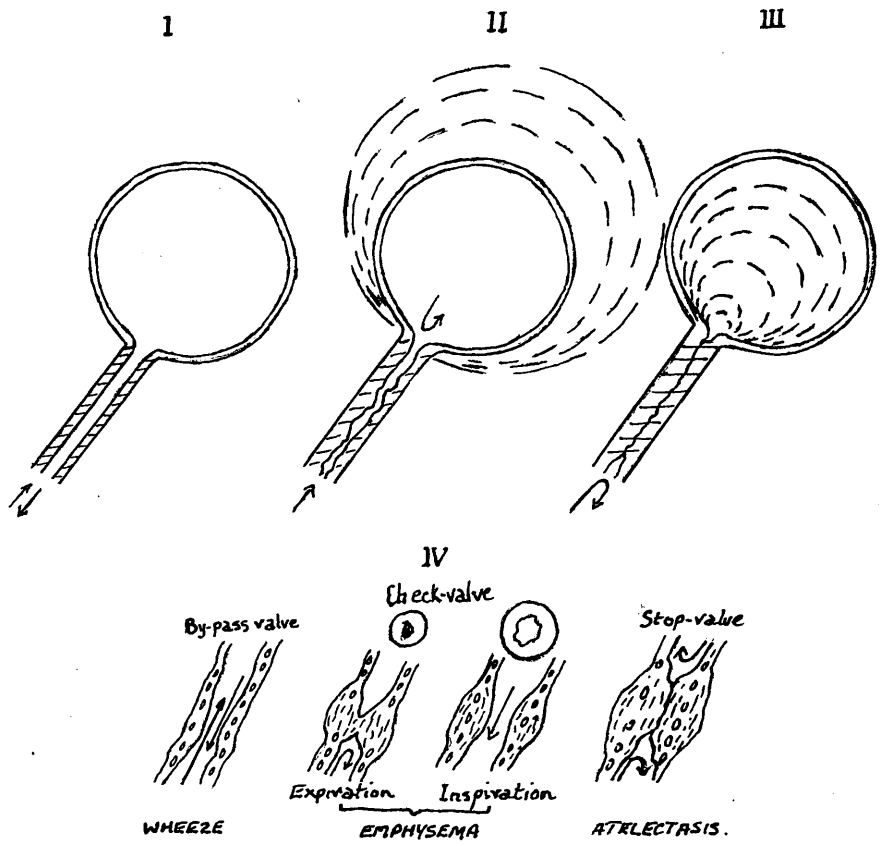
The evidence for this theory consists of analysis of the cavity gases, manometric observations of the pressure of gas in the cavity, pathological evidence and lipiodol and dye studies.

Coryllos (83) introduced a needle directly through the chest wall into the cavity and attached it to a water manometer. When the bronchus was open, the pressure oscillations were from minus 1 to plus 1 cm. of water. This corresponds to the pressure fluctuations in the bronchial tree. (Vide Goldman, 89). The analysis of the gas in such a case gave: O_2 16 to 19%, CO_2 less than 1%. In check-valve cavities, the pressure was about plus 2 plus 4 and the oscillations were absent or weak and showed on inspiration only. Gas analysis gave O_2 from 10 to 17% and CO_2 2 to 3%. A fluid level in the cavity on the X-Ray frequently showed that drainage was defective. In a closed cavity, especially after thoracoplasty, the pressure was always negative and no oscillations were observed. The oxygen fell in a few hours to below 1% and the CO_2 rose to over 5%.

Eloesser (88) also needled cavities and withdrew the secretions for examination for tubercle bacilli. If these were found while the sputum was negative, this was taken as evidence of bronchial blockage.

Eloesser also found that with a syringe attached to the needle and the patient under the fluoroscopic screen, moving the piston of the syringe back and forth caused a corresponding fluctuation in the size of the cavity. This, of course, would only be so if the walls of the cavity were quite elastic. He found, further, that constant manometric pressures occurred in the presence of thick-walled cavities while high pressures indicated stop-valve block. This is the same as the intermittently obstructed tension cavity of Coryllos. He noted that respiratory fluctuations do not refute the presence of a blocked bronchus as the pressures may be

Schematic presentation of the Physiological Action of Draining Bronchi.



I. Bronchus open. Ingress and egress of air free.

II. Check-valve cavity. Allergic inflammatory or proliferative narrowing of bronchus may produce one-way valve mechanism. Ingress of air can take place but egress is hampered. Obstructive emphysema is produced causing ballooning of the cavity.

III. Closure of bronchus causes atelectasis, i.e. absorption of air trapped in the cavity and shrinkage and disappearance of the cavity without any conspicuous scar.

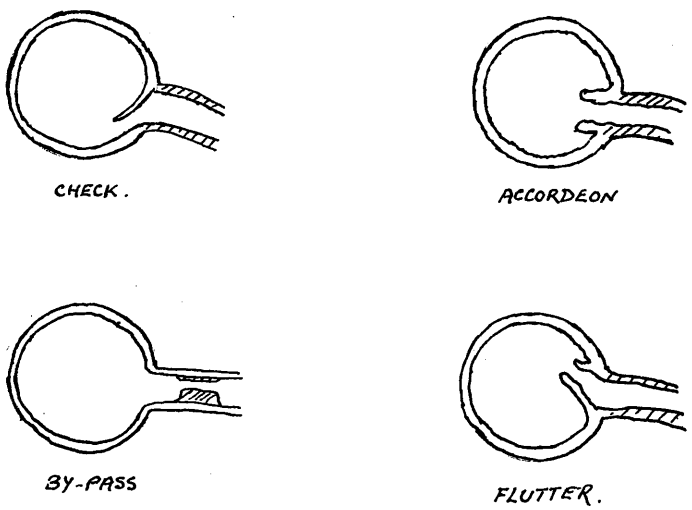
IV. Similar conditions in cancer of the lung (Jackson, 10).

/Coryllos, P.N. Amer. Rev. Tuberc. 33 : 639, 1936/

transmitted through thin-walled cavities, a fact also noted by Goldman (89). In the latter's cases, it was found that the broncho-cavitery relationships were at most times valvular and this valve was usually 100% efficient in the direction cavity to bronchus. This was shown by running air in to a pressure of plus 18 to plus 24 cm. of water. This pressure was maintained. Air, however, entered the cavity when the pressure was made "highly negative".

Post-mortem studies of two cases (Coryllos) showed in one a closed cavity with a closed draining bronchus and, in the other an unclosed cavity with an open bronchus draining it. The draining bronchus was usually found to be long and narrow. The opening of the draining bronchus may be on a level with the cavity wall or be raised on the summit of a nipple-like projection or at the bottom of a funnel-like depression. Furthermore, the opening may be set obliquely in the wall of the cavity and it is easy to see that raising the intracavitary pressure may cause such an opening to act in a valve-like manner.

Goldman et al (89) in a hundred post-mortems found tuberculous changes in the draining bronchus in every case. They also noted various changes at the broncho-cavitary junction which could lead to valvular effects. These different kinds of valve are shown below in the sketch. It is easy to see how, in expiration with bronchial narrowing occurring, these valves may close and completely prevent the egress of air from the cavity.



The importance of localised bronchial changes was also stressed by Eloesser (88). He stated that any tendency to trap air in the lung is obviously increased by any bronchial stenosis, irregularity of the surface or the presence of sticky secretion. The mucosa of the bronchus is frequently red, oedematous and covered with exudate. Desquamation and blockage of mucous glands further accentuate the bronchial narrowing. Muscle and cartilage may be destroyed with fibrous replacement (Coryllos). These pathological findings have been confirmed by Shipman (86) who, in an investigation of post-mortem material to determine the extent of bronchial tuberculosis, found approximately 10%. Aufses (97) found it in 35% of cases and Pillmore (98) in 40%. Obviously, the percentage will vary to some extent with the observer's interpretation of the findings. This is especially so with bronchoscopy.

Cavernoscopy.

Cavernoscopic investigation of certain "giant" cavities" was carried out by Coryllos and Ornstein (87). The instrument, a miniature thoracoscope, was introduced through a small cannula directly into the cavity and the interior could then be clearly visualised. Usually the bronchial opening could be located easily. If not, lowering the pressure of the air in the cavity had the effect of causing air to bubble into the cavity through the secretions at the broncho-cavitary opening. Several different shapes of bronchial openings could be distinguished, as noted above. Usually there was only one or a large principal draining bronchus. In upper lobe cavities this was situated in the lower part. If saline was run in, it remained for some time in the cavity. If air was injected to raise the pressure, the saline could not be forced out but, by reducing the pressure, air entered the cavity and the saline then ran out. Similar cavernoscopic findings were made by Goldman (89).

Bronchography.

By the injection of Lipiodal into the cavity, one can see whether the bronchial outlet is freely open or not. If apparently closed, it may still be open to the passage of air at certain phases of respiration while not allowing a relatively thick fluid such as Lipiodal to pass. Bronchography also shows various narrowings and obstructions of the bronchi in the neighbourhood of the cavity. Areas of bronchiectasis are also commonly seen.

The Role of Secondary Invading Organisms.

A corollary of Coryllos's theory is that blockage of the draining bronchus, by which he means complete block, leads to closure of the cavity drained by that bronchus. Eloesser (88) and Pinner (66) state that block of the bronchus may sometimes lead to fever, toxaemia and disaster and that secondary organisms play an important role here. However, Coryllos and Ornstein found only tubercle bacilli and no secondary organisms in material taken from cavities at cavernostomy.

A consideration of the foregoing paragraphs leads one to the conclusion that the maintenance, enlargement and non-closure of many cavities depends on the presence of a broncho-cavitary valve which allows air into the cavity easily but only out of it with difficulty, thus producing an abnormal tension of gas in it. If the valve is efficient, compression of the lung only tends to augment the tension. Such cavities tend to disappear when the pressure is stabilised at or below atmospheric.

Healing of Tuberculous Lesions.

Tuberculous lesions have an evolutionary development. That is, they form mature, regress and heal. Progression may be rapid or slow and the lesion may be intermittently progressive, stationary or regressive (99). The course of the lesion depends on numerous factors, among which are: the type and dosage of bacilli, the condition of the host, the host's environment, including diet, habits, work, mental or physical strain, mentality, the presence of other diseases. As is noted frequently, healing may commonly be found in one part and progression in another.

Healing of the lesion depends on at least a local equilibrium being established. If this is reached at an early stage, before the lesion has spread beyond the cellular barrier, the small focus becomes encapsulated and undergoes healing by fibrosis, calcification or even by resorption.

If arrest of progression occurs later, there is usually resorption of the exudative elements, including perifocal exudates and cellular infiltration. The caseous portions are usually involved by fibrous encapsulation and may later become calcified. Smaller caseous lesions may become permeated by capillaries and be resorbed, leaving only small fibrous scars containing many blood vessels.

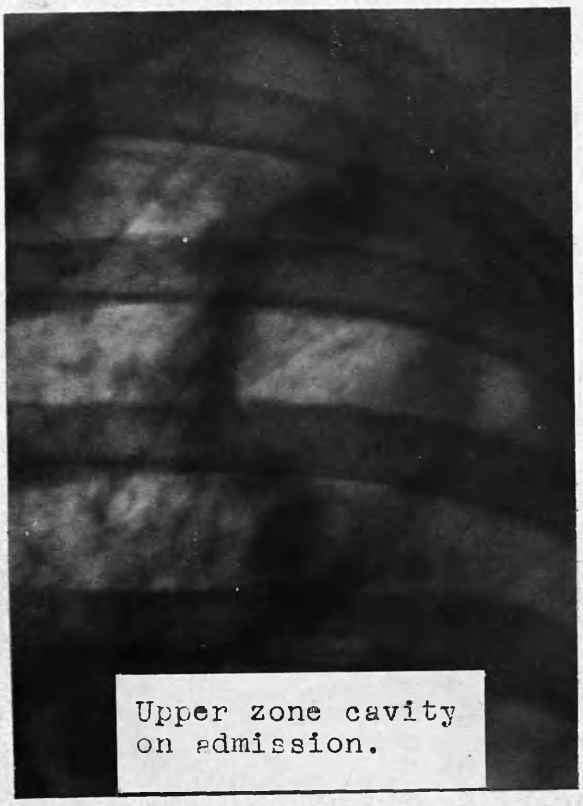
An example of healing by fibrosis was described by McPhedran and Long (100).

The case was one of bilateral sub-apical infiltration, seen first in 1928. At post-mortem in 1934 following cerebral tumour, a healed primary lesion was found in a lower lobe and, in an upper lobe a stellate scar was seen made up of dense fibrous tissue with a necrotic centre. There was marginal infiltration with lymphocytes and fibroblasts. None of the usual criteria of tuberculous activity were seen and no tubercle bacilli were found. An interesting point was that the bronchus leading to the scar was somewhat contracted by fibrous tissue, and the lumen filled with partially degenerated epithelial cells. As the history of the case showed that the sputum had been positive in 1928, it might be assumed that this was in reality a case of cavity healing although the presence of cavity originally is not stated.

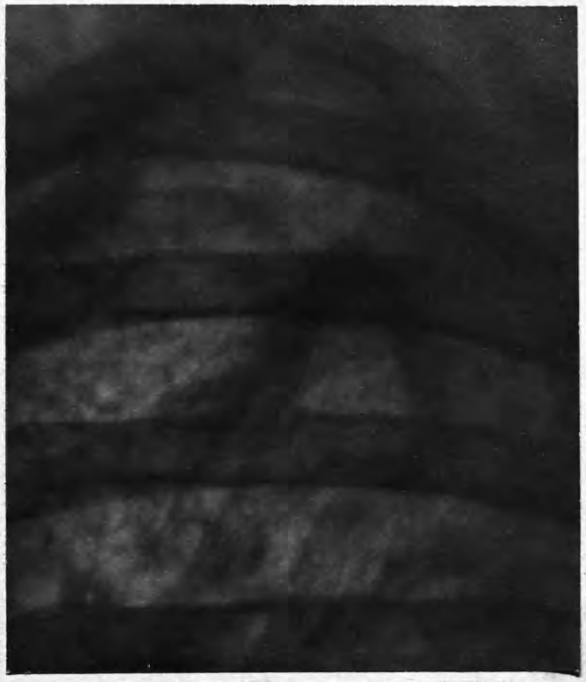
Resolution.

Amberson (101) saw resolution of some degree in approximately 5,000 cases. This was mostly evident in exudative pneumonic lesions (102). The effectiveness and extent of resolution is limited by the extent of caseation and the part to resolve most readily is the zone of collateral inflammation. The caseous nucleus, on the other hand, usually persists indefinitely. Perifocal inflammation and small caseous areas may resolve without permanent damage, but appearances are deceptive and there is nearly always some damage to tissue, leading later to impaired respiratory function.

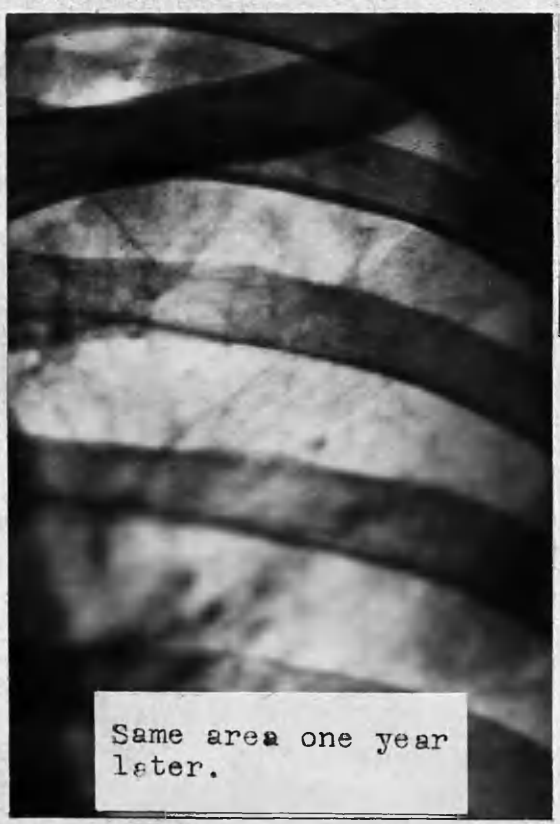
Three cases referred to by Amberson (101) showed striking X-Ray resolution. However, at post-mortem, in the regions where this resolution had been most apparent, he found calcified nodules and caseous residues containing tubercle bacilli (after ten years in one case). Some caseous foci were encapsulated but not calcified. There was fibrous thickening of alveolar walls and organisation of alveolar exudate. One case showed a cavity, closed off from the bronchi and filled with fluid rich in cholesterin crystals although the cavity had been invisible on X-Ray for ten years. He concluded "it is wise to consider resolution as only a part and an initial phase of the healing process, no matter how striking it may be".



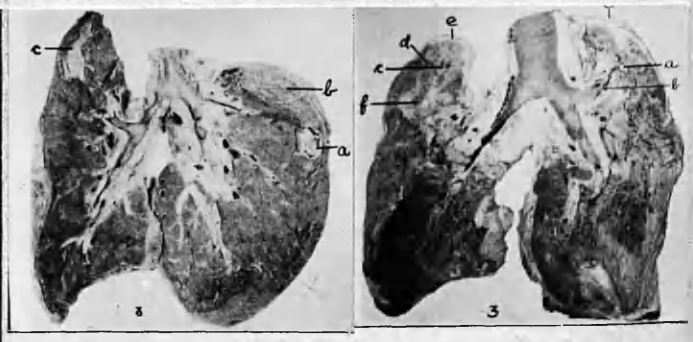
Upper zone cavity on admission.



After one month



Same area one year later.



Above

Two methods of cavity healing.

Left: Conversion to a solid focus. Bronchus closed.

Right: Open healing.

From Auerbach, O. & Green, H. Am. Rev. Tuberc., 42: 707, 1940.

The damage to the elastic tissue and the fibrosis involving alveolar walls, lead to a common condition in healed pulmonary tuberculosis, namely emphysema. Koral (103) regards this as usually vascular in origin, resulting from atherosclerosis, endarteritis or thrombosis of bronchial or pulmonary arteries. However, this may be, emphysema is often seen as a complemental process filling in the space left by shrinking healing fibrotic lesions and surrounding healed cavities. Apical bullae are often seen.

Bronchiectasis is another common result of the healing process. It is most common in the upper lobes, another way of saying that tuberculous lesions are commoner here than in other parts of the lung, and it is most marked where there has been much peribronchial infiltration. It may be caused by areas of atelectasis or actual fibrotic shrinkage.

Cavity Healing.

An elastic interpretation of the term cavity healing is required for practical purposes. Pinner (66) demands for healing that all caseous elements disappear and maintains that healing of this sort is rarely found. Morbid anatomists are less optimistic than clinicians about the possibility of healing. Auerbach and Green (104) state that their idea of cavity healing is serial X-Rays showing cavity closure and negative sputa. Cavity closure and sputum conversion is the initial aim of collapse therapy but healing, if it does ever occur, comes much later.

Methods of Cavity Healing.

(A) Gradual filling up of the cavity with granulation tissue and subsequent obliterative fibrosis. Sweany (99) believes that this organisation of granulation tissue is of importance, as does Mayer (105), but the latter emphasises that this is only likely to happen to small cavities. He estimates that connective tissue proliferation can obliterate a cavity of not greater than $\frac{3}{4}$ inch diameter.

(B) Concentric Fibrosis leading to gradual shrinkage. Mayer believes that this slow process may obliterate even a large cavity. Calmette (106), however, believed that large cavities rarely cicatrised completely. Pinner and Parker (82) point out that the fibrous tissue around a cavity may act in two different ways. The one is a more or less concentric shrinking and the other is

scar retraction which may keep the cavity open. It is easy to see that radiating bands of fibrous tissue, some of them attached to the adherent pleura, may provide a firm barrier to collapse in one direction at least (107).

Sweany (99) believes that the organisation of fibrous tissue between the layers of the pleura leads to cavity closure but Huebschmann (108) takes the opposite view and states that this is more likely to prevent closure. With the latter statement I am in agreement, as one of the most important factors in cavity healing is the concentric relaxation of the pulmonary tissue surrounding the cavity. Nowhere is this better seen than when an artificial pneumothorax has been induced. Adhesions are the most frequent bar to this relaxation process.

(C) Closed Healing by Bronchial Block. Bronchial block is considered by many authors to be an essential preliminary factor in the healing of a tuberculous cavity (87, 91, 96, 104, 108, 109, 112, 117). Others (66, 88) believe that bronchial block could lead to toxaemia and evil results for the patients owing to the fact that cavities were frequently infected with secondary pyogenic invaders. As already mentioned, however, Coryllos and Ornstein (87), obtaining material from cavities opened during muscle-flap operations, found only tubercle bacilli and Auerbach (104) confirmed the fact.

It is quite definite that bronchial block, if not essential to cavity healing, is, in most cases, at least one factor in healing. From our knowledge of the pathology of bronchial tuberculosis, it is easy to see how the bronchi, especially the smaller tubes, become completely blocked. In bronchi, already narrowed by disease, the walls can easily come into contact and, by the organisation of granulation tissue, firm fibrous union can occur. Narrowing can also be caused by fibrosis outside the wall of the bronchus and apposition can be easily further assisted by collapse therapy. Blocking, too, occurs by obstruction with granular debris and a fairly firm plug forms. This form of plug is, however, capable of later extrusion.

Pagel (112) collected thirty-nine reports on healed cavities. Of these, twenty-two became converted into solid nodules and bronchial occlusion had occurred either by caseous bronchitis or caseous plugging.

Closed healing may take two forms.

(a) Conversion to a solid focus.

Following the blocking of the bronchus leading to the cavity, the air is absorbed from the cavity, as it is from any other closed space in the body (55). The cavity then fills up with caseous debris which becomes inspissated and encapsulated with fibrous tissue. Organisation of this walled-off caseous material may take place but is rarer. Calcification, at least partial, is not so uncommon. Healing by this method is at best uncertain.

Pagel and Simmonds (91) described three cases. One of these had calcified foci and the others showed caseous and calcifying foci. The bronchi leading to these areas were occluded by similar caseous and calcifying material.

Auerbach and Green (104) gave three instances of this form of healing. In the first case there had been bilateral open cavitation present for six years. Then closure of the cavities occurred with conversion of the sputum to negative. At autopsy, cavities were found in which the lumina were filled with inspissated caseous material. No bronchial communication could be found. Microscopically, an inner caseous layer was seen resting on a zone of tuberculous granulation tissue and, outside this, a zone of hyalinised connective tissue. Outside this again was found loose fibrous tissue with many collapsed alveoli. Other alveoli showed dilatation. In the other two cases, similar findings were made.

Herington (110) also described a case which showed this method of cavity closure and he likened it to an Assmann's focus in reverse.

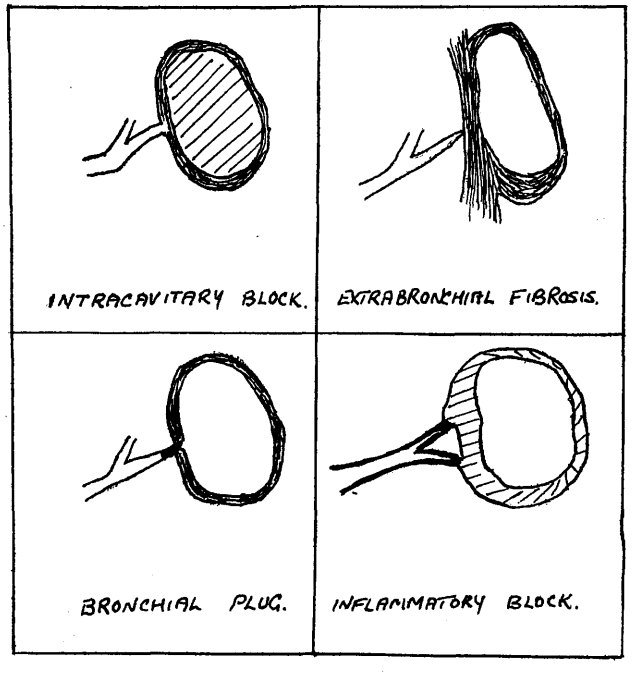
The walls of cavities closed in the above manner are finally composed of concentrically arranged hyalinised connective tissue with, perhaps, an occasional giant cell. The material within the cavity shows progressive calcium deposition and decrease in the nuclear elements. The bronchus is always closed but the dangerous volcano-like nature of this type of healing is shown by the fact that tubercle bacilli can be isolated from the centre and the cavity contents can be easily evacuated at post-mortem.

An investigation with different conclusions was made by Salkin et al (111) to determine what importance, if any, could be attached to bronchial occlusion in cavity healing.

The whole study was carried out post-mortem. X-Rays were taken following the injection of radio-opaque substances intra-bronchially and also directly through the chest wall into cavities. Only cavities of over 3 cm. diameter were included. Seventy cases were studied and thirty-seven of these had also post-mortem examinations of the lungs. It was found that 14% of the cavities had no apparent bronchial connections of any sort and it was concluded that a cavity can be maintained without an open bronchus and that bronchial occlusion was not a factor in cavity closure.

However, it does not follow that, because Lipiodol will not pass in or out of the cavity, there is no connection with the bronchial tree in all phases of respiration and during life. It has already been shown that changes in the bronchial diameter occur during respiration. In addition, Auerbach (104) found in 2,000 cavities that those with a diameter of 3 cm. or over always had a communication with the draining bronchus although a fine probe might be needed to demonstrate this.

The types of bronchial occlusion found by Salkin et al are shown on the following diagram:-



From this diagram it can been seen that the first type represents closed healing of the type now under discussion, the second is a closed-off cavity undergoing the slow cirrhotic shrinking described above, the third is blocking by a plug which could possibly be dislodged, and the fourth shows a type of block which may not be really a block at all but a partial occlusion in which air may be able to pass into the cavity in certain phases of respiration. In other words, one must distinguish between temporary and permanent block and also between what happens in the living, functioning lung and what is found post-mortem. Incidentally, the commonest cause of blocking of cavities in the above investigation was tuberculous endobronchitis.

In addition to closed healing with conversion of the focus into an encapsulated caseous focus, there occurs a rarer form but this time of more complete healing, namely:-

(b) Healing by fibrosis of the Cavity.

After blocking of the bronchus occurs, if there is little or no debris in the cavity, the cavity walls become apposed as the air is absorbed and healing occurs by fibrosis resulting in a non-specific scar. This form is not so common as inspissation of the caseous contents (104, 113, 114). However, cases have been described (99, 100, 114, 115, 116). Thickening of the cavity wall takes place by an increase in the connective tissue fibres and proliferation of granulation tissue within the lumen. By contraction of the outer layers the lumen of the cavity will then be obscured (108).

After the bronchus closes what happens is somewhat as follows according to Auerbach (104). There is an increase of fibroblasts, epithelioid cells and collagen fibres within the pyogenic membrane lining the cavity. The zone of tuberculous granulation tissue and the outer fibrous layer reveal an increase in connective tissue fibres and a decrease in the cellular elements. With the organisation of the pyogenic membrane, the granulating surfaces of apposed walls fuse and at first partially and then completely fill the lumen of the cavity.

Gilbert (116) describes the post-mortem findings in two cases where healing was by fibrosis.

Case 1: This patient was observed from 1922 until 1931 when death occurred from seminoma. Originally, there was a left apical cavity which disappeared within 18 months with rest. At post-mortem, the position of the cavity was occupied by a small grey stellate scar. Dense collagenous fibres were seen, traversed by numerous vessels with thickened walls. Lymphocytes and anthracotic pigment were present, but no specific tuberculous elements, no caseation and no core of calcification were found.

Case 2: Briefly: 1924 Onset .... rest only. 1926 .... definite left apical cavity .... left A.P. induced. 1930 .... new lesion in right upper zone .... cavity ... right A.P. Ballooning of this cavity, perforation, death. At post-mortem, two small cavities were found embedded in scar tissue at the left apex, the site of the original cavity. Small areas of caseation and calcification were also found. A second small cavity had a non-specific fibrous wall with a few giant cells. The lung tissue around was atelectatic and contained a few fibrous nodules enclosed in fibrous tissue. Here, healing appears to have been complete in the first case and incomplete in the second.

Cases have also been described by Loesch (113, 114). In his first series of five cases, healing was by conversion into caseous inspissated foci. In the second five cases, three instances of complete cavity healing were observed. In one of these sloughing out of the caseous cavity lining apparently occurred and in the other two, there may have been fibrous transformation of the caseous encapsulated foci which took place over a number of years.

Pinner (115) describes a case in which a cavity became replaced by a fibrous scar, the bronchus ending blindly in this scar tissue. From the normal appearance of the bronchial lumen, it was suggested that the bronchus became occluded because of the cavity closure and not that the cavity healed because the bronchus was first blocked. It seems from this example that fibrotic obliteration of a cavity can occur in the presence of an open bronchus but ample evidence exists to show that bronchial occlusion is a very frequent antecedent happening.

As has been stated before, Coryllos believes that bronchial block is necessary to cavity healing. This first occurs by absorption of air. The tubercle bacilli in the cavity and in its walls become starved of Oxygen. The tension factor disappears. The pericavitary zone of atelectasis re-expands and the walls become apposed. Healing then proceeds by fibrosis or the remains of the cavity fills with debris. Conditions in these inspissated foci cannot reach anaerobiosis, however, as it has been shown that viable bacilli can be recovered from encapsulated caseous foci.

(D) Open Healing. Open healing occurs following the shedding of the inner caseous layer and the conversion of the granulation tissue lining into fibrous tissue. In addition, more or less epithelialisation frequently occurs. Tubercle bacilli disappear from the sputum.

Auerbach (104) describes a case. Here a cavity had been observed on X-Ray to persist for eleven years. The sputum had been persistently negative. At autopsy the cavity was seen to have no pyogenic or caseous lining and partial epithelialisation with squamous and columnar cells had taken place. There was a free communication with the draining bronchus. The surrounding lung was contracted and fibrotic or emphysematous. This type of healing is similar to what occurs in the closed cavity without retention of contents, a gradual replacement of specific tuberculous elements with fibrous tissue.

Pinner believes that epithelialisation is rare and only occurs in small cavities but Auerbach saw partial replacement with epithelium in some cavities with caseation spreading in other parts of the wall of the same cavity. This process has also been noted in the walls of cavities after the operation of cavernostomy. The surface of such a cavity becomes smooth red or grey and, histologically, it is seen that partial epithelialisation has occurred.

Open healing may occur with persistent active disease elsewhere in the lung. One explanation is that the widely patent bronchial opening allows easy shedding of the infectious lining of the cavity. More important is the change in the immunological relationships between the host and the organism.

As regards the frequency of the various different forms of cavity healing there is a paucity of pathological reports. Pagel and Simmonds (112) collected all available reports up to that time and found that, out of a total of 33 cases, open cavity healing occurred in 5 cases, scar healing was seen in 11 and closed healing by conversion of the cavity into a solid focus occurred in 16 cases. In Loesch's cases 7 became closed foci and 3 healed by fibrous transformation.

The reason for the frequency of the closed focus type of healing lies most probably with the early closure of the draining bronchi, diseased or otherwise, which is found especially with collapse therapy. Many of these bronchi are very small tubes and are easily occluded, thus making it impossible for drainage to occur.

Adhesions.

It is the rule rather than the exception to find adhesions connecting the lung to the chest wall and by their presence preventing the ideal selective collapse of the lung from developing. As adhesions are always preceded by more or less localised pleuritis with organisation of the exudate, it is commonest to find them occurring over just that area of the lung which it is most important to collapse.

As a result of generalised pleuritis, diffuse adherence of the pleural layers may take place and with this form of adhesion we are not further concerned as it is impossible from the first to introduce any air between the visceral and parietal pleura. Thus pneumothorax may be impossible or small localised pockets of air may form which, however, do not affect the issue as the pneumothorax is quickly abandoned and no thoracoscopy is possible. Where the pleuritis is more localised, adhesions of various shapes and sizes result (118, 119).

Threads, strings and cords are frequently seen. They are composed of fibrous tissue and have narrow attachments at both their visceral and peripheral ends. A number of threads or strings together may suffice to prevent collapse but one cord by itself may be enough.

Band adhesions are commonly encountered. They vary in width and length but are flat. They consist of connective tissue and, if short, may contain lung tissue and also blood vessels large enough to give rise to considerable bleeding when cut.

Fan-shaped adhesions: These are band-like but have a narrow visceral attachment, widening out as they reach the parietes. I have seen them mostly anteriorly and medially. They may contain small blood-vessels but only rarely lung tissue. They are said to arise from a group of organised superficial conglomerate tubercles (119).

Cone-shaped: These are important as the visceral end is broad and the lung tissue is usually pulled out into the adhesion in the interior of which the elongated projection of a cavity may be found (see diagram). The causative pleuritis seems to be very circumscribed with close attachment of the visceral and parietal pleura.

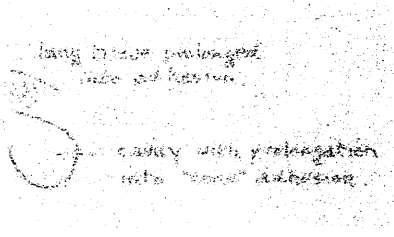
Buttressed adhesions: These are frequently seen attaching the upper lobe to the dome of the pleura. They nearly always contain lung tissue in their central part, in fact, the lung may extend right out to the parietes, while the buttresses consist of connective tissue. They may involve important vessels such as the subclavian vein and they are dangerous to cut unless one can see all round them.

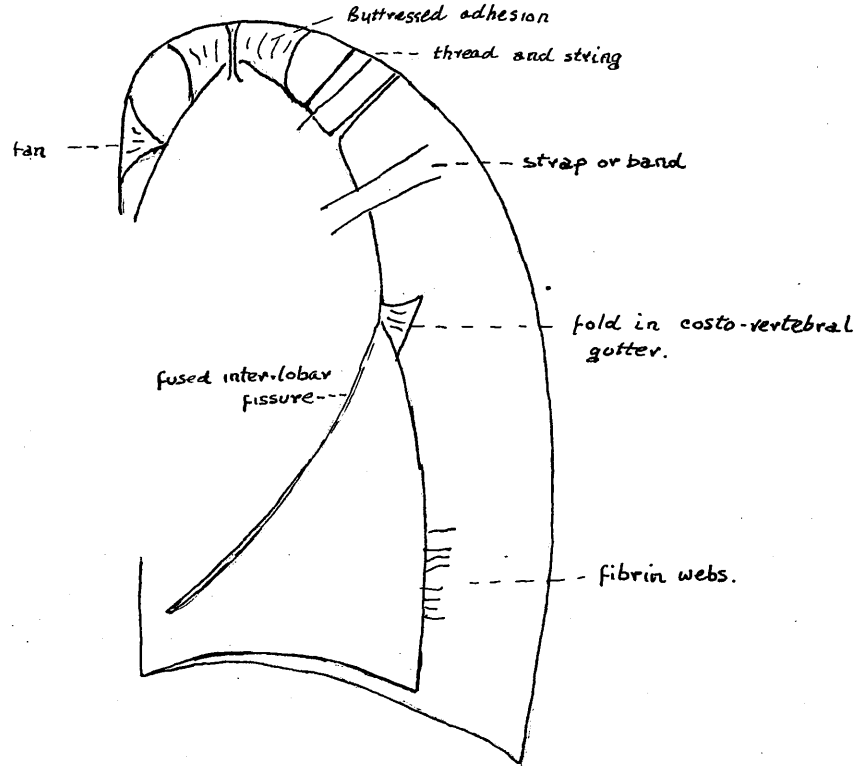
Folds of various sorts are frequently seen attaching the lung to the parietes in the costo-vertebral gutter. According to Matson (119), they commonly have their origin in an interlobar pleuritis which gums together the pleura lining the interlobar fissure and at the same time involves the parietal pleura. I have seen them frequently where the disease involves the apex of the lower lobe.

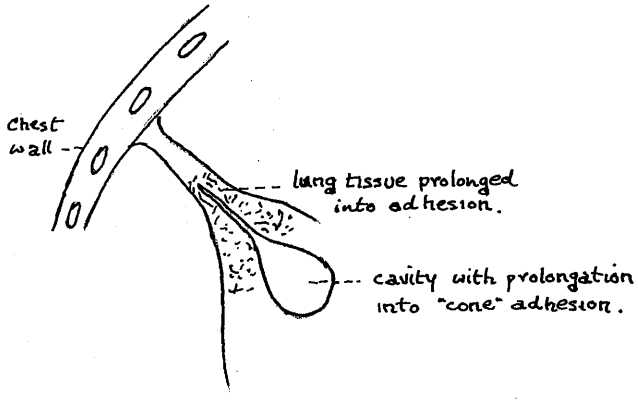
Localised symphysis of the pleural layers, frequently associated with diffuse folds of various shapes may nullify attempts at collapse and make efforts at cutting them out of the question. They arise from a more generalised pleuritis and, in places, the lung may be simply fused to the chest wall. Parts of the diffuse type of adhesion may be thin and weblike and composed of connective tissue only, while other parts are thick, vascular and contain obvious lung tissue.

Flabby, recent, weblike adhesions are frequently seen in the lower or more posterior parts of the pleural space especially if there has been some recent exudate. In time these organise into firm adhesions of the web, string or cord types previously mentioned but they do not contain lung tissue or important blood vessels.

Alexander (118) quotes various writers who have shown that lung tissue may be prolonged for considerable distances into adhesions. This has been discussed above. With the drawing out and attenuation of this pulmonary tissue, alveoli are collapsed and, in time, the adhesion consists of fibrous tissue. However, the possibility always exists that lung tissue containing tuberculous elements may extend fairly near to the parietal pleura and caution is required in cutting such adhesions lest the tuberculous tissue be cut through and an empyema result. Again, partial section of certain types, especially the buttressed adhesions, may allow tearing to take place later through tuberculous lung or even through a cavity with disastrous results.







Schematic drawing of certain types of adhesions commonly met with at thoracoscopy.
Antero-posterior view.

C H A P T E R F O U R

RATIONALE OF COLLAPSE THERAPY

INDICATIONS AND CONTRA-INDICATIONS

BRONCHIAL TUBERCULOSIS

MANAGEMENT OF ARTIFICIAL PNEUMOTHORAX

CERTAIN COMPLICATIONS

DURATION OF COLLAPSE

RE-EXPANSION

RATIONALE OF COLLAPSE THERAPY

Properly employed and with a knowledge of its limitations, collapse therapy is one of the most valuable weapons against pulmonary tuberculosis. By itself, however, it does not cure pulmonary tuberculosis unless the patient possesses the requisite resistance to heal the lesions which are present (121).

The object of all treatment of tuberculous lesions of the lungs is to assist the healing forces of nature (66). In certain limited or even fairly extensive non-cavernous lesions, this may be accomplished by rest treatment, properly carried out and sufficiently prolonged. It is not intended to convey that collapse therapy is in any way a substitute for rest. The one is complementary to the other.

When persistent cavitation is present, however, and in spite of the fact that a small proportion of cavities do heal without collapse (122), it is very frequently necessary to employ collapse measures to overcome certain mechanical obstructions to healing. These were described in the last chapter and are, briefly:-

(a) The centrifugal retraction force of the pulmonary elastic tissue.

(b) The bronchial or inflation factor.

(c) The pull of the chest wall.

Collapse therapy aims at putting the lung in the best condition for the healing of parenchymatous lesions and for the closure and healing of cavities. All the various collapse measures have this in common and, therefore, a discussion of artificial pneumothorax must take place against the background of collapse therapy as a whole (36).

The chief healing factor in pulmonary tuberculosis is fibrosis and there seems to be no doubt that this fibrotic process is definitely enhanced by collapse therapy. It is, in fact, hastened and exaggerated. It can be seen in the encapsulation of tuberculous foci, by the organisation of tuberculous granulation tissue and

FIG. 6—Right lung of patient C.C. Note the fibrous encapsulation of all tuberculous foci, moderate peribronchial and perivascular fibrosis, partial obliteration of the interlobar fissure, slight thickening of pleura, hypostasis of lower lobe, ancient chalky foci in hilar lymph nodes.

in the increase of preformed connective tissue in areas of tuberculous disease (57). See photo. (Alexander, 118).

How this takes place is not definitely known but the following factors must be considered as having influence:-

(a) Some degree of local rest. When the lung is completely collapsed, it approaches a state of almost complete functional rest but, under modern conditions, selective collapse and selective rest of the diseased area is sought. As can be seen by fluoroscopy, this is only partial rest, the diseased area taking little part in ventilation while the healthy portion of the lung continues to function to a large degree.

(b) Relaxation of lung tissue (123) in a more or less concentric manner which abolishes the tendency to retraction leading to cavity closure and allowing the contraction of scar tissue.

(c) Bronchial occlusion (55). If this is complete and the cavity walls not too thick, absorption of the contained air takes place and the cavity closes.

(d) Diminished circulation discourages haematogenous spread and this factor and the lessened lymphatic drainage from the area are chiefly responsible for the rapid lessening of toxaemia which follows collapse of the lung (124).

(e) Diminished respiratory movement lessens the risk of bronchogenic spread.

The above factors together diminish toxaemia, probably encourage fibrosis and, in addition, diminish the oxygen supply to the part which may be sufficient to retard the growth of tubercle bacilli and make their survival more difficult (96).

Clinically, the closure of cavities is a striking result of collapse therapy. Reports of pathological healing are not abundant but such as do exist show that, in time, cavities become encapsulated with fibrous tissue

and, eventually, in some cases, healed by fibrosis (113, 114, 116).

It is true that the simpler forms of lung collapse or relaxation should be adopted in a particular case if they will suffice. Artificial pneumothorax is frequently considered to be one of those simpler measures but the long-term results in all but a few cases give the lie to this assumption. The aim of therapy is to leave the patient at the end of his treatment with a re-expanded healed lung and as little functional loss as possible. Very frequently, however, complications arise during the course of treatment which prevent re-expansion or re-expansion is accompanied by such symptoms of mediastinal displacement that refills have to be kept up indefinitely or the pleural space obliterated by thoracoplasty years after the initial plan of treatment is made. Therefore, the more radical methods of treatment, such as thoracoplasty or resection, must be considered in the initial assessment of a case which requires collapse therapy. Treatment must be individual from the start and the patient as a whole must be considered and not merely his lung disease. Too much emphasis must not be laid on the "hole in the lung" (72). A proper selection of cases for the existing forms of collapse therapy is most essential (36).

In the treatment of cavities, collapse aims at overcoming the obstructions to cavity healing previously mentioned. In unobstructed pneumothorax, where the lung is able to retract away from the chest wall, the centrifugal retraction force ceases to operate as does the pull of the chest wall, the bronchus becomes kinked or closed and cavity closure results. In thoracoplasty, the same object is sought. The chest wall pull is overcome by rib resection and apicolysis and the retraction of the freed lung plus more or less compression, leads to cavity closure. It is thus a mechanical effect that is sought in the first instance.

The most effective form of artificial pneumothorax is that which provides selective collapse of the diseased portion of the lung, leaving the rest of the pulmonary tissue to continue functioning more or less normally. More will be said about this later but here it may be mentioned that one advantage of thoracoplasty over pneumothorax is that selective collapse and selective rest of the diseased area is obtained from the start.

Indications for Collapse Therapy.

It may be said from the outset that healing of tuberculous pulmonary lesions without collapse therapy is to be preferred to that with it. There is a definite risk entailed in any of the present methods of collapse treatment, even with seemingly inocuous phrenic nerve crush and, when it comes to artificial pneumothorax, the risks of early, and especially later, complications are indeed formidable and cannot be ignored. Healing without collapse leads to less later disablement. As a practical consideration, however, it is found in this hospital that probably ninety-per-cent of admissions with pulmonary tuberculosis only require to be considered for collapse treatment and a proportion of these are too far advanced to be considered safe subjects for collapse at all.

It must first be decided for any case whether or not healing is likely to take place without collapse (125) and for this an initial period of bed rest is absolutely essential. It may be objected that there is some danger of spread of the disease during this waiting period and that adhesions are likely to form making it perhaps impossible to carry out pneumothorax if it should be considered necessary later. However, under close observation, the danger of spread is minimised. As for adhesions forming, they are frequent in any case and can frequently be freed by pneumonolysis. It is not likely that pleural symphysis will take place during the observation period if it has not occurred before. The initial rest period need only be long enough for a definite decision to be reached; in some cases this will be short, in others longer.

Having seen the extent of the disease, the next step is to determine, as soon as possible, its status and to form an idea of its tendencies. This can be accomplished most successfully by a careful comparison of serial X-Rays taken at a suitable interval of time. This interval varies from case to case. In the early exudative type of disease it may be only a week or so whereas, in the more chronic types, a month or longer is safe.

Here it should be mentioned that the symptoms of toxaemia may lessen, the temperature, pulse and blood sedimentation rate may come down to more normal levels, the cough and sputum may decrease and there may be some

gain in weight, all from simple bed rest and all without meaning that the lesion is really tending to regress. This symptomatic improvement may, in fact, merely be a sign that a caseous lesion has cavitated. As regards physical signs, spreading rales may indicate that a lesion is extending.

This is also the time to consider various background factors connected with the patient. The previous environment, the mode of life and habits generally may give a guide as to what can be expected from bed rest. The race, age, sex and colour factors must also be considered. Broadly speaking, the young female is a candidate for earlier collapse than the young man. The prognosis without collapse is poorer in the coloured than in the white.

Having emphasised that there should be no rushing into collapse therapy except, perhaps, in certain cases of haemorrhage, one may define the broad indications for collapse as follows:-

(a) Disease which progresses with bed rest alone.

(b) Cavity which fails to show prompt diminution in size with rest.

(c) Certain lesions which experience shows are unlikely to heal with rest only.

(a) As long as cavitation is absent, it is quite safe to wait, provided that there is careful observation of the case on the lines indicated above. Here it may be stated that bed rest means at least being in bed twenty-four hours a day with strict regulation of all mental and physical exertion. The sanatorium regime is still the backbone of all treatment of pulmonary tuberculosis. Second-rate measures, such as ambulatory pneumothorax, may have to be adopted in certain circumstances where the shortage of sanatorium beds makes this imperative, but it must be recognised that this is a makeshift procedure.

(b) When a cavity is present the position is different. This almost always calls for collapse of some sort. Frequently, however, a watchful waiting policy is in order. Spontaneous healing of cavity sometimes takes place. Wiese (122), among others, has pointed this out. In his series of 125 cavity cases, twenty closed on rest alone. As was shown in the chapter on pathology and as

Bloch et al (35) have pointed out, the size of some cavities at the start of treatment does not represent the actual size of the lung defect. Rest leads to the loss of the inflationary effect leaving a small cavity which may then close without collapse.

Generally, however, the presence of cavitation is the most important indication for some form of collapse of the lung. If there is no lessening in its size within a reasonable period of rest, this should be initiated. This is assuming that the sputum is positive. The dangers of open cavity with positive sputum do not need to be further stressed.

Cavities are not all suitable for pneumothorax treatment. The best are fairly young cavities, not too large, situated fairly centrally in the lung. The large, circular, tension cavities usually need other treatment. Medial apical cavities are difficult to close as are those at the apex of the lower lobe. Generally, apical cavities are more favourable for closure than basal ones. It may be noted that lateral X-Rays are frequently needed to localise a cavity accurately.

(c) There are certain lesions which would appear to call for collapse of the lung without delay. They are those which experience teaches are unlikely to benefit from rest alone and include most cases of far-advanced cavitation with distortion of the mediastinum and pleural thickening indicating fibrosis. There are some workers who would say that tuberculous pneumonia falls into this category as these lesions are almost invariably about to cavitate and early pneumothorax may limit the extent of this destruction.

The foregoing are the fairly widely accepted indications. There follow some which are more debatable:-

(a) <u>The Minimal Lesion</u>.

Rafferty has collected numerous opinions on this subject and the consensus seems to be that such lesions should not hastily be considered as requiring collapse. Bloch et al (35) have stated that the tendency of really early cases to heal without collapse seems to have been overlooked by the more enthusiastic advocates of early collapse measures. It should be mentioned in passing that cases with cavity or, what amounts to the same thing, persistent positive sputum should not be classified as early or "minimal disease".

These lesions require close observation, under bed-rest conditions in many cases, to determine their status just as for more advanced disease. It is not a question of waiting for such lesions to become far-advanced. Under close observation collapse can be done early. Amberson (126) has emphasised that these early lesions are exudative in nature and hence unstable. An X-Ray may be required as often as weekly in the early stages. In such a manner, the already stable and even healed lesions can be weeded out and the progressive ones can be collapsed as required.

Pinner (66) has summed the matter up. Minimal lesions, he reminds us, are not a homogeneous group. Some need collapse and some do not. All lesions should be evaluated in terms of activity, dynamic status, potential developments, bacteriological findings and systemic reaction and all this against the background of the factors indicating the constitutional make-up or the vulnerability of the patient. Individual treatment is required and it seems most unwise to employ a blanket collapse measure, usually pneumothorax with all its potential complications, when probably 90% or more of such minimal lesions will heal with adequate rest. Woolaston (127) makes a plea for the use of phrenic paralysis in addition to rest for these limited early lesions.

(b) Haemoptysis.

It is seldom that haemoptysis per se forms the basis of a decision to collapse the lung. Haemorrhage is seldom fatal and then usually in cases where it is obvious that some form of collapse measure is required without haemorrhage or in cases where previous efforts to obtain collapse have failed. Other cases have already been turned down as unfit to undergo collapse treatment.

It is a fact that in certain cases, one decides on an urgent attempt at collapse of the lung as a result of haemoptysis, whereas the other indications would have encouraged one to wait and persist with rest. One must initially try to decide which side needs collapsing, a thing which is not always easy. The patient's sensations are of some value here as is a careful study of the X-Rays. A fairly rapid collapse of the lung with large refills of air is required usually but, on the other hand, a small collapse by allowing the blood vessels to relax may be enough to stop the bleeding. A contralateral collapse may also be mentioned here if all else fails.

(c) Pleural Effusion.

A large percentage of cases of pleurisy with effusion precede or occasionally accompany active parenchymal disease. When confronted with such a condition, it is therefore wise to have careful X-Ray studies of the lungs made. If it cannot be seen that the parenchyma is clear of tuberculous disease, one can then proceed to aspirate some of the fluid when the acute febrile phase has settled down. This will encourage re-expansion of the lung and, as it re-expands, a careful watch should be kept for lesions of an active nature in the lung. If these are found, the fluid can then be replaced by air and the refills continued as required.

(d) In Pregnancy.

Uncontrolled tuberculosis is as dangerous in association with pregnancy as without it and, therefore, an attempt should be made to get it under control with rest in the first instance and, if that is not sufficient, collapse must be considered. If possible, collapse measures should be delayed until after delivery. It is at this time especially that a close watch must be kept on the state of the lesions as rapid progression may follow the descent of the diaphragm (128).

Type of Collapse.

Collapse measures are of two fundamentally different types, namely, temporary or reversible and permanent and irreversible (36). A theoretically reversible procedure such as pneumothorax should only be employed where it is considered that re-expansion can reasonably be expected or safely allowed. Permanent measures are required where there can be no reasonable hope of re-expansion or where it would be unwise to allow this to occur.

In these days when thoracoplasty has become a relatively safe operation and when resection of a lobe or a whole lung for tuberculous disease is coming increasingly into use, the principle of primary thoracoplasty and primary resection for certain types of disease must be accepted. It is no longer good practice to consider every case as one for pneumothorax and, only when this fails, to advise one of the more "drastic" procedures. For example, artificial pneumothorax in a case of large, old-standing, apical cavity may ultimately be a much more drastic procedure with its possible complications of cavity rupture, empyema and in-expandable lung than, say,

cavernostomy followed by a two-stage thoracoplasty. As Keers and Rigden (129) say: "there is no longer a case for adhering rigidly to a fixed programme beginning with a minor procedure and progressing, by a process of trial and error, to an ultimate thoracoplasty".

Having thus, according to the general principles outlined above, decided that collapse therapy is necessary, it remains to decide which form this is to take and an initial differentiation into temporary and permanent is required. The indications for artificial pneumothorax are thus those for major collapse therapy and the contra-indications include those cases which require some form of permanent collapse from the start.

As with other forms of treatment, so in pulmonary tuberculosis, compromise is often required. Some patients may be temperamentally quite unsuited to prolonged bed rest and some cannot be convinced of the necessity for it. For them, and for those who cannot afford prolonged treatment, collapse therapy may have to be advised before there has been a proper trial of bed rest.

Contra-indications to Artificial Pneumothorax.

Firstly, those cases which, while requiring collapse therapy, are considered more suitable for some form of permanent collapse from the start. They include:

(a) Cases with large apical cavity, especially if the disease is of long-standing and associated with much fibrotic distortion and pleural thickening. Thoracoplasty, with or without tube drainage or cavernostomy, gives much better results even supposing that the attempt to obtain pneumothorax space does not fail in the first instance.

(b) The grossly damaged fibroid shrunken lung. If the other lung is clear of disease, lobectomy or pneumonectomy is the procedure of choice and, if there is doubt of the condition of the opposite lung, thoracoplasty may give results which are entirely satisfactory.

(c) Bronchial tuberculosis with stenosis great enough to be visible by the bronchoscope or giving rise to marked symptoms and signs. If the stenosis is not due to gross fibrosis, streptomycin may lead to healing of the lesion. More will be said about this importamt subject later.

Secondly, those cases where the disease is of such extent that collapse therapy of any kind is unlikely to affect the issue. Judgement is required here. Sometimes surprising results are obtained with long-continued bed rest so that the patient may eventually be considered for some form of collapse. There is here sometimes an indication for a preliminary phrenic paralysis, alone or associated with pneumoperitoneum. Pneumoperitoneum may be tried alone with bed rest. Six months or longer of this treatment may bring the disease to the point where one may reasonably attempt some more radical form of collapse treatment with the hope of permanently controlling the lesion. It is in such cases also that the use of streptomycin may sometimes lead to resolution of the exudative elements to such an extent that collapse measures can be considered.

Bilateral disease is, of course, no contra-indication by itself provided that sufficient functioning lung tissue remains to carry on respiration. Respiratory function tests here find a useful application.

Thirdly, silicosis associated with tuberculosis is, generally speaking, unsuitable for pneumothorax or any other form of collapse therapy. Pulmonary function is usually so much impaired that treatment by collapse may only hasten on right heart failure or leave the patient with so little respiratory reserve that it would have been better to leave him alone. It must be remembered that the effects of silicosis are progressive in most cases and this must be taken into account when assessing any case for a trial of pneumothorax. With the improved methods of dust control here in the mines of the Witwatersrand, silicosis appears only after a long period underground. Hence the age factor must also be considered. Many silico-tuberculotics retain a fair measure of health and to add the doubtful benefits of pneumothorax and the very real possibility of its complications seldom does much but harm.

Exceptions do occur, however, and, in the writer's experience, an occasional case has been seen in which pneumothorax has prolonged life to an extent which would have seemed unlikely without collapse treatment. The only form of collapse which it is permissible to attempt in these cases is some form of temporary collapse such as artificial pneumothorax or, perhaps, pneumoperitoneum.

Fourthly, tuberculous pneumonia. There is considerable disagreement as to whether this type of disease contra-indicates collapse.

There are those who say that this is the one form of disease where early pneumothorax is required on the grounds that it is a lesion which, by its nature, is not static and tends always towards cavitation. It is better, they say, not to wait for this to occur and with it the danger of spread and also the likelihood of pleural adhesions forming.

Others, such as Bloch et al (35), consider that pneumothorax is definitely contra-indicated as an early measure because of the gravity of the patient's condition and because of the likelihood of serious pleural infection occurring. They are prepared to wait until the disease has subsided into a more chronic state. If the disease does not subside with bed rest, or actually progresses, one must consider pneumothorax as the lesser of the two evils (36). Empyema can be dealt with if and when it arises. Here, again, streptomycin has a place in treatment as it is in the exudative type of lesion that it has so far proved its worth. In the basal pneumonic type of disease, this may be combined with pneumoperitoneum.

Fifthly, associated diseases, tuberculous or non-tuberculous, must only be considered as contra-indications if they are:-

(a) lethal in themselves,

(b) terminal manifestations, or

(c) likely to interfere with cardio-respiratory function to such an extent as to make collapse of the lung hazardous.

Thus, tuberculous lesions of other parts of the body, unless falling under (b), are not contra-indications but rather strong indications for collapse. This applies to tuberculosis of the bowel and larynx. Similarly, controlled diabetes and compensated heart disease do not negative collapse.

As a further contra-indication should be mentioned the difficulty in some parts of the country of obtaining the necessary refills and reliable advice over

a long period of time. This may be impossible or the expense of such prolonged treatment may make some form of permanent collapse preferable. It is also almost useless to begin a prolonged treatment such as this if the patient is not likely to cooperate for the necessary time. In addition, where cooperation is out of the question, as in some forms of mental disease, it is useless to attempt pneumothorax.

Bronchial Tuberculosis.

There has been considerable discussion in recent years on the question of what form of treatment to employ for pulmonary tuberculosis in the presence of complicating bronchial tuberculosis.

Chamberlain et al (130), in a series of 100 cases, all with gross bronchoscopically visible bronchial tuberculosis, concluded that pneumothorax was contraindicated because of the complications which occurred following its use. Twenty-two of their cases had pneumothorax. Progression of the disease and empyema (11 cases) were the chief complications and one-third of the cases died. On the other hand, results with thoracoplasty were much better. Of 25 cases treated thus, 85% were apparently arrested or improved and only 12% were unchanged or worse. None died.

In another series published about the same time, Tuttle et al (131) dealt with 92 cases. As regards the treatment intended to control the parenchymal disease, pneumothorax was used in 47 patients and thoracoplasty in 35. In only 12 of the pneumothorax cases was control adequate. In 22 cases this form of treatment was abandoned because of the inability to control the disease or because of empyema or atelectasis. Fifteen of these cases had tuberculous empyema, 2 with broncho-pleural fistula. Twenty-two had inexpandible lungs. The worst results were obtained with pneumothorax where a high degree of bronchial stenosis was present. They, and other writers (132, 133), have stressed that bronchoscopy must be done more frequently if there is any suspicion of bronchial disease in order to find it, if possible, in an early and treatable stage.

Rafferty and Shields (134) studied 40 cases of bronchial tuberculosis diagnosed by bronchoscopy.

Pneumothorax was used in an effort to control the pulmonary disease and in 17 cases death resulted. In a further 3 the disease was uncontrolled. The complications were atelectasis, usually lobar in extent, in 17 cases; the lung would not re-expand in most of these. Infection of the atelectatic lung with anaerobic streptococci occurred in 7 cases. Tuberculous empyema occurred in 17 cases and, in 12 cases which had empyema plus atelectasis, 11 died. Their impression was that these cases were definitely made worse by pneumothorax thus confirming the findings set out above.

A further analysis was made by the above authors of the cause of death from the point of view of:

(a) the parenchymal disease, and

(b) the bronchial lesion.

The severity of the bronchial disease was of much more importance than that of the parenchymal disease in determining the prognosis. Complications were also much more frequent when the pneumothorax was induced during an acute phase of the bronchial disease. When the bronchial lesion had healed or become indolent, no complications occurred (7 cases).

Bronchial tuberculosis thus affects the treatment of the parenchymal lesion chiefly as a problem of defective drainage. Atelectasis, retention of secretions, anaerobic infection, progressive tuberculosis, tuberculous empyema, bronchiectasis and inexpandable lung, are the complications to be feared where pneumothorax is used in the presence of extensive bronchial disease in the acute phase. Such complications are relatively less frequent if pneumothorax is reserved for those cases where the bronchial disease is less extensive or where it is indolent or healed without appreciable stenosis. Once stenotic lesions are present, lobectomy or pneumonectomy are needed (133) and from the results in a recent series reported by Curreri et al (135) this would seem to be preferred treatment to thoracoplasty.

To summarise, when tuberculous bronchitis is diagnosed, it is better to abandon the plan of pneumothorax for the time being and to treat the bronchial lesion by means of bed rest, drainage by posture, possibly bronchoscopic application of silver nitrate and

also streptomycin. If, after a period of rest, the parenchymal lesion still is in need of collapse, the type of collapse should depend on the bronchial status at that time. Pneumothorax should not be used if the ulceration is extensive or even moderate but progressing, or if there is partial stenosis with a healed or indolent bronchial lesion. Only if the bronchial disease is minimal or has responded well to treatment can pneumothorax safely be attempted.

Phrenic Paralysis with Artificial Pneumothorax.

It is well to remember firstly that, apart from such operative risks as haemorrhage, damage to the thoracic duct and paralysis of nerves other than the phrenic, phrenic interruption depletes the respiratory reserve to a varying extent. According to Alexander (136), the pulmonary volume may be diminished by from one-sixth to one-half. In addition, the duration of the paralysis is very variable after crushing the nerve. Thorburn and Riggins (137) found that in only 20 of their 100 cases of phrenic crush was there full return of diaphragmatic function after from one to eight years. Forty patients had impaired function and, in 5, the diaphragm was motionless. This must be remembered when it may be necessary or desirable to carry out thoracoplasty later on. The patient then may not have the necessary respiratory reserve or defective movement of the base of the lung may encourage post-operative atelectasis to develop.

Enthusiasm for the use of phrenic paralysis was high some years ago. For example, Leslie and Anderson (138) reported that in 1,124 cases treated by intensive collapse treatment, no less than 25% had pneumothorax and phrenic paralysis together. The measure is now less popular and, as far as concerns the present work, there is one unequivocal indication for this combination of procedures. That is when pneumothorax is ineffective or only partially effective, the lung being stretched between uncuttable apical and diaphragmatic adhesions with persistent positive sputum and open cavity and where thoracoplasty is out of the question (66). However, thoracoplasty need not be wholly out of the question before attempting this procedure because certain cavities from their situation in the mid-lung would require an extensive thoracoplasty to effect their closure and this is not justified if simpler measures can be safely adopted having an eye to the long term results.

Thorburn and Riggins (137) published their results with the combined treatment in 100 cases who had first pneumothorax, but in whom the cavity persisted and the sputum remained positive. In 44% cavity closure resulted and a further 19% closed their cavities after pneumolysis or thoracoplasty had been added.

In O'Brien's (139) series of 500 phrenic nerve operations, there were 145 in which the operation was supplementary to pneumothorax. Of these, 102 or 70.8% of cavities closed. There were 84 in which the pneumothorax was useless owing to adhesions holding open the cavity and of these, 42 closed, 36 became smaller, 4 were unchanged and 2 became larger.

The type of cavity present has more to do with the success or otherwise of the procedure than its situation in the lung. O'Brien (139) found that the small "moth-eaten" type of cavity showed the highest percentage of closure (90%). Thin-walled cavities were next with 58.8% closure. The irregular thick-walled cavities, associated with much fibrosis, did least well. Closure is hardly to be expected of such cavities unless they are very small (136).

Apart from the main indication stated above, this combined procedure finds a useful application where the cavity is situated in the lower lobe (140) and commonly at the apex of the lobe. Even after complete pneumolysis has been carried out, the cavity in this situation may resist closure and almost total pneumothorax may be required to effect closure. In order to obtain a more selective collapse, phrenic crush may be added and the pneumothorax kept more shallow. Selective collapse of the lower lobe then occurs more readily and there is less unnecessary collapse of healthy lung tissue. Thoracoscopy may show uncuttable adhesions in the paravertebral gutter between the chest wall and the region of the apical part of the lower lobe. Here again, phrenic paralysis may have the same effect as above, namely, to allow a selective collapse of the lower lobe to develop. The alternative would be a total pneumothorax with, even then, a chance of the cavity remaining open, or a basal thoracoplasty.

In a number of cases in this hospital, not necessarily with basal disease, we have combined pneumothorax, phrenic crush and pneumoperitoneum successfully in obtaining the closure of persistent cavities where

the condition of the other lung has precluded thoracoplasty or where the position of the cavity in the lung has not been ideal for closure by the major operation.

Pneumothorax plus phrenic paralysis is contraindicated if the disease is of such extent that control is unlikely with these measures or if the pulmonary reserve is markedly depleted and also if thoracoplasty is preferable and possible. Little value can be expected if thick-walled cavities are present or if there is multiple cavitation or extensive destruction of the lung.

THE MANAGEMENT OF ARTIFICIAL PNEUMOTHORAX

A detailed description of the technique of artificial pneumothorax induction and subsequent refills is not necessary in this work. The method is well known, It may be mentioned that pneumothorax machines are of various types. The essential requirements are some method of introducing air into the chest or removing it in measured quantity and a manometer to measure the intrapleural pressure. The Peter Edwards machine has been in use in this hospital for a number of years. A fine needle with a small round side opening is used for induction and a larger one with a side slot near the end for refills. Local infiltration of the chest wall with 2% Procaine is used for the first few refills only. No other preparation of the patient is necessary. Except for visits to the X-Ray room and theatre, he is kept at rest for a few weeks after the induction.

Artificial pneumothorax is, ideally, a relaxation measure and compression of the lung is not aimed at except in certain specific instances to be mentioned later. The object of all collapse measures is to collapse diseased tissue with as little interference with normal lung as possible. In the case of pneumothorax, this is achieved most readily through selective collapse. As we have seen, diseased parenchyma shows a tendency to retract more than normal tissue so, provided that the pleural space is free and refills are given at the proper intervals, this selective collapse will be obtained. Unfortunately, adhesions are common and their most frequent site is over the diseased area which it is desired to collapse. Thus, if refills are given without pneumolysis, gradually the less diseased area of the lung will become more collapsed (usually the base) and contra-selective collapse is the result. Pneumonolysis is required in practically every case if selective collapse is to develop.

If the division of adhesions should prove impossible by internal pneumonolysis, the pneumothorax must be regarded as ineffective and, unless a good collapse can be obtained fairly soon, it should be abandoned in favour of some other collapse procedure. It may be that the adhesions will stretch and become cuttable later and therefore it is justifiable to wait for a few weeks for this to happen. It is, however, usually dangerous to attempt to stretch the adhesions by

giving large refills under positive pressure. The case may be considered a suitable one for open pneumonolysis although this is an operation rather rarely performed nowadays. Or an extrapleural strip may be carried out with fenestration into the pneumothorax space and refills thereafter continued. The pneumothorax may otherwise be abandoned and a limited extrapleural strip carried out which can later be converted into an oleothorax if obliteration of the space seems imminent. Lastly, thoracoplasty may be carried out after the lung has been re-expanded.

In the ideal case, with localised disease in the upper third of the lung, selective collapse may be expected to develop if refills are kept small and given fairly frequently. It is advisable to give not more than 250 to 300 c.c. at induction, to repeat this on the following day and again two days later. The pressures are kept negative. After the third refill, the patient can then be screened before each refill and the amount of air to be given depends on the findings. Thereafter, refills are given twice weekly until the required degree of collapse is obtained and then the interval is usually lengthened to one week.

If adhesions are noted, refills are given in the same manner, no attempt being made to stretch the adhesions but thoracoscopy and, if possible, division of the adhesions is carried out as soon as there is space to manipulate the instruments.

The amount of air required is very variable. The giving of small frequent refills is recognised as the best method of maintaining collapse. This has the advantage of producing the least "concertina effect" of alternate collapse and re-expansion of the lung (141). This can usually be carried out while the patient is in hospital but, as an out-patient, weekly refills are more convenient and hence larger amounts are usually required. With the passage of time the pleura often becomes less permeable to the pneumothorax gases making absorption slower and hence refills can be given at longer intervals. This is much more marked where there has been a pleural effusion - a common occurrence. To avoid causing discomfort, the maximum refill is usually about 500 c.c. although 600 c.c. or even 700 c.c. may be required in certain cases to maintain the optimum collapse.

It should be emphasised here that X-Rays taken at fairly frequent intervals (3 to 4 weeks while in hospital and 2 to 3 months when out-patients) provide the only sound guide to the progress of treatment. In addition, it is necessary to screen these patients frequently, preferably before and after refills to see that the collapse is properly maintained. Manometric readings then serve as a further check to keep the pressure sub-atmospheric and also to indicate when a lessening in the amount of air and a lengthening of the interval between refills becomes necessary.

Positive Pressure Collapse.

Although the ideal is to keep the pneumothorax at a negative, or less than atmospheric pressure, to avoid forcing down the more healthy areas of lung, occasionally it may be necessary to maintain the pressure in the pleural space somewhat positive. This means that the manometer registers a greater-than-atmospheric pressure in all phases of respiration. To do this, frequent refills are usually necessary.

Previously, this method was used to attempt to close cavities held open by adhesions but it carries the danger of tearing these adhesions at their pulmonary attachments with the risk of opening into tuberculous tissue and consequent empyema and broncho-pleural fistula. Nowadays, a freer use of internal pneumonolysis and earlier resort to other collapse measures when pneumothorax is seen to be ineffective have largely obviated the need for this unsurgical procedure.

It is occasionally used to attempt to "hold back" obliterative pleuritis. Where obliteration is gradually occurring from the base upwards and where the disease is apical, it may be possible to maintain a small pneumothorax space by this means for longer than would be possible with negative pressures.

As Rafferty (36) declares, thoracoplasty is now generally used for those cases which would formerly have had positive pressure pneumothorax. If thoracoplasty is not feasible, a combination of pneumothorax and phrenic paralysis may effect closure. As a last resort, if thoracoplasty is not feasible and a low respiratory reserve makes phrenic paralysis inadvisable, positive pressure pneumothorax may be justifiable as being less risky than uncontrolled disease.

Bilateral Pneumothorax.

Simultaneous collapse of both lungs is in frequent use today. The indications are the same as those for unilateral pneumothorax. As the collapse cannot, of course, be complete, it is best used for disease limited to the upper third of the lung so that a selective collapse can be obtained on each side (142). Respiration is then carried on by the lower lobes and breathing capacity is surprisingly little impaired.

As to which lung to induce first, usually that with the more advanced disease is chosen and, while this is being collapsed, the better lung may show enough improvement to avoid the necessity of collapsing it (102).

With regard to management, it is best to try to obtain selective collapse, with cavity closure, and to carry out pneumonolysis, if necessary, on one side before commencing the collapse of the opposite lung unless the disease in it is advancing rapidly, making delay dangerous. The optimum degree of collapse, the amount of air to give and the interval between refills must be worked out for each lung as a separate problem (118) according to the principles outlined for unilateral collapse. Both lungs may be refilled on the same day if necessary or on separate days if there is any tendency to dyspnoea. The interval between refills may be different on the two sides.

Accidents Associated with Pneumothorax.

Haemorrhage: A subcutaneous haematoma, through the transfixion of a subcutaneous vessel occurs occasionally and is easily controlled by pressure. Intrapleural haemorrhage from damage to an intercostal vessel or from a vessel in an adhesion is very much rarer. Screening beforehand avoids inserting the needle into lung tissue and careful insertion of the needle should avoid damage to intercostal vessels.

Emphysema: This may be due to injecting air into the tissues outside the pleural space. Careful technique ensures that no air is allowed to flow unless the manometer registers a free swing indicating that the needle is between the pleural layers. Cough following induction may force air along the needle track into the subcutaneous tissues and the emphysema may be quite extensive and uncomfortable. It clears up in a few days. Again careful induction technique avoids lateral movement of the needle and the risk of tearing the pleura.

Pneumoperitoneum: It may be necessary to insert the needle rather low to avoid adherent lung and it may pass through the diaphragm into the peritoneum. Banyai (143) records two such cases. Alexander (136) describes a case in which a paralysed dome of the diaphragm had risen so high into the chest that the needle was inserted into the stomach. Careful attention to screening and to the manometric findings should help to obviate these accidents.

Air Embolism: This is a serious and fortunately rare complication which may occur during induction or during one of the earlier refills when the lung is still near the chest wall. It seems to be due to puncture of the lung with the needle point which comes to rest in a pulmonary vein and air is then drawn into the vein from the apparatus. The needle may also cause a communication between alveoli and a pulmonary vein. In either case air enters the circulation and coronary or cerebral embolism may follow. Cardiac arrest or blindness may occur or paralytic symptoms which may pass off or remain permanently or death may ensue rapidly.

Pleural Shock: Sudden death may occur when the pleura is punctured by the needle at induction. Some say that this is really air embolism and the fact that it occurs when the lung is near the chest wall and a vessel is most likely to be damaged by the needle is in support of this. Vaso-vagal attacks may occur during the aspiration of pleural effusions and they are not necessarily associated with pain. These may be minor pleural shock phenomena.

Novocain Reaction: An occasional patient is encountered in whom a marked hypersensitivity to Novocain exists. Injection of the solution may be followed by restlessness, anxiety, excitability or delirium with weak pulse. Convulsions may follow leading to coma and death or death may occur rapidly from intravenous injection of the drug.

Superadded Spontaneous Pneumothorax: This may be caused by the tearing of an adhesion, the rupture of an unsupported superficial caseous focus or, what is probably more frequent, the tearing of the visceral pleura by the point of the needle and to the escape of air from the lung. Minor degrees of this are probably

frequent as, commonly, the degree of collapse is greater than would seem to be warranted by the amount of air given at the induction. This is not serious and the small hole in the pleura probably quickly closes as the lung collapses. In more serious degrees, however, valvular pneumothorax or tension pneumothorax results and is accompanied by marked collapse of the lung, dyspnoea, pain in the chest and shock. X-Ray shows a much-collapsed or airless lung and displacement of the mediastinum. Air mustbe withdrawn immediately and perhaps repeatedly or a needle may have to be left in the chest attached to a valve or an underwater seal to allow the air to escape.from the chest as it accumulates. Careful technique again minimises the chance of this occurring. Following the induction, the patient should lie quietly in bed and coughing should be discouraged. Barnwell (118) cites a case where a three-inch gash in the visceral pleura resulted from coughing at induction.

COMPLICATIONS

PLEURAL EFFUSIONS.

(a) NON-PURULENT:

An effusion of fluid into the pleural space is probably the commonest complication occurring in pneumothorax therapy (144).

The reported incidence varies considerably. For example, Fishberg (145) states that 50% to 70% of all pneumothoraces develop an effusion. Jennings (146) found 53.5% and Tak Eng (147) 68.3%. As Heaton (148) has pointed out, the reported incidence varies very much because of the different definitions of what constitutes an effusion. With frequent screening of the chest, it is probable that some pleural fluid could be detected in most, if not all, cases of pneumothorax at some time during the treatment.

The majority of effusions are serous or serofibrinous and remain so but a significant proportion develop into frank tuberculous empyemata and a smaller number become secondarily infected with pyogenic organisms. Transitory, symptomless, small effusions are unimportant, except for the slight degree of pleural thickening which may occur but, because every substantial effusion is a potential empyema and because of the undesirable late effects of even a serous effusion, every effusion should be evaluated as soon as possible in terms of its possible long-term results.

Causes - Various causes have been mentioned to account for the frequency of effusion as an accompaniment to artificial pneumothorax. These include the use of cold air for refills, the effect of cold damp weather, over-exertion, the dropping of novocain on the visceral pleura, the frequent puncture of the parietal pleura, too large refills, too high pressures. They have also been attributed to the increased permeability of the pleura and to the aspirating effect of the negative pleural pressure. This may be a factor in the ex-vacuo exudate which accompanies re-expansion of the lung in many instances.

According to Matson (119), the mere presence of air and the unphysiological intrapleural pressures are sufficient to account for some effusions. Hutchin-

son and Blair (149) suggest that effusion is often due to a small rupture of the lung and Simmonds (150) states that most effusions are due to actual cavity rupture which may occur following adhesion section. The latter also considers that the cause may be blood-borne infection of the pleura or direct spread of tuberculosis from the lung to the pleura.

Packard et al (151) have two categories of serous exudate:-

(a) Benign (appears early, is symptomless and disappears rapidly), and

(b) Tuberculous.

In spite of the list of subsidiary causes mentioned, most pleural effusions are true tuberculous pleuritides (57). Pinner et al (152) found tubercle bacilli in 12 of 15 effusions examined and this author quotes Oshima and Suzuki as having found tubercle bacilli by cultural methods in 90% of 20 effusions.

Pathology - The pleura shows a fibrino-cellular exudate; granulation tissue may develop with or without secondary caseation. There is always fibrotic thickening of the pleura and, especially if empyema develops, the collapsed lung may be enclosed in a rigid case of white scar tissue having the consistency of cartilage (57).

As regards the pathology of the underlying lung, the more active and acute the pulmonary lesion, the more frequent and the more serious the pleurisy (151). Thus, effusion occurs very frequently in acute pneumonic tuberculosis and is likely rapidly to become purulent. It is also frequent in broncho-pneumonic disease but less frequent in the less acute fibro-caseous type. The effusion seems to occur relatively early in the course of treatment when the lung lesion is still active. Thus Mattill and Jennings (144) found 26% of effusions occurring within the first month of treatment and 77% within the first six months.

Results of Effusion - The presence of an effusion may lead to any of the following undesirable results:-

(a) Thickening of the pleura. This occurs to some extent in all effusions but is more marked where the fluid has been allowed to remain in the pleural space for any

length of time. It makes eventual re-expansion of the lung difficult or impossible. This thickening of the pleura also applies to adhesions and these may shorten as the fibrous tissue contracts, thus dragging open cavities and causing re-activation of the pulmonary disease.

(b) Obliterative pleuritis may set in and the collapse of the lung may rapidly be lost.

(c) Over-collapse of the lung. Selective collapse of the upper portion of the lung may be abolished by a collection of fluid in the pleural space which collapses the relatively healthy basal portion. Thereafter, pleural thickening may prevent re-expansion of the lung or obliterative pleuritis may occur so that the whole space is lost. This may be very important in bilateral collapse where selective collapse is essential.

(d) Fluid may cause unusual tension on adhesions situated over diseased portions of the lung. These may tear leading to the formation of broncho-pleural fistula.

Some of the following effects may be less undesirable:-

(a) The encouragement of fibrosis in the lung. If this were confined to the area of disease it would be of benefit in hastening healing. However, bands of fibrous tissue may dip into the lung from the thickened pleura along the pulmonary septa (57), making the lung incapable of ultimate re-expansion.

(b) There may be a beneficial effect from auto-inoculation. This is not measurable but may do good.

(c) In rare instances, the development of effusion may cause a basal cavity to collapse.

(d) The pleural thickening may stiffen an unduly mobile mediastinum. More "compression" may

serous, persist for some time, stabilise and gradually resorb, it may lead to the undesirable effects mentioned and it does, in a proportion of cases become eventually frankly purulent. Packard (151) noted that 10 - 20% of relatively mild effusions became purulent.

Management - The benign effusion can usually be left entirely alone. Refills may be suspended while the fluid is present and, afterwards, may require to be given less frequently. If the fluid is causing an unwanted collapse of the base of the lung, it may be replaced by air.

In the severe toxic type the fluid should be aspirated for diagnostic purposes and, if it tends to become opalescent or frankly purulent, the condition should be treated as an empyema.

The intermediate type must be treated on general principles. It is agreed that it is best not to aspirate during the acute febrile phase except for diagnostic purposes. Fluid must be removed if there are pressure symptoms, such as pain in the chest, dyspnoea, or if screening shows that there is shift of the mediastinum to any marked degree.

If fluid develops, as it usually does, early in the course of treatment, it must be decided whether or not to continue with the pneumothorax. Bearing in mind the remote ill effects of certain long-standing effusions, it may sometimes be better to abandon the pneumothorax and proceed to thoracoplasty if collapse therapy should still be required. On the other hand, most pneumothoraces can be continued after aspiration and air replacement. Fluid should not simply be left just because there are no symptoms. The question of pleural thickening and consequent inability to re-expand the lung is important. Pneumothorax was designed as a temporary measure of collapse but if fluid is left, the lung may never expand requiring indefinite refills for the patient's comfort or the space may fill up with fluid which may become purulent, requiring eventual thoracoplasty when the condition of the patient may not warrant it.

Any effusion should be aspirated shortly after the acute stage subsides. This should be within a week or so. Aspiration may have to be carried out repeatedly before the effusion disappears. Care should be taken to keep the collapse as shallow as is compatible with safety in case the pleura thickens and re-expansion becomes impossible. If fever is prolonged, the pleural space should be kept as dry as possible and the collapse kept shallow, in case an empyema is developing and it becomes necessary to re-expand the lung without delay.

A watch may also be kept on the cytology of the fluid. If the cells go on increasing in number or if the fluid becomes obviously opalescent and, especially if the temperature be prolonged beyond a week or two, the development of empyema should be expected, the collapse kept shallow and re-expansion thought of. In addition, streptomycin or para-aminosalicylic acid can be given in the hope of preventing an empyema.

The effusions occurring later in treatment, usually are relatively benign and may be left to fill up the pleural space which is commonly partially obliterated by this time. The terminal ex vacuo effusions are really a sign of inexpandable lung. They fill up what is left of the pleural space and act as a cushion over the lung indefinitely. They may, however, eventually change into empyemata and the patient should be kept under observation for a prolonged period. Patients with such effusions in localised walled-off pleural pockets usually do very well.

There is thus no routine treatment of pleural serous effusion. The weight of evidence is in favour of removing fluid frequently enough to prevent any of its harmful sequelae (118).

(b) PURULENT:

Pure tuberculous empyema may be defined as the presence of sero-purulent or purulent fluid in the pleural space containing tubercle bacilli demonstrable on direct smear or by using cultural or biological methods. Mixed infection empyema occurs when, in addition, other pyogenic organisms gain entrance to the pleural space, either through a broncho-pleural fistula or by introduction from without.

The occurrence of tuberculous empyema constitutes a real obstacle to the successful use of artificial pneumothorax.

Incidence - This varies in numerous reported series (136, 144, 153), but it probably occurs in from 5 to 20% of all patients in whom pneumothorax treatment is used. In the present series of 100 consecutively induced pneumothoraces, this complication was present in 12 cases, 10 accompanying pneumothorax and 2 following extrapleural strip with fenestration into the pleural space.

There are two aspects to be considered when discussing the incidence of empyema:-

(a) The state of the collapse.

Matson (119), in a series of 480 cases of pneumothorax treatment, found that empyema was just as common in the "satisfactory collapse" group as in those cases with partial collapse and adhesions under tension. Simmonds (150), on the other hand, stated that effusions of any kind are less frequent if the lung is freed adequately by adhesion section. Cutler's series of 476 cases showed definitely that empyema was nearly four times as frequent in the presence of a mechanically ineffective collapse as in those cases with effective collapse of the lung (154). The proportion in the present small number of cases was approximately three to one in favour of ineffective collapse.

(b) The state of the underlying pulmonary disease.

Few patients with minimal disease develop empyema but, as the pulmonary lesions become more extensive and acute, the number with empyema increases (144). Cutler's series, mentioned above, showed a much higher incidence associated with far-advanced disease.

The association of empyema with acute pneumonic tuberculosis in which artificial pneumothorax is induced during the acute phase is well known. In extensive cavernous disease, empyema is relatively common and

common, too, is its association with attempts to collapse large apical cavities with pneumothorax.

The association of empyema, pneumothorax and stenotic bronchial tuberculosis has been discussed in a previous section.

Prognosis - The literature does not allow any exact conclusions regarding the effect of empyema on the prognosis (36), but there is no doubt that it has a distinctly unfavourable effect, especially from the long term viewpoint. One must consider the risks from the presence of the empyema itself and also those of the (usually) uncontrolled pulmonary disease. As regards the empyema there are the following risks:-

(a) Long-continued suppuration leading to amyloid disease.

(b) The occurrence of broncho-pleural fistula with possible spread of the thin purulent fluid through the bronchial tree and consequent widespread extension of disease. There is also the grave possibility of superadded infection of the pleural space with pyogenic organisms. This does not happen in every case. For example, Woodruff (153) found that 12 of 47 patients with known broncho-pleural fistulae did not develop mixed infection.

(c) A collapsed permanently inexpandable lung.

(d) The effect of the long-continued toxaemia.

The present survey agrees with Rafferty (36) that the pneumothoraces which furnish most of the unfavourable data in statistical tables are those cases which should have been re-expanded and listed as exploratory measures.

Pathology - The accepted explanation for pleural infection is active tuberculous disease of the visceral pleura, itself infected from the lung by direct extension of the disease or via the sub-pleural lymphatics.

According to Matson (119) and also Penington (155), empyema usually arises from ulceration into the pleural space of a sub-pleural caseous focus and I find it surprising that this does not occur with greater frequency considering the fairly frequent observation of these foci "pointing" through the visceral pleura at thoracoscopy. Spontaneous pneumothorax and the stretching and tearing of adhesions are other possible causes (119, 156).

Simmonds (150), from his study of 250 cases of cavitation, concludes that all effusions, whether clear or purulent, are due to the rupture of cavities. In Woodruff's series of 154 empyemata, there were 47 known persistent broncho-pleural fistulae (153).

Classification - Empyemata can be classified in various ways but the clinico-pathological classification of Packard et al (151) is the most generally useful. It is as follows:-

(a) Mild or benign tuberculous empyema.

(b) Severe tuberculous empyema.

(c) Severe tuberculous empyema with secondary infection.

(d) Empyema, frequently secondarily infected, originating in a broncho-pleural fistula.

Onset - Although empyema may develop rapidly as, for example, in pneumonic tuberculosis or with broncho-pleural fistula, there is practically always an initial phase during which the fluid present is serous in nature. In most cases, this thin serous fluid is present for weeks or months before becoming purulent and, therefore, a point in the prevention of empyema is to aspirate fluid early and to endeavour to keep the pleural space as dry as possible.

Packard et al (151) state that 10 - 20% of all serous effusions become purulent and Eglee and Wylie (157) noted that most empyemata develop slowly from pre-existing simple effusions.

Pus can form in various ways:-

(a) Gradually, as has been said, from a persistent serous effusion. No special symptoms are noted and the change in the nature of the fluid is noted at aspiration.

(b) Rapidly, on top of an acute pleurisy with effusion. A severe initial onset of serous effusion and prolonged fever point to the likelihood of such effusions becoming purulent.

(c) As a sequel to broncho-pleural fistula.

In Eglee and Wylie's series of 72 pure tuberculous empyemata, 58 developed from serous effusion incident to pneumothorax treatment, 3 followed spontaneous pneumothorax, 2 occurred after closed pneumonolysis and 9 occurred during "neglected re-expansion", i.e. without close observation (157).

Prevention - Shipman (158), over a period of 4 years, managed to avoid this complication altogether by observing the following rules:-

(a) Always have an initial rest period before inducing pneumothorax except where immediate collapse is dictated by a haemorrhagic emergency. This avoids the use of artificial pneumothorax in the acute phase of pneumonic tuberculosis.

(b) The use of frequent, small refills at subatmospheric pressures. There must be no "compression" of the lung.

(c) Early abandonment of pneumothorax unless it is effective or can rapidly be made so by the section of small adhesions. This is the most effective preventive measure (36).

(d) The stopping of refills if fluid forms, aspiration of fluid and, if it is persistent, the abandonment of the pneumothorax.

While these are sound rules and have proved their usefulness, it has been pointed out that their application may mean denying effective treatment to certain cases (36). The aim of treatment is the control of the parenchymal disease and, in certain cases, the only feasible treatment may be pneumothorax with its risk of empyema.

In advanced or even moderately advanced disease, it may be better from the start to consider some form of collapse procedure which does not involve the separation of the pleural layers (121). One must be prepared quickly to expand the lung in the presence of tension cavity lest rupture of the cavity occur.

Eglee and Wylie (157) have recommended more "individualization" of pneumothorax to prevent empyema. They are rightly against any form of standardised treatment. This is another way of saying that every case must be carefully studied and, if collapse therapy is required, the type of collapse suitable to the particular circumstances must be chosen. Real prevention of empyema begins here (36). This choice of cases has already been discussed under indications and contra-indications.

Further, it should be remembered that close observation is required from start to finish of treatment and for some years thereafter. In this way the use of pneumothorax in unsuitable cases will be avoided. Pleural effusions will not be ignored and change of treatment can be decided upon, if necessary, at the optimum time.

Active Treatment of Empyema - When empyema develops in any case undergoing pneumothorax treatment, there are several factors to be considered before undertaking any plan of management. The first of these is the effectiveness or ineffectiveness of the collapse and the second the nature of the infection. Also important are the condition of the opposite lung and the presence or absence of a broncho-pleural fistula.

For the purposes of treatment empyemata may be divided into two groups, the pure tuberculous and the mixed infection empyema:-

Mixed Tuberculous Empyema - This is generally due to the occurrence of a spontaneous pneumothorax on top of a therapeutic pneumothorax. In other words, a broncho-pleural fistula is present (159). Coryllos would have it that all empyemata are due to the presence of a broncho-pleural fistula, mixed infection only taking place in the larger ones (156). Mixed infection may follow the unwise cauterisation of adhesions, especially when partially severed adhesions are left "on the stretch" and tearing takes place through tuberculous tissue leading, it may be, to spontaneous collapse. Again, the

infection may take place during an attack of "influenza" or follow unsterile aspirations of a simple effusion.

However it occurs, this complication has to be treated as an emergency. The prognosis is grave and treatment, if it is to be successful, must be prompt.

In those cases without a demonstrable broncho-pleural fistula, medical measures such as wash-outs with various anti-septics and the employment of chemo-therapeutic agents may be successful in reconverting the condition into one of pure tuberculous empyema. Roberts et al (160) have had some success in cases where the contaminating organism was sensitive to Penicillin, although the mechanical presence of large fibrinous deposits may interfere with aspiration and make it necessary to drain the pleural space surgically following rib resection. Petroff (161) has used sodium tetradecyl sulphate with success in these cases.

When, as is usually the case, a demonstrable fistula exists, tube drainage, with or without rib resection is required. This provides efficient drainage and may in some cases encourage the lung to expand and thus to obliterate the infected pleural space. If the opposite lung is sound, closure of the pleural space by extensive thoracoplasty should be proceeded with as soon as the condition of the patient will allow. Aspiration and instillation of Penicillin may avoid the necessity of carrying out a thoracoplasty at a dangerous stage for the patient (162). If there is disease in the opposite lung, drainage of the empyema plus rest and possibly Streptomycin or P.A.S.* treatment may eventually render the patient fit for a later thoracoplasty to obliterate the empyema. The period of delay should be as short as is compatible with safety as the considerable pleural thickening which takes place in the interval makes such obliteration very difficult.

Brock (163) published an illuminating account of the treatment of these cases before the days of Penicillin and Streptomycin. Thirty-five cases had drainage only. Of these, 32 died and 3 are alive. Forty-nine cases were treated by combined drainage and thoracoplasty, 14 of these died and 35 are alive, 15 of these without a sinus. The above author believed that persistent fever was rather an indication for operating than for temporising with less radical methods.

* para-aminosalicylic acid

Persistent sinus following such treatment is a complication in some cases but with further plastic operations to obliterate the small residual space and the use of Streptomycin and, recently, para-amino-salicylic acid, these should be reduced to a minimum.

Pure Tuberculous Empyema - There still exists some difference of opinion on the subject of treatment for this condition.

Chandler (164), the extreme conservative, insisted that these effusions should be left alone unless there was some very good reason for interference and quoted 12 cases who had considerable collections of tuberculous pus in their chests for up to 20 years, and who remained in good health. No details were given of the state of the collapsed lung although it may be inferred that that was under control. It would be of interest to know what proportion of empyema cases end up in this relatively happy although precarious state. Some would agree with Chandler and most tuberculosis physicians have at least a few such cases, most with abandoned pneumothoraces and quiescent pulmonary disease, with localised collections of tuberculous pus in their pleural spaces and apparently in good health and fit for their occupations. But Woodruff (153) strikes a jarring note with the statement that of 154 such cases observed for 3 to 8 years, no less than 30% suffered perforation or secondary infection.

At the other extreme is Coello (165) who states: "Everyone will agree that in a case of tuberculous empyema the first step is to encourage by all means the expansion of the lung and the obliteration of the pleural space". With this Rafferty would agree.

Some years ago the recognised treatment (136, 147, 166) was first to give a thorough trial to such non-surgical measures as aspirations, wash-outs and instillation of various anti-septics in an attempt to stop the formation of pus and to continue the pneumothorax, but even then it was recognised that such treatment, even if energetically pursued, might fail because of the inadequate collapse of the pulmonary lesions responsible for the condition. Resort was then had to oleo-thorax or to phrenic nerve operations to reduce the size of the pleural space or, finally, to thoracoplasty. It was recognised that thoracoplasty should not be too long delayed lest so much pleural thickening took place that the pleural space could not readily be obliterated (151).

Such treatment of even the so-called "benign" empyemata in Packard et al's series led to very poor results. Of 32 such cases, 16 died, 6 from extension of disease to the opposite lung, 5 from extension in both lungs and 5 from pulmonary perforation. Of the other 16 cases, 9 were "cured" but onethird only were working.

A further interesting comparison was made in an article by Mattill and Jennings (144) from which the following table was taken:-

Treatment	No.	Working	Alive	Dead	Empyema at death	Empyema cleared
PNX contd.	11	3		8	4	
PNX discontd. no further collapse	36	8	3	25	21	15
Phrenic	11	5	1	5	1	10
Surgical collapse	60	24	16	20	10	49
Catheter drainage or rib resection only	13			13	13	
Total:	131	40	20	71	49	81

These authors recognised that such cases may do well for a time but the risks which have been mentioned before are always present. Aspirations and irrigations they found of little value in curing the empyema although by their use toxaemia might be reduced and the empyema pocket kept clean. Treatment, they said, should be directed towards cavity closure and, for this, re-expansion and surgical collapse offered the best chance. The fatality rate was high for those who were unfit for surgery.

To attempt to clarify a rather obscure position, the following principles may be put forward:-

(a) If the underlying pulmonary disease is uncontrolled by the pneumothorax, then it is useless to concentrate treatment on the empyema which is present. Control of the disease in the lung is what is aimed at. To continue pneumothorax in the face of pleural infection is courting disaster and the sooner the pneumothorax is abandoned and the lung re-expanded the better. Some other form of collapse therapy, usually thoracoplasty, can then be undertaken if the opposite lung is clear of disease or if the disease in the other lung is quiescent or under control. The surgical collapse then has the dual purpose of controlling the lung disease and obliterating the empyema space (167).

(b) If the pulmonary lesion is controlled, it is reasonable to attempt to dry up or render serous the effusion by frequent aspirations and wash-outs and the use of, for example, Azochloramide-T (sodium tetradecyl sulphate) in the hope of continuing refills. This may be possible but, even here, pleural thickening and eventual inability to re-expand the lung may be the outcome and so, if the effusion takes place, as it usually does, early in the course of pneumothorax treatment, it must be considered whether it is not wiser to re-expand the lung while it is still possible and substitute a more selective and permanent collapse by thoracoplasty. In certain cases of empyema occurring towards the end of treatment where the pleura is much thickened and the pus walled off, it may be permissible to leave it alone.

(c) Decisions as to the effectiveness of the collapse and as to the likelihood of medical treatment proving effective in the treatment of the empyema should be made early so that, if desired, re-expansion of the lung can be carried out.

(d) The best treatment for tuberculous empyema is prevention. Apart from those preventive measures connected with the proper selection of cases for pneumothorax, there is reason to believe that Streptomycin is of value in preventing its occurrence (168).

ADHESIONS.

The presence of adhesions is not so much a complication of pneumothorax treatment as something to be anticipated in practically every case and to be dealt with as part of the plan to obtain as soon as possible a good selective collapse of the diseased area.

An account has been given of the pathology of adhesions in a former section and some attempt made to classify them according to their structure. What is of concern at this stage is whether they are cuttable or not. If they are cuttable, an inadequate, contra-selective collapse can be converted into one in which the diseased portion of the lung is selectively and concentrically relaxed (102). If they are not cuttable and are not likely to become cuttable by gradual stretching within a relatively short time, then the pneumothorax may have to be supplemented by other procedures or given up in favour of some other form of collapse therapy.

Although many adhesions can be seen on the X-Ray and fairly accurately localised by this means, the only way to decide whether they are cuttable or not is to introduce the thoracoscope and inspect them closely. Matson (119), who uses the electro-surgical cautery, has found that intrapleural pneumonolysis will convert approximately 70% of cases of unsatisfactory pneumothorax into satisfactory ones.

The method adopted here is to use the galvano-cautery with two cannulae, one for the thoracoscope and the other for the cautery. The procedure is essentially that described by Jacobaeus (169). No attempt has been made to enucleate adhesions from the chest wall using the technique of Maurer (170), although in his hands this method has led to some spectacular results.

Indications for Adhesion Section - Thoracoscopy should be carried out in all cases where the degree of collapse is inadequate to control the lesions present. It is especially necessary for the persistence of cavity and sputum which is positive for tubercle bacilli. Matson has also noted that paroxysmal cough, pain in the chest and febrile reactions after refills may be due to adhesions. The presence of such adhesions may be unsuspected on the X-Ray picture and so thoracoscopy may be justified in the presence of the above symptoms. Laird (171) advocates inspection of the pleural cavity in all

cases, but this does not seem to be necessary where a good collapse is obtained and no adhesions are seen on X-Ray or screening.

Pneumonolysis should not be attempted:-

(a) In the presence of tuberculous empyema.
(b) In the presence of acute febrile pleurisy with effusion.

It is doubtful if it should be attempted:-

(a) Where adhesions are relatively large and attached to large peripherally placed cavities.
(b) Where extensive adhesions make success unlikely.

Complications - Haemorrhage is the complication most to be feared. This may occur from damage to intercostal vessels on introducing the trochar and cannula or when actually cutting the adhesion or from injuring the great vessels in the cupola, especially if these are obscured by the nature of the adhesions or pulled into abnormal positions. Matson reported 9% of haemorrhagic effusion in his series of 249 cases. Blood appeared in varying amounts but he had only 3 cases where the haemorrhage at the time of operation was of an alarming extent. It is likely to be a complication in a higher proportion of cases where the more heroic attempts to sever extensive adhesions are used. There were no cases of serious bleeding in the present small series, probably due to the very cautious attitude adopted.

Effusion may be expected to occur following section of adhesions. This is commonly small in amount and serous in nature being transient. Empyema, however, may follow the section of short adhesions and no doubt tuberculous tissue is frequently cut through by the cautery. The greatest danger seems to lie in the partial section of fairly thick adhesions which contain lung fairly far peripherally. Tearing of such partially severed adhesions may take place later as a result of coughing and tuberculous tissue or even a cavity may be opened into. Brock (163) found 1.4% of tuberculous empyemata and 1.4% of mixed infection empyemata occurring in 442 operations.

Considerable emphysema is fairly frequent after the operation, due to uncontrolled coughing. This may lead to considerable loss of pneumothorax space. This should be obviated by careful adjustment of the intrapleural pressures before the patient leaves the theatre.

Various injuries to nerves have been reported and Horner's syndrome has been described as occurring.

Results - Good results depend on the completeness of the adhesion section. Many series have been published to show the value of internal pneumonolysis as an adjunct to pneumothorax treatment. Usually the earlier the adhesions can be severed the better, as selective collapse can be obtained and the operation is carried out before the adhesions become thickened by the presence of pleural fluid. In the present series it was customary a few years ago to wait for from 3 to 4 months, but now thoracoscopy is carried out as soon as there is space for the introduction of the instruments, a state of affairs obtaining commonly within 3 to 4 weeks. Thoracoscopy is also carried out as early as possible where there is any doubt of the safety of proceeding with the pneumothorax.

As pointed out by Goorwitch (172), the only results of internal pneumonolysis itself are:-

(a) Alterations in the anatomical nature of the collapse, and

(b) complications.

Changes in the status of the disease and cavity and sputum conversion are results of the continuation of pneumothorax therapy.

Goorwitch (172) recently reviewed the literature on internal pneumonolysis and found the following complications:-

Small intrapleural haemorrhage	...	1.2 to 4%
Large " "	...	0 to 2.3%
Spontaneous pneumothorax	...	0 to 4%
Broncho pleural fistula	...	0 to 2%
Small serous effusion	...	5.5 to 40%
Large " "	...	3 to 23%
Pure tuberculous empyema	...	0 to 5.5%
Non tuberculous pyogenic empyema	...	0 to 1%

Mixed tuberculous empyema	...	0 to 2%
Loss of pneumothorax space	...	0 to 2%
Death	...	0 to 8.7%

The incidence of complications was greater in the early years following the introduction of the procedure and dependsto some extent on the clinical material and the judgement and skill of the operator.

Day et al (173), in a series of 1,000 closed adhesion sections, found fairly similar complications, falling within the above-noted limits. In 59.5% of cases there were no early post-operative complications (within 4 weeks of operation).

DURATION OF COLLAPSE.

In 1930, Rist (173), from a study of his cases treated between 1921 and 1927, concluded that a too long period of collapse was as bad as one that was too short. He recommended a minimum of 4½ years and, in fact, most of his cases had collapse for 5 years.

Various authors (174, 175, 176) up to about 10 years ago were advocating a minimum period of collapse of 2 to 3 years, although it was recognised that certain cases required longer. Dufault and Laroche (174), in fact, suggested 5 to 7 years for fibro-caseous lesions. Dundee (177) pointed out that the time should be measured from the last positive sputum and the disappearance of cavitation and that among the important factors to be considered were the extent and nature of the original lesion. He recommended that some pneumothoraces should be kept up permanently.

Todd (178), in a survey of the position in 1938, noted that general statements of the time required could be misleading and emphasised again the importance of the state of the lung before collapse. He gave certain minimum periods on a basis of extent of disease and pathology.

Thus:

Early small infiltrates with no symptoms	...	less than 2 years
Fibro-caseous disease without cavity	...	3 years at least

Acute broncho-pneumonic disease with much toxaemia	... None less than 4 years.

Among the more recent reports, Hurst and Schwarz (179) found relapse much less frequent where collapse was continued for 4 or more years than in those whose duration was 3 years or less. However, Aycock and Keller (180) found that the incidence of relapse was just as great after collapse for 3 to 4 years as after collapse for only 1½ to 2 years. They felt that prolonged and indefinite collapse periods presented definite disadvantages, the chief being complications such as marked pleural thickening, unexpandable lung and right heart hypertrophy and failure. As has also been pointed out by Morriss (181), earlier re-expansion avoids certain late complications and leads to better anatomical restitution.

From a consideration of the foregoing, it can be seen that no hard and fast rules can be made. Accumulated experience shows that a period of 3 to 5 years is usually adequate but, as always in collapse therapy, the individual case requires careful consideration. Nowadays, the tendency is to use permanent collapse measures for disease which requires permanent collapse. Thus, many cases are recognised ab initio as not being suitable for pneumothorax which, in earlier years, would have had this form of treatment and which would have required prolonged or indefinite refills. Nowadays, also, adhesions are severed if possible and, where this cannot be done, the pneumothorax is generally given up and thoracoplasty substituted where the condition of the other lung makes this possible.

The indications for pneumothorax have been narrowed considerably in recent years and the relative safety of thoracoplasty and resection recognised. It is no longer necessary to induce pneumothorax in cases unsuitable for it or to continue its use in cases where it is seen to be ineffective. Thus there should be little need for greatly prolonged pneumothorax (36).

It must be emphasised that what is now under consideration is voluntary cessation of satisfactory pneumothorax. In many cases considered suitable, no space can be found initially. In a further proportion, a satisfactory collapse cannot be obtained because of the existence of uncuttable adhesions. In others, re-expansion is considered advisable because of the development of empyema and in some loss of the pneumothorax space occurs

from pleural obliteration. So that what is left is a select group in which satisfactory and clinically effective collapse has been maintained. One has then to decide when it is safe to allow re-expansion. An estimate has to be made for the individual case, the duration of collapse being taken to date from the time when it became effective with reference to permanent conversion of the sputum and the closure of all cavities.

Taking 3 years of effective collapse as a minimum, one can at the end of this period review the case from the beginning. All the X-Rays should be available at this time. The original extent of the disease and its type, together with the size and location of cavities should be considered. The more extensive disease and larger cavities need longer collapse as does the more fibrotic disease in comparison with the more exudative. A rapidly effective selective pneumothorax for originally limited disease need not be prolonged beyond the minimum. A collapse which was slow in leading to cavity closure and sputum conversion requires longer collapse. The condition of the other lung needs evaluation. If the disease here has been apparently static, it can be ignored in deciding on re-expansion but, if there has been obvious activity, collapse may need prolongation. If it is progressing, so that collapse is required for its treatment, it may be a reason for rather shorter collapse on the side originally treated and now under consideration.

The presence of complications during the period of collapse may modify its duration. Certain complications, as has already been discussed, may indicate that the collapse should be abandoned early, in which case the collapse, being ineffective, is outside the scope of the present discussion. Febrile effusion during the course of an effective pneumothorax probably indicates that a rather longer collapse is needed.

The mentality of the patient must be considered. To most patients, the continuation of the collapse gives a sense of security. Others are only too glad to have it stopped. Those who have never quite learned to live the restricted life required had better have a longer rather than a shorter time of collapse. Conditions at home and as regards employment and finance should be as stable as possible before re-expansion. No re-expansion should take place during pregnancy and probably not for six months thereafter.

Unfortunately, we have no means of verifying whether the disease in the lung has healed or not. The only way to find out is to re-expand after a time which seems suitable in view of the needs of the particular person. If one is in doubt, it is better to err by keeping the refills going somewhat longer than necessary.

RE-EXPANSION.

Although some writers (179) favour abrupt cessation of refills, a better method is probably to allow slow expansion to take place (36, 121, 151, 181). If refills are given in diminishing amounts and at lengthening intervals the lung, provided it can expand, gradually resumes its function. X-Rays can be taken at frequent intervals and any sign of re-opening cavity or reactivation can serve as a signal to resume refills. Aycock and Keller (180) did not find that exacerbation occurred in any of their cases under 12 months following re-expansion. Rafferty has pointed out that the re-opening of a cavity during re-expansion probably indicates that the pneumothorax was never really effective and that a permanent form of collapse is required (36).

The gradual method of re-expansion is said to avoid highly negative pressures in the pleural space and to discourage the formation of pleural fluid (36). In the small personal series of re-expansions carried out at the clinic pressures have seldom been highly negative, except with unexpandable lungs but more or less terminal effusion has been practically invariable.

As regards this terminal effusion, the fluid may be aspirated a few times to encourage complete expansion but usually it is a sign of unexpandable lung and, if serous, should be left alone. If small in amount, it commonly fills the space left over the summit of the lung forming a semi-permanent cushion and gradually being reabsorbed and becoming organised (151). Rafferty, however, advises aspiration in these cases.

UNEXPANDABLE LUNG.

Where a small selective collapse has been maintained, re-expansion usually takes place smoothly and relatively quickly and with fairly complete restoration of the lung to its normal anatomical relationship. With

long-continued collapse, especially where there has been serous or purulent effusion, re-expansion, owing to thickening of the pleura may be very incomplete. The intrapleural pressures become very negative and the mediastinal structures dislocated towards the side of the collapse. In some cases, and if the dislocation is not marked, no complaint may be made by the patient but, if dislocation is marked, respiratory embarrassment and symptoms of right heart failure may make their appearance. Phrenic paralysis may help to diminish the pleural space into which the lung must expand, but thoracoplasty may be required to "straighten out the mediastinum".

In certain cases with a much-thickened pleura and rigid mediastinum, a permanent air space remains and one is faced with continuing refills permanently or obliterating the pleural space with a thoracoplasty. Usually such permanent pleural spaces fill with fluid and if this is serous in nature it can be left alone. If it is purulent, however, thoracoplasty is probably indicated in view of what has been said before of the danger of leaving a permanent collection of pus in the pleural space.

A further alternative exists in the possibility of carrying out a decortication of the inexpandable lung (182). While this may be unwise where there has been extensive pleural or parenchymal disease, it is of help where the disease has been confined to the upper lobe. The lower lobe can then be decorticated and allowed to re-expand, but no attempt is made to remove the fibrous covering from the diseased area which then remains collapsed.

CHAPTER FIVE

RESULTS AND CONCLUSIONS

RESULTS

There is now no doubt that a programme of treatment including all the various forms of collapse therapy leads to better results in the long run than one relying entirely on unaided sanatorium treatment. The table reproduced on the following page shows this adequately:-

The evaluation of any form of treatment in pulmonary tuberculosis is very difficult, for the following reasons:-

(a) Tuberculosis is a chronic disease and, therefore, a sufficiently lengthy observation period after treatment is essential. A certain percentage of cases is lost, thus influencing the results.

(b) Tuberculosis, in a number of cases, tends to heal with or without special treatment.

(c) There is great difficulty in finding suitable controls. In any two groups to be compared, numerous factors must be matched. These include the following: the extent of disease, its pathological type and distribution; the size, number, location and type of cavities; the age, sex and race of the patient; his economic and social conditions and his temperament. Thus a large series of cases is required, so large as to be beyond the experience of any one observer.

An approach to the problem may be made in two ways:-

<u>The Statistical Method</u>:

Large numbers of patients treated by collapsing the lung are compared with a similar number not having such treatment. Comparison is inaccurate because:

(a) Details of the treatment vary among the different groups included.

(b) The observers vary as to their degree of optimism or the reverse.

T A B L E

Comparison between results at time of discharge from two sanatoria that used an exceptionally wide variety of collapse therapy operations and 278 sanatoria that used but little collapse therapy.

SANATORIUM	No. of Pts.	Stage of T.B. at admission			Use of Collapse Therapy	Results				
		Min.	M.A.	F.A.		A or A.A.	Q.	Imp.	Unimp. or Worse	Dead
		%	%	%	%	%	%	%	%	%
Michigan State	823	10.9	31.9	57.1	72.3	47.3	13.8	6	15.4	17.5
Jackson County	109	12	28	59	48.4	54.9	15.7	11.9	0.9	18.3
278 Others	42,107	16	30	54	10	17	41		19	23

Min. : Minimal
M.A. : Moderately advanced
F.A. : Far advanced.

A. : Arrested
A.A. : Apparently arrested
Q. : Quiescent
Imp. : Improved

(Alexander, J. The Collapse Therapy of Pulmonary Tuberculosis, Springfield, 1937).

(c) A method of treatment likely to do good is not usually withheld from those patients whose condition appears to warrant its use. The treated cases thus become a select group.

(d) Such cases as refuse treatment are not good controls, either, as they are likely to be more irresponsible and less co-operative about the regime without collapse therapy.

Patients treated in the pre-collapse era may be compared with those receiving modern treatment. Apart from the collapse treatment, however, more emphasis is now laid on prolonged rest than formerly and there is also to some extent the factor of earlier diagnosis.

As examples of the statistical method there are the following:-

(a) The report of the Joint Tuberculosis Council (183). This compared the survival rates of 3,021 cases of pulmonary tuberculosis treated in 42 hospitals by artificial pneumothorax with 1,329 cases from the same hospitals where pneumothorax was attempted but failed. The ratio of actual to expected deaths among the treated group was half that of the failures but it was admitted that the controls were really unsuitable for accurate comparison. These results were then compared with 2,750 cases treated at Midhurst Sanatorium where approximately 1% received pneumothorax treatment. The ratio of actual to expected deaths was twice as high in the pneumothorax group as in the Midhurst group. At Midhurst, however, the patients came from a different social environment which introduced a factor of great importance in making any comparison between the groups.

(b) Bentley's (34) report of the experience of the London County Council compared 677 pneumothorax cases with 3,309 cases having only conservative treatment. Correction was made for age, sex and clinical classification. The rate of survival was 20% higher in the pneumothorax group but pneumothorax was only applied to 10% of all patients. Thus an improvement all round of only 2% was noted

with pneumothorax treatment. The prognosis was found to be best in those having complete collapse of the lung and in those leaving hospital negative.
Bentley pointed out the difficulties in assessing results. These were caused by:

(i) Paucity - conclusions from too few cases.
(ii) Selection - writing up only certain cases.
(iii) Precipitancy - too short follow-up.
(iv) False control - e.g. comparing pneumothorax with those having no space.
(v) Equivocation - lack of definition of collapse.
(vi) Failure to trace - losing sight of a high proportion of cases.
(vii) Lack of detail - not adequately describing the extent of disease.

The foregoing reports are now 13 years old and refer to methods of treatment which are, in many respects, quite different from those in use today. Nowadays, the indications for pneumothorax are becoming narrowed down. Surgery has made great strides and resort to it comes earlier than formerly. There is wider use and a more efficient performance of pneumonolysis than formerly. Ineffective pneumothorax is now given up early, thus minimising the occurrence of complications associated with its continued application. There is more insistence on strict bed rest. Pneumothorax is more and more taking its proper place in the comprehensive programme of treatment of pulmonary tuberculosis.

Rafferty (36) quotes two examples to show the difficulty of employing the statistical method. One is that of Soderstrom (184) in which the non-collapse group is compared with the collapse group. But the collapse group usually have the worse prognosis. In this series, the non-collapse group had a 5-year survival rate of 98%. In other words, many of these cases must have been quiescent in the first place. In the collapse group the survival rate was 89.6%, itself exceptionally good, yet the conclusion was drawn that collapse therapy could not be shown to have had value. The second is that of Drolet (185) where the ratio of case-fatality rates to the number of new cases was surveyed and little change was noted over a 20-year period. But 75% of the cases surveyed

did not even have hospital treatment and from Whitney's report (186) of the same period covering 75 sanatoria, more than two-thirds of the patients had no surgical treatment.

In a more controlled statistical study by Potter (187), the results of treatment are compared in two groups, one before collapse therapy was in widespread use and the other treated by more modern methods.

Group	Cases	Deaths	Percentage
1926 - 1932	1,349	1,026	76
1932 - 1938	1,600	708	44.2

The Clinical Method:

To decide the place of pneumothorax in the scheme of treatment and the amount of benefit to be expected from its use, more reliance can be probably placed on the results of small groups of cases dealing with properly administered collapse therapy. It is still too soon to assess these in terms of the long-term effect on prognosis. The smaller series comes within the compass of one observer's experience and the emphasis is laid on good treatment.

The following table taken from Rafferty (36) shows the results in 6 series recent enough to include fairly modern ideas:-

Author	Patients	Deaths	Follow-up
Potter (187)	118	3	1 - 8 years
Eglee & Jones (188)	100	1	3.2 years average after re-expansion
Bloch et al (35)	60	4	1 - 12 years
Hurst & Schwartz (179)	117	1	1½ - 6 years
Soderstrom (184)	201	21	5 years and up
Morriss (181)	370	75	5½ years average after re-expansion
Total:	966	105	

The cases in the foregoing series form a select group in that they all have had the advantage of adequate pneumothorax treatment. In this connection it is interesting to note that Pinner has estimated that, of every thousand hospital admissions, the actual figure for pneumothorax success is probably not more than 12 - 15%. (66)

From the foregoing remarks and from other published reports it is definite that the prognosis for the individual case has greatly improved, provided that the patient is admitted to hospital with his pulmonary tuberculosis in a stage amenable to the forms of treatment at our disposal. How much of this improvement is attributable to pneumothorax cannot accurately be assessed as this is merely one of a number of collapse measures and these in turn are part of a large treatment programme which includes adequate sanatorium treatment, rehabilitation and adequate after-care.

In the present series of one hundred cases, a long enough period of observation has not yet elapsed for worthwhile long-term conclusions to be drawn. The results up to the present have accordingly been set down without much comment. The empyema cases and certain other cases showing points of interest have been discussed in some detail and certain conclusions drawn.

Results of treatment in one hundred consecutively admitted patients who had artificial pneumothorax

There were 34 males and 66 females in the series.

Extent of Disease:

Although a very few cases were of anatomically minimal extent, none were pathologically minimal, that is, none were without a positive sputum and/or demonstrable cavitation at the time of admission. All were moderately or far advanced. Twenty-five were unilateral and 75 bilateral.

Lung collapsed:

Forty patients had right pneumothorax only, 39 had left only and 21 had bilateral collapse. Thus one hundred and twenty-one pneumothoraces are considered.

Fate of the Pneumothorax:

1. Abandoned as ineffective. There were 32 in this category and the duration of refills is shown as follows:-

Abandoned within three months	...	17
Abandoned within six months	...	11
Abandoned after six months	...	4

Fifteen of the above pneumothoraces were abandoned without thoracoscopy being carried out as the adhesions appeared too extensive radiologically or the pneumothorax space was too small for the manipulation of the instruments. In 17, thoracoscopy was performed. In 4 of these, no adhesion section was attempted; in the remainder some section of adhesions was done but not enough favourably to influence the collapse.

The 32 pneumothoraces abandoned as ineffective were divided into two groups, namely, those having further collapse treatment and those not having such treatment.

(a) Those having further collapse treatment. This consisted of:-

Thoracoplasty	...	3
Extrapleural pneumothorax	...	1
Phrenic crush & pneumoperitoneum	.	3
Cavernostomy	...	1

The present status of the above cases is as follows:-

Well and fit for work	...	2
Convalescent	...	1
Disease not yet controlled	...	2
Worse	...	1
Lost trace	...	1
Died	...	1

(b) Those having no further collapse treatment. The reasons for this are as follows:-

Refused further treatment	...	10
Unsuitable for further collapse	...	10
Awaiting further collapse	...	2
Died (air embolism)	...	1

The status of these patients now is as follows:-

Well and fit for work	...	1
Worse	...	1
Died	...	10
Lost trace	...	6
Unchanged	...	4
Convalescent	...	1

The following table shows the status at present of the 31 patients in whom pneumothorax was abandoned as ineffective:-

	Total	Further collapse	No further collapse
Fit for work	3	2	1
Convalescent	2	1	1
Unchanged	6	2	4
Worse	2	1	1
Died	11	1	10
No trace	7	1	6
Patients	31	8	23

2. <u>Ineffective collapse but continued for extrapleural strip with fenestration</u>. There were 4 such cases. Two of them are now alive and well and continuing refills. The other 2 developed tuberculous empyema and the collapse was then abandoned. One is alive with negative sputum, the other requires thoracoplasty.

3. Cases in which the pneumothorax space was lost.

(a) Completely - 13

Within 6 months:	4 -	One is worse, 2 alive and well, 1 - lost trace.
Within one year:	3 -	One dead, 1 lost trace, 1 requiring further treatment.
Within two years:	3 -	2 alive and well, 1 with pneumothorax continuing on the opposite side, 1 worse.
Within later periods:	3 -	All from 3 to 3½ years. Two fit for work and 1 requiring treatment for active disease in the opposite lung.

(b) Partially - 5

Three pneumothorax spaces became partially obliterated and were filled with oil. One of these now, after 3 years, has an open cavity in the collapsed lung. One has had an effective collapse for 9 months. In the third case, the oleothorax is preserving a small apical space of doubtful value.

Two other cases became partially obliterated in the presence of small pleural effusions. After a few aspirations in each case, the fluid was left to fill up the pleural space and a satisfactory degree of collapse is being maintained.

4. Voluntary Re-expansion. There are only 4 cases in this category. In 1 the lung was allowed to re-expand after only 1¾ years as the opposite lung required collapse. Unfortunately, a fresh cavity appeared within a short time and it was found impossible to re-collapse the lung. The other 3 cases had effective collapse for about 3½ years and have recently been re-expanded without incident.

5. Pneumothorax continued to date under personal supervision. These cases have been divided into groups according to the length of time during which an effective collapse has been operative.

Under one year	... 10
One to 2 years	... 8
Two to 3 years	... 10
Three to 5 years	... 12

The status of these patients is shown in the following table:-

	Total	Under 1 yr.	1 - 2 years	2 - 3 years	3 - 5 years
Fit for work	24	1	6	7	10
Well, not yet fit for work	12	8	2	2	
Active disease in other lung	4	1		1	2

6. Ineffective collapse continued. There are 3 cases in this category. The pneumothorax is being continued because they are not fit for thoracoplasty and the collapse is apparently having some effect in reducing toxaemia.

Actual Status of the original One-hundred Patients at Present:

Fit for work:	39
Well, but not yet fit for work:	18
Active disease, at home:	5
Still in hospital, disease not controlled:	12
Dead:	12
No up-to-date trace:	14

Note: These patients classified as "fit for work" are all actually employed in gainful occupations or, in the case of most of the females, carrying out their usual domestic duties which, in this country, are of a fairly light nature. Such females included in this group have been judged fit for gainful employment. Where this was not the case, although they may be doing some domestic work, they have been included under "well, but not yet fit for work".

Pleural Effusions in these cases:

Of the one hundred and twenty-one pneumothoraces, eighty have been noted to have fluid in the pleural space during my observation of them. Late effusions in those who have left hospital are likely to occur, making the incidence for this series eventually considerably higher and I do not think it is too much to say that every case of pneumothorax, if followed closely enough, will show a little fluid in the pleural space at some time or other during the course of the collapse. For convenience of discussion, these effusions have been divided into three groups:

(a) Small effusions, not aspirated

There were 27 small effusions without symptoms, only noticed on screening or X-Ray. Most of these were transient, although a few persisted or recurred. None required tapping.

Seventeen had no obvious effect on the collapse but 10 of these showed some pleural thickening on X-Ray which may later interfere with full re-expansion. Six others were associated with gradual obliteration of the pleural space. One case of bilateral pneumothorax had bilateral small effusions with fairly marked pleural thickening on both sides.

Eleven of these small effusions were associated with a degree of collapse which never was effective. All have now been given up except 3 which seem to be partially effective.

The other 16 occurred in cases where the collapse was or still is effective. In 11, effective collapse continues. Of the remaining 5, 2 obliterated completely but without re-activation of the disease, one was allowed to re-expand elsewhere and later relapsed with bilateral spread of the disease. One partially obliterated and the space was filled with oil. This, however, did not prevent the cavity re-opening at a later date and the case now awaits further treatment. The opposite pleural space in this case obliterated completely without recurrence of the disease.

Four other small effusions were associated with voluntary re-expansion.

Re-expansion at end of treatment:	2
Re-expansion of ineffective collapse for further treatment:	2

Small terminal effusions were noted in 2 cases.

(b) Effusions requiring aspiration (excluding empyemata)

Thirty-two effusions required aspiration. Marked symptoms (fever, pain, dyspnoea, tightness in the chest, malaise) were present in 15 cases. Minor symptoms only were noted in 9 and no symptoms, in spite of moderate to marked effusions, in 8 cases.

All the fluids aspirated were thin, clear yellow or slightly opalescent. Not all were examined for tubercle bacilli but a proportion were positive.

The time of onset with relation to the induction was as follows:-

Within three months:	13
Three to six months:	9
Six months to one year:	7
Total within one year:	29

Three occurred later, one after 1½ years, one after 3 years and one after 3½ years. The two latter cases developed shortly after childbirth.

This bears out the view that most effusions occur relatively early and may be associated with continuing activity of the disease process and also with the presence of adhesions.

Of such effusions in the present series requiring aspiration, 7 occurred within 6 weeks of thoracoscopy. In one, thoracoscopy only was done, in 3 adhesion section was incomplete and in 3 the adhesions were completely divided.

(c) Empyemata.

Twelve empyemata occurred in the present series. Two followed extrapleural strip with fenestration and are mentioned separately. Of the remainder, only one was not associated with the section of adhesions. Six occurred within 6 weeks of thoracoscopy and 3 occurred later. These are now described in some detail.

Case 1:

A.E.M. Male. 34 years. Admitted 13/7/44. Died 11/3/45.

Far-advanced bilateral disease, predominantly exudative. Moderately large cavities at right apex and in the left mid-zone.

Right artificial pneumothorax induced almost immediately. Apical adhesions present preventing closure of the cavity.

Thoracoscopy and partial adhesion section four months after induction. Cautery tip broken off and left in the pleural space. Pneumonolysis had little effect on the collapse and the adhesions were seen to be on the stretch after the operation.

Pleural effusion developed almost immediately. First aspirated 6 days after operation. The fluid was then clear and no tubercle bacilli were detected in it. The fluid quickly became purulent and frequent aspirations were required. Concentrated solution of Urea was instilled on a few occasions. Steady downhill course. Tuberculous laryngitis later. Died after 8 months in hospital.

There are two points of interest in this case. Firstly, there seems to have been undue haste in inducing pneumothorax. Nowadays, Streptomycin would probably be used, in conjunction with a liberal period of bed rest, in the hope of overcoming the exudative element before proceeding to collapse therapy. Pneumoperitoneum might also be of value in tiding the patient over the acute phase.

Secondly, it is very probable that the partial adhesion section which was performed was a direct cause of the onset of empyema, either from opening into tuberculous tissue or from tearing of adhesions which were seen to be on the stretch after the operation. One or other of these mechanisms may have led to the development of a small broncho-pleural fistula as the pleural space showed no sign of diminishing in size, although aspirations were carried out frequently. The prognosis without treatment was undoubtedly bad but the course of treatment adopted no doubt hastened the fatal issue rather than retarding it in any way.

Case 2: J.D. v.d. W. Male. 26 years. Admitted 3/7/44. Discharged 12/5/45. Re-admitted 9/7/48. Discharged 8/11/48.

Limited fibro-caseous disease in the right upper third with a moderately large cavity. Infiltration at left apex. (Case 2, Fig. 1).

Right artificial pneumothorax induced after a fortnight. Apico-lateral adhesions seen on X-Ray. Pleural effusion with moderate symptoms 2 months after induction. Aspirated a few times - fluid clear, no tubercle bacilli found. Thoracoscopy 5 months after induction. Three small adhesions severed but apex held up medially by short wide area of adherence. No section of this attempted.

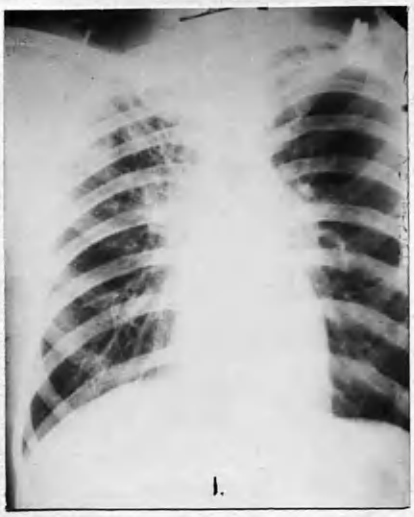
Collapse slowly improved. Discharged after ten months in hospital with cavity apparently closed (Fig. 2) and the sputum negative. Refills were continued. Two years and four months after induction, he developed tuberculous empyema without any marked symptoms. As the cavity was still apparently closed, aspirations were carried out and the pneumothorax continued. After a month the cavity again became visible and the patient was admitted to hospital. Thoracoplasty was advised but refused. The pneumothorax was then abandoned and the lung left to re-expand.

One year later the space had almost obliterated (Fig. 3) and a six-rib two-stage thoracoplasty was carried out. Streptomycin was given in a dosage of 1 Gm daily for 3 months before, during and after the operation. Now, 9 months later, the patient's condition is good. The cavity remains closed (Fig. 4), the sputum is negative and he is fit for light farm work.

Looking back on this case, it seems reasonable to doubt whether the cavity was really ever properly closed by artificial pneumothorax. It is quite likely that, at the time of discharge, a small remnant still persisted at the apex. However, refills were continued and no obvious cavity was seen over a period of nearly $2\frac{1}{2}$ years. At the end of this time it was found that a tuberculous empyema was present. I do not see how this could have been anticipated or prevented.

The refusal of the patient to undergo thoracoplasty for a further year might have been expected to make the operation difficult on account of the pleural thickening and, in fact, at operation only a small degree of apicolysis could be obtained but the result of the operation was satisfactory in that cavity closure and conversion of the sputum was obtained.

CASE 2



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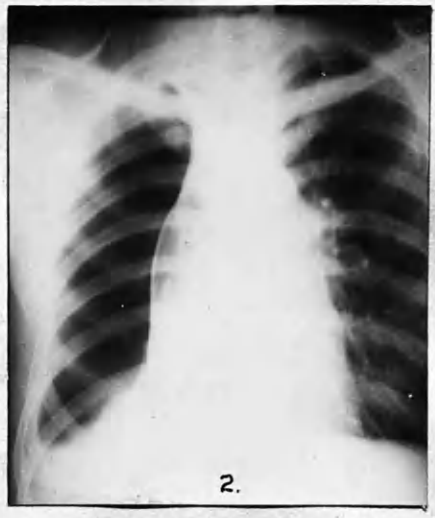

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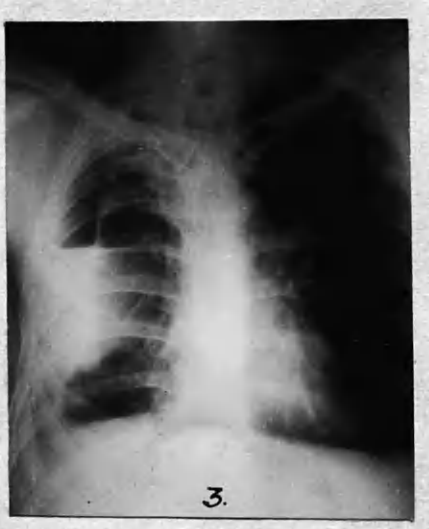

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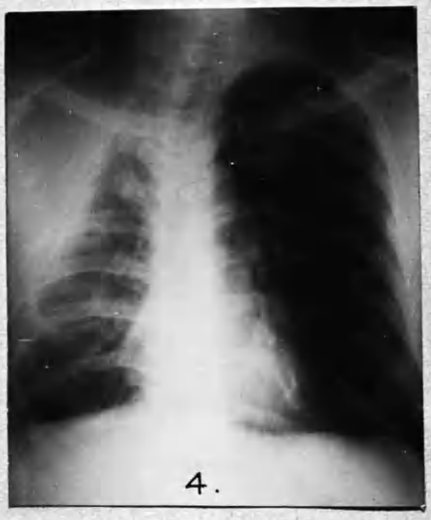

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Case 3:

P.A. Female. 19 years. Admitted 4/7/44. Discharged October, 1945.

Far-advanced bilateral infiltration with moderately large cavities in both upper zones ... febrile.

Right pneumothorax induced after only a few days in hospital. Adhesions seen holding up the upper lobe which appeared partially atelectatic. Thoracoscopy 6 weeks later and complete section of several complex apical fold adhesions.

Next day spontaneous collapse of the lung with atelectasis of the upper lobe in which the cavity could be clearly seen. An effusion developed immediately and the patient was very ill for 3 weeks. Fluid at first thin but became purulent 2 months after thoracoscopy. No organisms detected in it. A few aspirations were required and concentrated Urea solution was instilled each time. The fluid dried up and refills were continued for a further 11 months until discharge. The collapse appeared satisfactory, although there was considerable pleural thickening.

Left pneumothorax induced after the empyema had settled. The patient left hospital with bilateral pneumothorax, no visible cavity in either lung and negative sputum. Report received $3\frac{1}{2}$ years later. Alive, well, working as a housewife, sputum negative, no sign of activity on X-Ray. Refills were given up "some time ago".

The onset of empyema in this case was definitely associated with thoracoscopy, possibly from cutting through tuberculous lung tissue. Although the patient was very ill for a time, the long-term result has been satisfactory. Unfortunately, after leaving hospital, the case was no longer personally handled and details of the state of the lung are lacking at this stage. To have given up the pneumothorax when the empyema developed would have almost certainly meant doing a thoracoplasty, a procedure for which the patient at that time was unfit.

Case 4:

C.G.F. Female. 20 years. Admitted 11/6/45. Discharged 3/9/48. Died March, 1949.

Far-advanced bilateral exudative disease with large right upper zone cavity and several small cavities in the left infra-clavicular region. Febrile. Strict bed rest for 3 months but cavity on the right became larger and further cavitation was noted in the same lung (Case 4 - Fig. 1). Right pneumothorax induced.

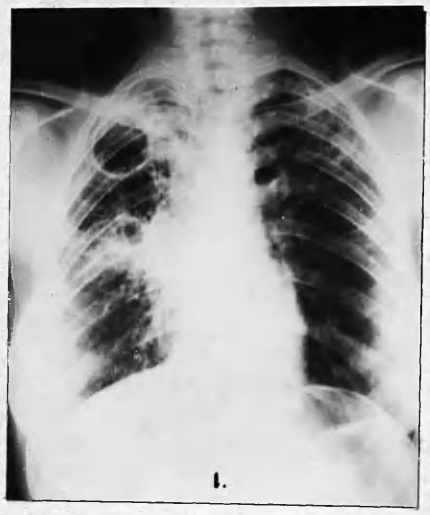
Thoracoscopy carried out after 2 months collapse. Complex apical bands, 2 of which were divided and a third partially severed. The apical cavity then assumed the appearance of a tension cavity (Fig. 2). In spite of this, 3 refills of 500 to 600 c.c. were given in the 16 days following the thoracoscopy, at the end of which time the patient developed a spontaneous pneumothorax followed by a mixed infection empyema.

Frequent aspiration of air and fluid and the instillation of Penicillin and Urea solution took place during the following 4 months. Then several sinuses developed in the chest wall through which drainage was free. A broncho-pleuro-cutaneous fistula persisted up to the time of death more than 3 years later. No other treatment was attempted as the other lung was grossly involved. Streptomycin for 2 months had no obvious effect on the course of the disease.

This case had in the first place a bad prognosis. A period of 3 months rest did not result in improvement and the attempt to obtain cavity closure by pneumothorax appears to have been justified. A different approach might, however, have been made. Pneumothorax might have been used first to attempt to control the left-sided lesion and thereafter drainage of the right-sided cavity followed by thoracoplasty.might have followed. The right upper zone cavity was apparently a tension cavity.

Again, after the thoracoscopy. the cavity showed a marked degree of inflation. At this stage the pneumothorax might have been abandoned promptly and the lung encouraged to expand by withdrawal of air, thus possibly preventing cavity rupture and the disastrous train of events that followed (Fig. 3).

CASE 4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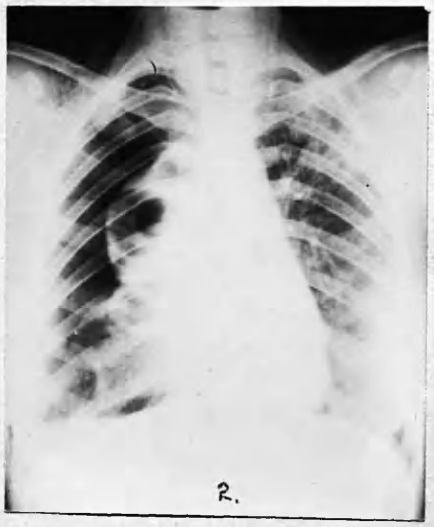
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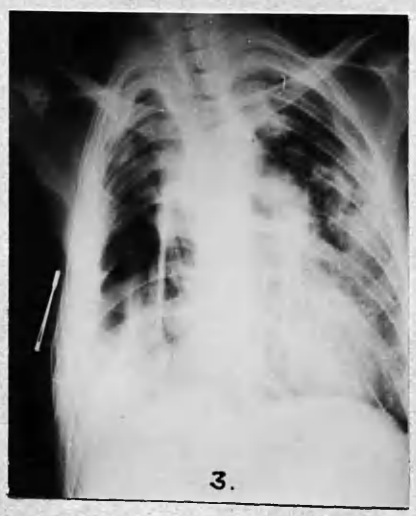
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Case 5:

J.E.V. Female. 22 years. Admitted 8/8/46. Died 2/6/47.

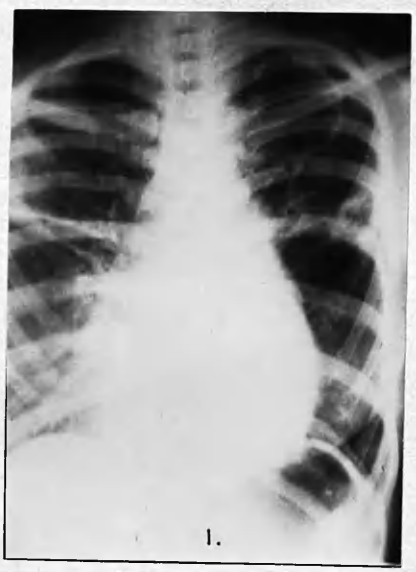
Moderately advanced bilateral disease, chiefly left-sided with a large apical cavity with a fluid level (Case 5, Fig. 1). Bed rest for 3½ months but the cavity became larger and assumed the appearance of a tension cavity. There was also some extension of the disease in the opposite lung. Febrile since admission.

Left pneumothorax induced. Sharp febrile reaction about 10 days after induction. X-Ray at this time showed only a small collapse and ballooning of the cavity (Fig. 2). Refills were continued for a further two months, up to 700 c.c. of air being given at a time, although the pressures were always left negative. Contraselective collapse of the lung developed but the cavity remained distended (Fig. 3). Refills were then stopped but a short time later rupture of the cavity took place and empyema developed. No further treatment was carried out. Three months later the patient developed tuberculous meningitis and a month later was dead.

Post-mortem confirmed the meningitis and showed a shrunken left lung with a large broncho-pleural fistula opening into the cavity and a small amount of pus in the pleural space.

In retrospect, pneumothorax was definitely not the treatment most likely to lead to successful cavity closure in this case. Further, the pneumothorax should have been promptly abandoned as soon as the cavity showed signs of becoming dangerously distended.

CASE 5



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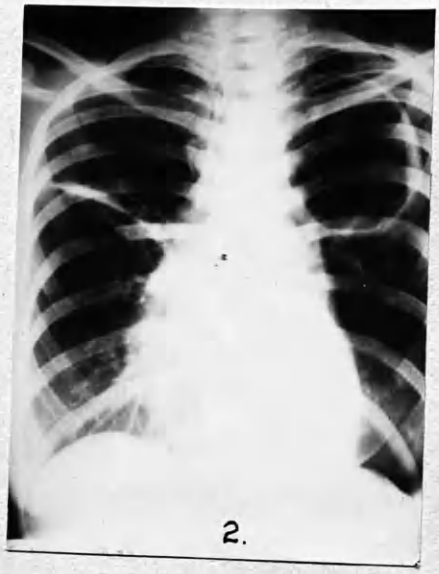

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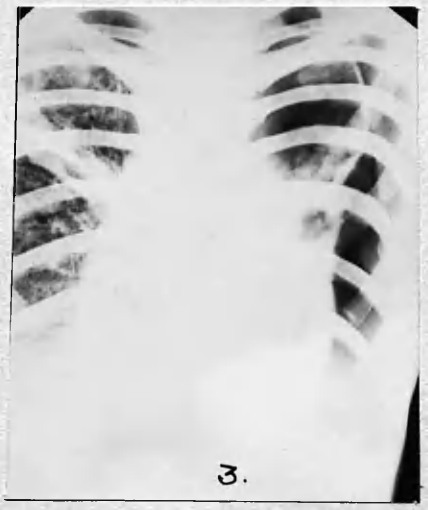

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Case 6:

J.C. v. R. Female. 22 years. Admitted 3/10/46. Died 19/9/47.

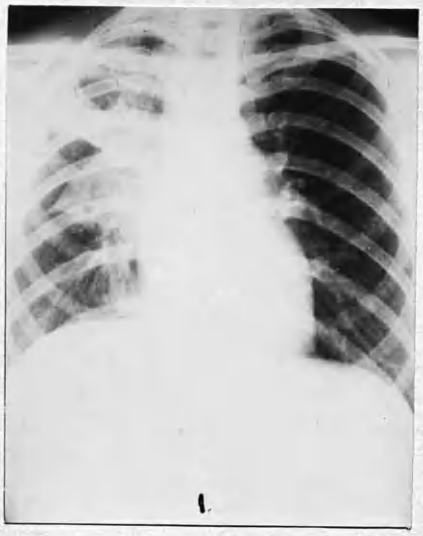
Moderately advanced unilateral disease with small upper lobe cavity and an area of ? atelectasis of the upper lobe (Case 6, Fig. 1). After 5 weeks bed rest right pneumothorax induced. Apical adhesions on tension noted (Fig. 2).

Thoracoscopy carried out 2 months later. Pleura acutely inflamed. Complex apical folds present - partial adhesion section only. Febrile reaction followed which settled but an effusion developed 3 weeks later which required a few aspirations. The fluid was clear and negative for tubercle bacilli. Further thoracoscopy 2 months after the first. Pleura thick and many tubercles noted on the visceral pleura. No section attempted. Ten days later the pleural fluid was purulent. Azochloramide was instilled but no further aspirations were required and no refills were given. The lung remained well collapsed except that the cavity remained open (Fig. 3). Three months later the patient was considered almost fit for thoracoplasty but sudden death took place after an attack of palpitation.

Post-mortem: Lung well collapsed with small filled cavity. Pleura thickened and some pleural pus present. Thymus persistent and large. Heart - no abnormality noted.

Possibly the partial adhesion section in the presence of an inflamed pleura was to blame for the empyema. The cause of death remained obscure but it does not seem to have been the tuberculosis alone. The cavity at autopsy was closed and filled with inspissated material.

CASE 6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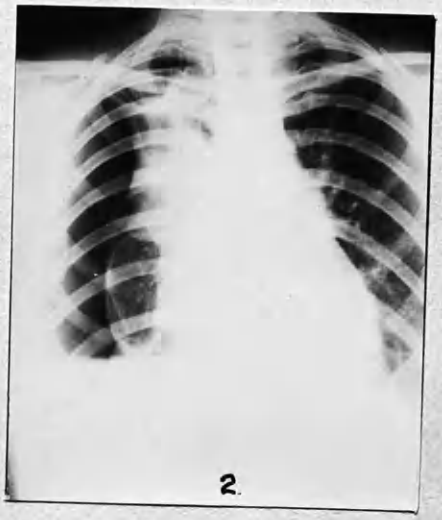
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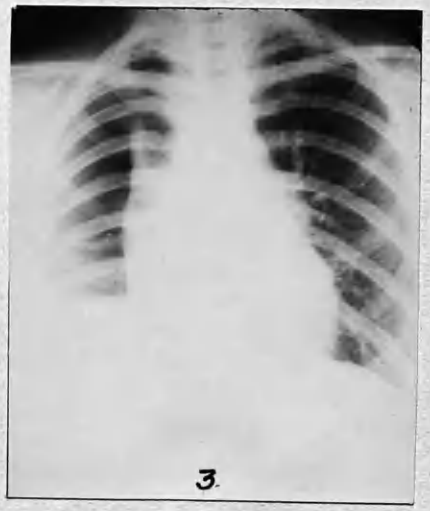
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Case 7:

S.M.K. Female. 20 years. Admitted 5/2/47. Discharged 15/12/48.

Far advanced bilateral disease with right hilar cavity and several smaller upper zone cavities (Case 7, Fig. 1). After 2 months bed rest X-Ray showed a further cavity towards the right base. Pneumothorax then induced.

Thoracoscopy carried out one month later. Two lateral apical straps were cut but a medial apical web could only be partially severed. Second sitting 2 months later and a further small cord cut (Fig. 2). Febrile effusion and complete flat collapse of the lung followed. Fluid was aspirated frequently in the 3 months which followed. The fluid was at first clear, later becoming purulent. Nine months later the lung had partially re-expanded, the cavity was still open, the pleura was thickened but the pus was no longer accumulating (Fig. 3). Small refills were, therefore, continued and have been kept up to date (one year and two months). Right phrenic crush was then carried out, followed by pneumoperitoneum and 4 months later the cavity could not be seen although the sputum was still positive (Fig. 4).

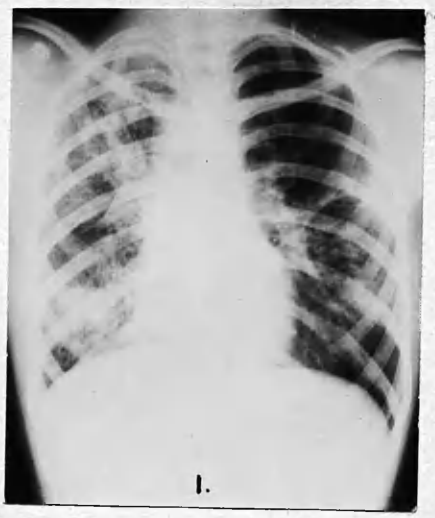
Streptomycin 1 Gm daily was then given for 2 months, resulting in some resolution of the lesions in the left lung and conversion of the sputum.

Now, 4 months later, refills are being continued and the patient has left hospital with the disease apparently under control.

There does not seem to have been any way of avoiding the empyema in this case except, possibly, by abandoning the pneumothorax after the first thoracoscopy when it was seen that the apical adhesion was uncuttable. As the collapse of the lung looked as if it would gradually improve, however, and as the other lung was involved, this was not done. Although the ultimate prognosis for this case remains obscure, the disease is at present under control and refills are being continued.

This case is also of interest in being one where several forms of therapy have been combined, namely, bed rest, artificial pneumothorax, phrenic crush, pneumoperitoneum and a course of Streptomycin.

CASE 7



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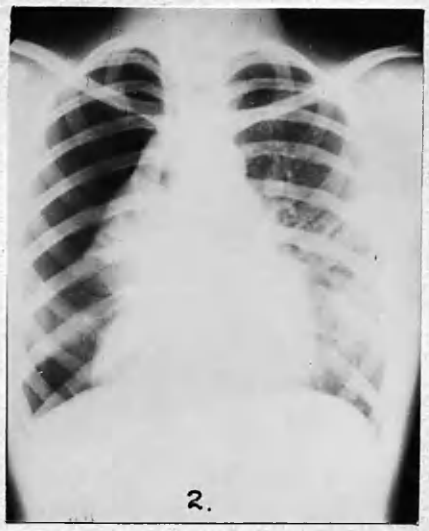

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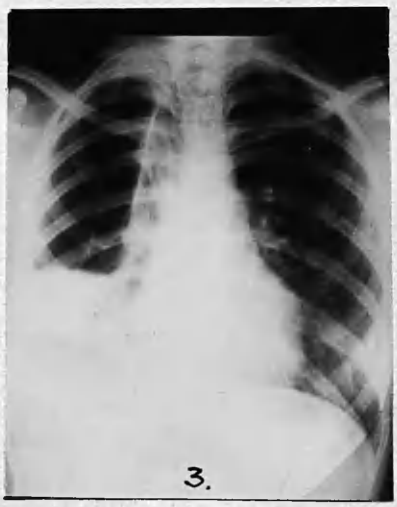

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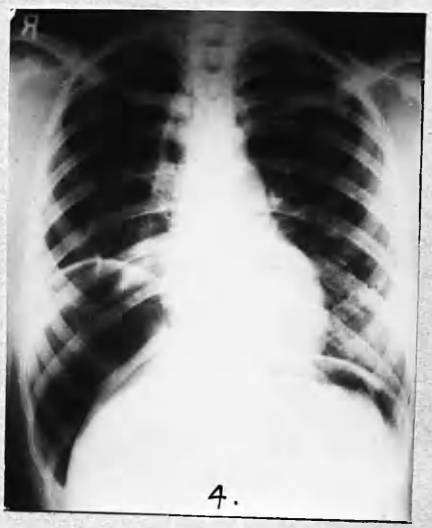

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Case 8:

A.S.B. Female. 37 years. Admitted 13/5/47. Discharged 20/1/48.

Moderately advanced bilateral disease with a thin-walled cavity in the right upper zone. Minimal disease in the opposite lung. After one month of rest, no change, pneumothorax induced on right.

Complete section of apical cone adhesions in 2 sittings 1½ months after induction. Fairly large effusion with symptoms developed within one month of the second thoracoscopy. Aspirated several times during the following 3 months. Pneumothorax continued and discharged to out-patient treatment after 8 months in hospital. Pleura thickened, costo-phrenic angle obliterated having small infrequent refills.

During the following year, gradual obliteration of the pleural space from the base upwards and collapse now only present over the upper third of the lung. This space is filled with fluid which is thin pus - negative for tubercle bacilli.

This is a more benign type of empyema in that the pus was only discovered one year and nine months after the induction of pneumothorax and when a considerable degree of pleural obliteration had taken place. The cavity remains closed and the pus is being allowed to remain as a cushion over the upper third of the lung. The patient is in good health.

Case 9:

J.C.E.C. Female. 35 years. Admitted 6/6/47. Discharged 25/9/48.

Far-advanced bilateral disease, chiefly right-sided with apical and hilar cavities. After one month of rest further cavitation noted in the left lung.

Right pneumothorax 6 weeks after admission. The cavity in the opposite lung then became larger. Thoracoscopy $3\frac{1}{2}$ months after induction. Partial section of web adhesions at the apices of upper and lower lobes. Followed by marked emphysema and febrile reaction for a few days. Pneumothorax continued although ineffective.

Left pneumothorax induced one month later and thoracoscopy after a further 6 weeks. Short thick lateral adhesion completely divided.

Next day over-collapse and black-out of the right lung, with effusion. Air and fluid aspirated. Ten days later right lung partially re-expanded but left lung over-collapsed and effusion on this side. Patient very ill, dyspnoeic and febrile for ten days. Air aspirated from the left side and Penicillin instilled. Then Streptomycin intramuscularly 2 Gm daily for 12 days. This had to be stopped on account of giddiness.

Thereafter the right lung was allowed to re-expand as the collapse was ineffective. Aspirations and small air replacements were continued on the left side for 7 months. The fluid then became purulent and this pneumothorax was also abandoned. Both lungs have now re-expanded except for small pockets over the upper thirds but the patient is unfit for further collapse treatment and the general condition is very poor.

The outlook for this case was from the first poor. Whether pneumothorax should have been employed is debatable. Possibly pneumoperitoneum with rest for a long period might have been better. The curious behaviour of the lungs following the second thoracoscopy is noteworthy.

Case 10:

W. de V. Male. 25 years. Admitted 10/10/47. Still in hospital.

Moderately advanced bilateral disease with 3 fairly large cavities of tension type in the left lung. After 3 weeks rest 2 of these had become smaller. Left pneumothorax then induced. Apex held up by adhesions. Left thoracoscopy after 3 months and the lung freed except along the mediastinum. The collapse improved and the cavities became smaller.

Five weeks later sudden onset of effusion with over-collapse and "black-out" of the left lung. Air and thin greenish pus were aspirated. Six weeks later this fluid was thick pus in which tubercle bacilli were detected. Aspirations were carried out as required and intrapleural Streptomycin 0.5 Gm daily was given for 3 weeks. The fluid became negative for tubercle bacilli although still remaining thick pus. The pneumothorax was abandoned at the onset of the effusion and considerable re-expansion of the lung took place although the pleura was markedly thickened.

Left thoracoplasty 13 months after admission (8 months after onset of effusion). Intramuscular Streptomycin for 4 months commencing one month before operation. Now, 5 months after operation, small cavity still visible at the apex and the sputum is positive.

The cavities in this case were definitely of tension type. It seemed justifiable to risk a cautious attempt at pneumothorax and having done so to attempt to divide the adhesions. It is difficult to decide whether the adhesion section, during which lung tissue may have been divided, or the rupture of a small tension cavity was responsible for the onset of empyema in this case.

From a consideration of the foregoing cases certain conclusions may be drawn:-

1. Careful choice of cases for artificial pneumothorax is necessary if the risk of empyema is to be minimised. This risk cannot be obviated entirely without denying effective treatment to certain cases. In certain doubtful cases it is worthwhile running the risk of empyema if no alternative treatment exists.

2. Cases of large thin-walled apical cavity where the bronchial obstructive element is well-marked are not suitable for collapse by artificial pneumothorax and this measure is dangerous.

3. The prognosis is worsened when empyema occurs. This is generally the case, yet certain cases subside after a time and the pneumothorax can be continued.

4. Partial adhesion section can be dangerous unless, post-operatively, attention is paid to certain details, namely, the withdrawal of air before the patient leaves the table to ensure that the intrapleural pressure is well on the negative side, X-Ray of the patient before returning to bed as a further precaution against a dangerous degree of collapse with tension on adhesions and careful posturing of the patient with the same object in view and sedation of cough for some time after operation.

5. Close observation of all pneumothorax cases is essential with frequent screening and radiography.

Two cases of empyema occurred following extrapleural strip with fenestration.

Case 11:

D.P.B. Male. 32 years. Admitted 27/5/47. Discharged 18/8/48.

Extensive infiltration throughout the left lung with a large cavity in the upper zone and minimal infiltration at the right apex. (Case 11, Fig. 1)

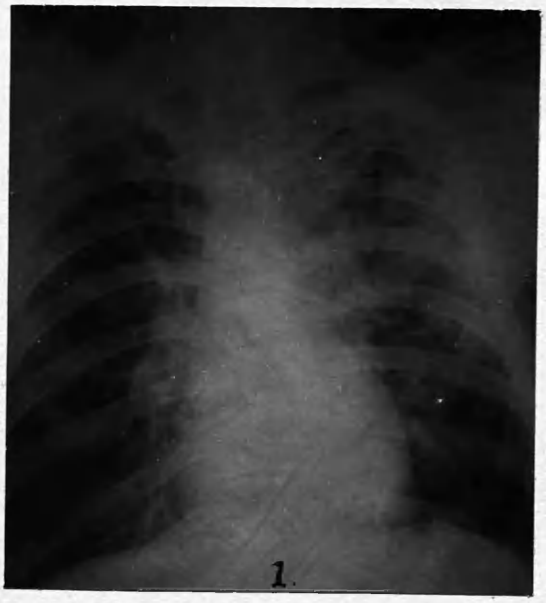
Left pneumothorax after 3 weeks rest. Apex broadly adherent. Thoracoscopy carried out twice but only partial section of adhesions without any real improvement in the collapse of the upper lobe. Moderate pleural effusion noted after the second stage but no aspiration required. The apical cavity persisted. (Fig.2)

Three months after the second thoracoscopy, left extrapleural strip and fenestration carried out. Thick extrapleural adhesion divided. Ten days after the operation, onset of febrile effusion which soon became purulent although no organisms could be detected. Aspiration of pus and air replacement was carried out during the following 5 months and Streptomycin 0.5 Gm daily given intrapleurally for 6 weeks. The fluid gradually diminished in quantity. (Fig. 3)

Five months after the operation the apical cavity was seen to be still open and air refills under positive pressure had no effect in diminishing its size. Thereafter, the pleural space was left to obliterate and it did so partially, the remainder filling up with pus.

Now, one year and three months after the operation, there is a pocket of fluid over the upper third of the lung. The sputum is positive and the cavity is still present near the apex. This patient now requires thoracoplasty. (Fig. 4)

CASE 11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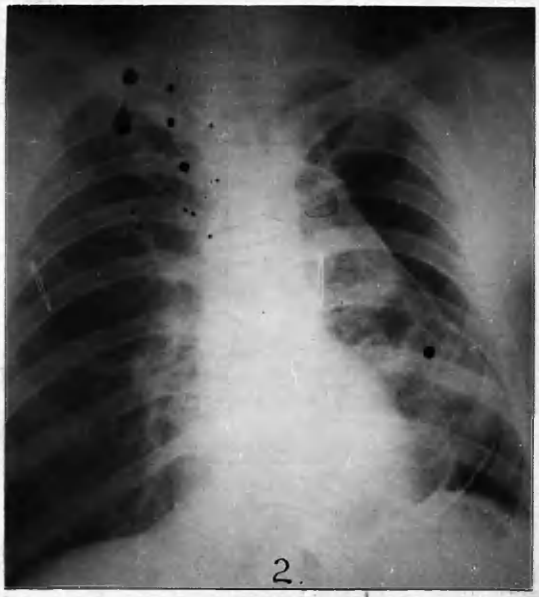
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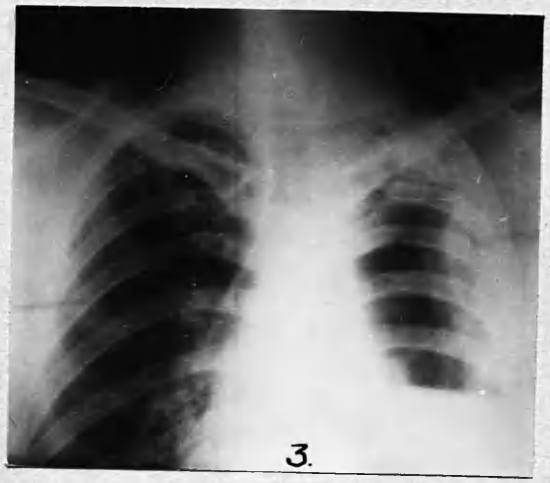
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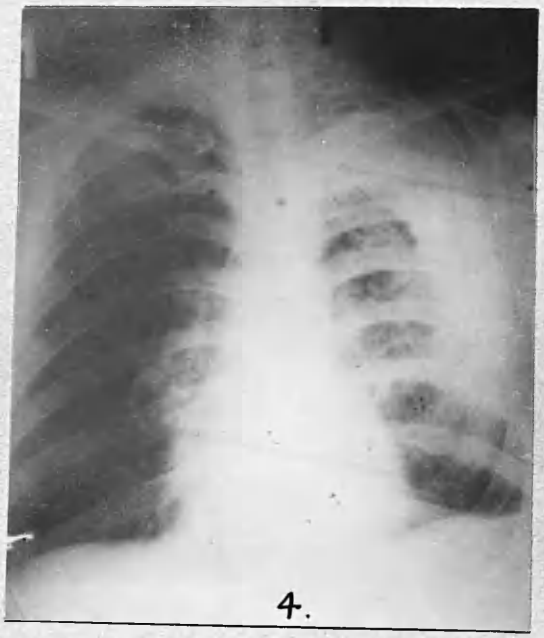
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Case 12:

G.F.S. Female. 37 years. Admitted 10/5/46. Discharged 28/4/48.

Bilateral disease with infiltration in the right upper and mid-zones. Moderately large cavity in the right outer infra-clavicular region and a probable early cavity in the mid-zone. Small area of infiltration and small cavity in the left-infra-clavicular region. Appearances unchanged after 2 months rest.

Right pneumothorax followed by 2 thoracoscopies and incomplete adhesion section. Moderate clear effusion aspirated twice. Gradual diminution in size of the left cavity and sputum became negative. The cavity on the right persisted.

Fourteen months after induction of pneumothorax, right extrapleural strip with fenestration. The strip was not complete at the apex because of dense adherence. Normal post-operative course for the first month. Then, following 2 pleural wash-outs with sodium citrate (because of some blood which could not be aspirated) patient developed a S.aureus empyema. Aspirations of pus and instillations of Penicillin at 2 to 3 day intervals for 3 weeks and then at longer intervals for a further 6 weeks. At this time fluid sterile for pyogenic organisms but tubercle bacilli detected therein. Streptomycin 0.5 Gm intrapleurally and 1 Gm intramuscularly daily for 22 days. Then stopped - giddiness. No tubercle bacilli detected in the fluid during the course of Streptomycin treatment or at the end of the course.

On discharge, no cavity visible in either lung. Sputum negative and general condition good. Has had a few aspirations since then, but none for the past 6 months and the empyema space has almost obliterated. No obvious activity of the pulmonary lesions.

The following cases are described in some detail because they illustrate certain points in connection with the choice of case for pneumothorax, the influence of the bronchial obstruction factor and the difficulty of closing cavities in the lower lobe:-

Case 13:

J.C.F.W. Female. 31 years. Admitted 23/4/47. Discharged 9/12/47.

Insidious onset. Hoarseness and dyspnoea marked on admission. Examination of the chest showed harsh breath sounds and loud piping ronchi over the right upper third lung-field posteriorly. X-Ray at that time showed infiltration and fibrosis throughout the right lung chiefly concentrated in the upper half with a small cavity behind the clavicle. Minimal infiltration at the left hilum. Vocal cords were doubtfully involved. Sputum positive. B.S.R. 37 (Westergren 1 hr.).

For the first 6 weeks, bed rest and vocal rest. During this time practically afebrile with an occasional "spike" to 100. Sputum 3/4 oz. daily. The cavity had become more obvious and it was decided to attempt pneumothorax.

Right pneumothorax induced 4/6/47 followed by thoracoscopy and apparently complete adhesion section after 5 weeks. Followed by atelectasis of the upper lobe, the cavity remaining visible and appearing somewhat distended. Refills were continued and the cavity gradually diminished in size and appeared closed about 4 months after thoracoscopy. Refills continue and the collapse is satisfactory.

There was a definite bronchial obstructive element in this case as shown by the physical signs and the behaviour of the cavity in the early stages of the collapse. However, the check-valve mechanism was eventually abolished with cavity closure and satisfactory collapse.

Case 14:

W.F.F.S. Male. 23 years. Admitted 17/2/48. Still in hospital.

Insidious onset. Well marked toxic symptoms on admission. Marked wasting generally and of the chest muscles in particular. Poor air entry left lung with scattered rales over the upper half. X-Ray showed a system of thin-walled cavities in the left upper lobe with scattered infiltration here. Also a small thin-walled right sub-apical cavity with a little surrounding infiltration (Case 14, Fig. 1).

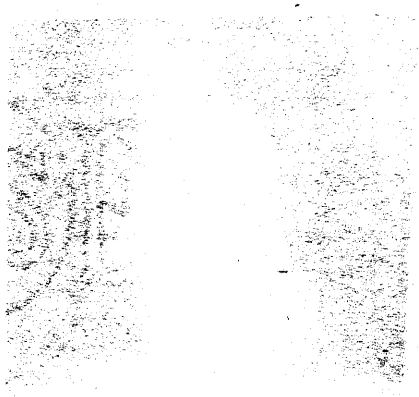
Rest only for the first 7 weeks. At the end of this time some fresh infiltration towards the left base and some increase in the size of the right sub-apical cavity.

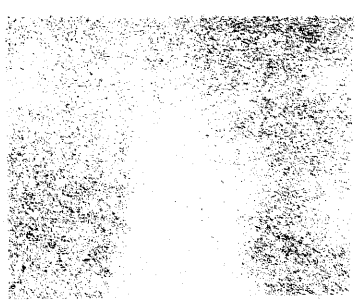
Left pneumothorax induced 7/4/48. Adhesions noted holding up the apex. Thoracoscopy carried out in June and again in August. Partial section on the first occasion and no cutting on the second occasion as the apex was seen to be broadly adherent to the cupola. (Fig. 2). A small effusion developed with fever and mild symptoms for a few days but no aspiration was required. The left pneumothorax was then relaxed considerably and has now been abandoned.

Meanwhile, on the right side the cavity had been enlarging and right pneumothorax was induced 6/10/48 followed by thoracoscopy on 30/11/48. One adhesion was sectioned but the apex was adherent medially and the apex of the lower lobe was adherent in the paravertebral gutter and no division of these was attempted. The cavity had been somewhat ballooned before thoracoscopy but diminished in size after the operation. A month later, with the same degree of collapse of the lung, the cavity was seen to become larger. An increase in the collapse caused the cavity to become again larger and there was a well-marked circum-cavitary zone of atelectasis present. The pneumothorax was considered to be ineffective and possibly dangerous and the lung was allowed to re-expand. Four months later the lung has now completely expanded and the cavity has assumed giant proportions (Fig. 3).

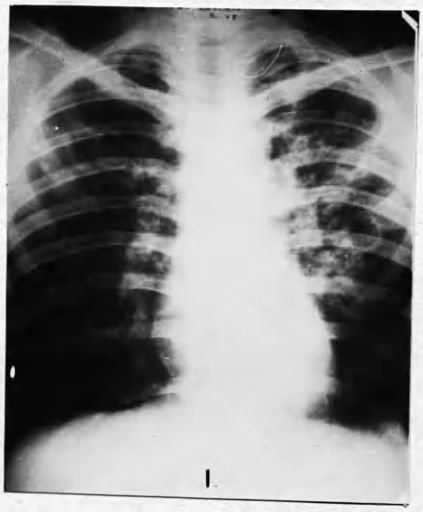
The treatment now proposed is cavernostomy on the right possibly to be followed by thoracoplasty on the left.

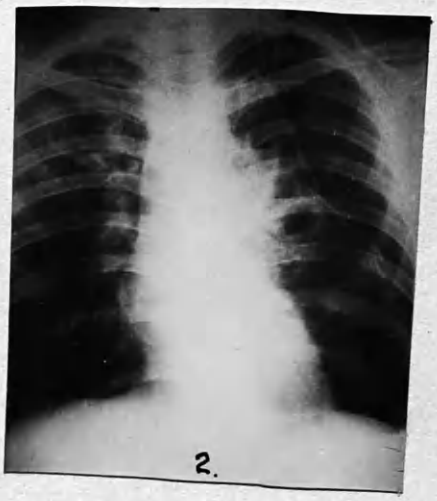
The behaviour of this cavity in the right lung illustrates very well the effect of partial bronchial obstruction and the difficulty of obtaining collapse with pneumothorax. Case 13 showed the other side. The cavity there, although exhibiting the behaviour of a tension cavity for a time, eventually became closed satisfactorily.

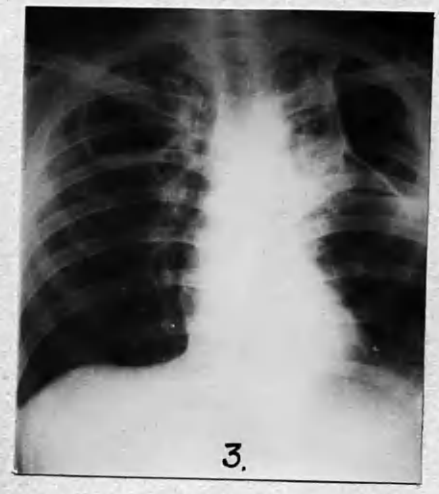


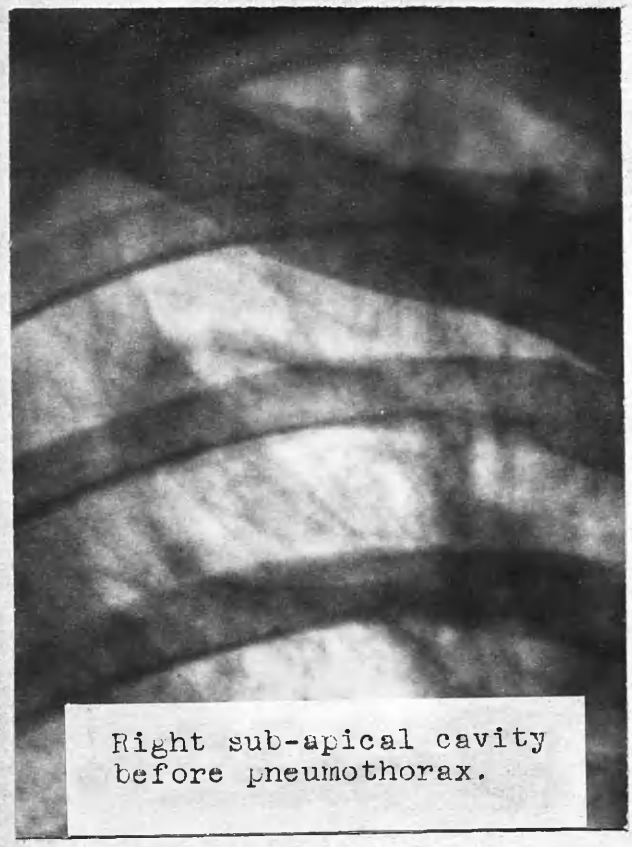


CASE 14

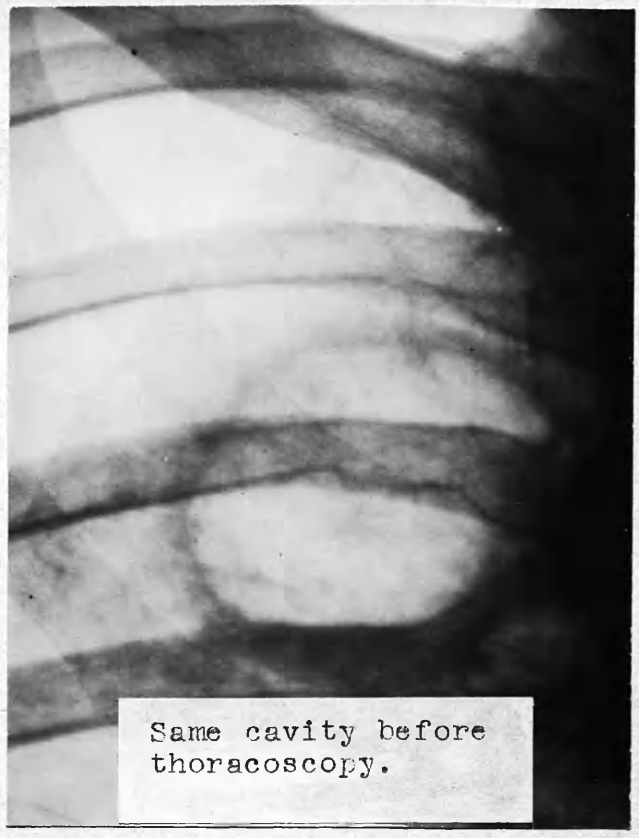






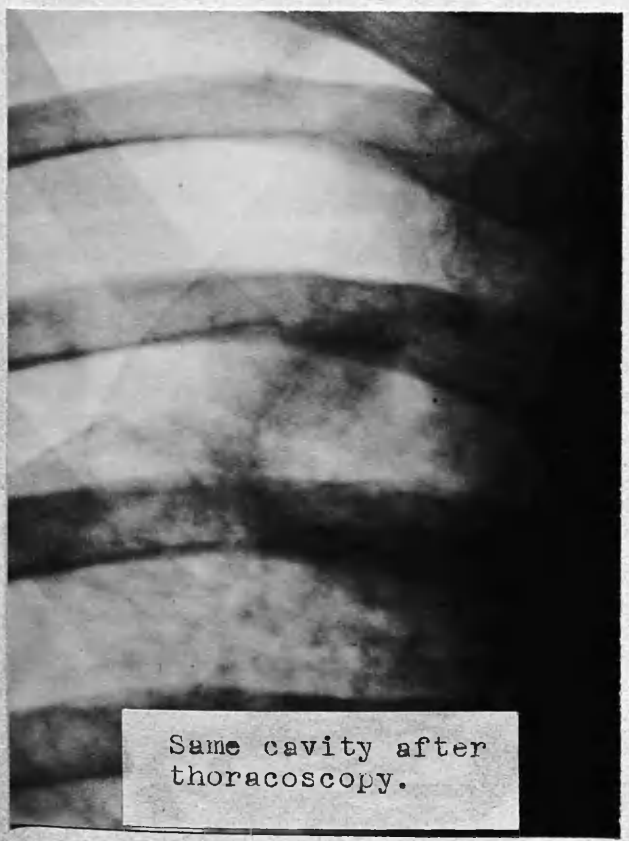


Right sub-apical cavity before pneumothorax.

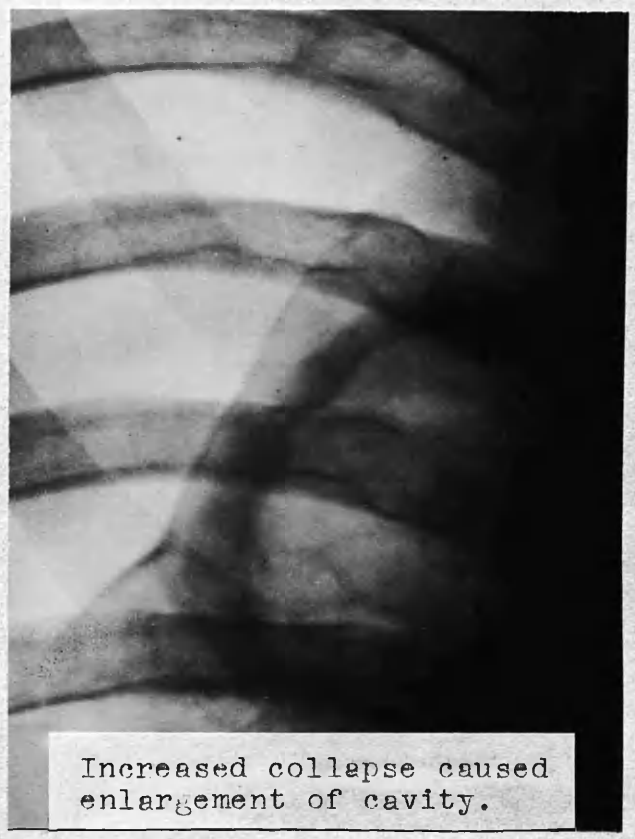


Same cavity before thoracoscopy.

CASE 14



Same cavity after thoracoscopy.



Increased collapse caused enlargement of cavity.

Case 15: P.J.L.C. Female. 28 years. Admitted 14/1/45. Discharged 7/12/45.

First admitted to hospital in November 1942 following insidious onset of illness. X-Ray at that time showed extensive right-sided infiltration with cavitation in the infra-clavicular and lower zones. Right pneumothorax was induced in January 1943. Adhesions were noted at the apex but the patient refused thoracoscopy and also phrenic crush and took her own discharge attending thereafter for refills. The position at the time of discharge was that there was a fair degree of collapse of the right lung but a small residual cavity probably persisted.

Refills were continued for a year and five months thereafter but then the patient had a small haemoptysis, signs of activity were noted in the left lung and X-Ray showed infiltration and cavitation in the upper zone left. The cavity still persisted in the right lung and the pleura was considerably thickened.

Left artificial pneumothorax was then induced, followed 3 months later by thoracoscopy and division of all adhesions. Within 3 months this cavity on the left had been replaced by a dense opacity. Just before discharge the right pleural space was filled with oil and the cavity then appeared closed.

For 2 years after discharge refills were continued on the left but during the first 3 months of 1948, that is to say, about $2\frac{1}{2}$ years after the cavity in the left lung closed, the left pleural space became totally obliterated. Now, more than a year later, there has been no sign of this cavity re-opening.

In January 1949 a small cavity again became apparent near the apex of the right lung. The oil was removed and replaced by air. It is just possible that this cavity may be closed by air refills but more probably thoracoplasty will now be required after 6 years of ineffective collapse.

This case illustrates the difference between effective and ineffective collapse. The right-sided cavity never was properly closed and a form of ineffective collapse was kept up for almost 6 years without effect. The collapse on the opposite side was quickly made effective and it is reasonable to say that cavity healing has taken place. It is to be noted that the patient refused to have the necessary supplementary treatment to make the collapse effective when it was most likely to have been successful.

Case 16:

M.I. du P. Female. 24 years. Admitted 19/12/47. Discharged 25/9/48.

Onset with "'flu" and right pleurisy 5 months before admission. Thereafter gradual development of symptoms. On admission marked lassitude and some cough and sputum.

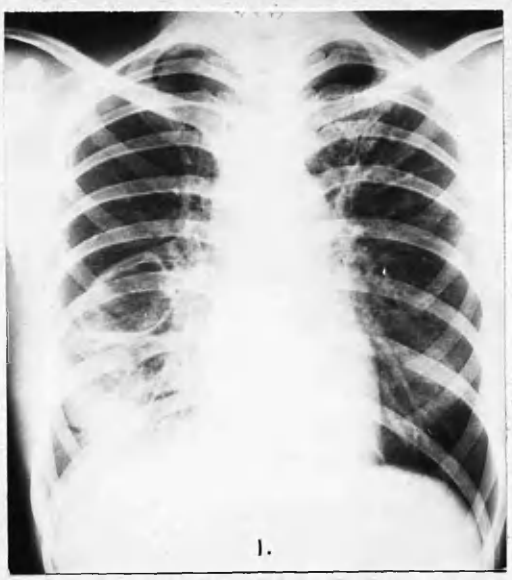
Examination showed very little in the way of physical signs but admission X-Ray revealed tuberculous infiltration of the right lower half lung field and a fairly large right "hilar" cavity of tension type at the apex of the lower lobe. There was also minimal infiltration at the left apex (Case 16, Figs. 1 and 2).

Further examination: slight daily rise of temperature, little sputum but this positive, B.S.R. 38. Bronchoscopy on 9/1/48 showed localised tuberculous bronchitis round the opening of the right lower lobe bronchus but no stenosis.

Right pneumothorax was induced on 14th January, there was a gradually increasing collapse of the lung with, at first, some diminution in the size of the cavity (Fig. 3), but later it remained stationary in size (Fig. 4).

In view of the failure to close the cavity and in the absence of radiologically visible adhesions, it was decided to paralyse the right dome of the diaphragm. Right phrenic crush was accordingly carried out on 6/4/48. Some elevation of the diaphragm resulted and in 3 months the cavity could no longer be seen (Fig. 5).

Two months later she was discharged. General condition was good. Sputum negative, no signs of activity. Six months after discharge she is having fortnightly refills which maintain an adequate selective collapse of the right lower lobe.



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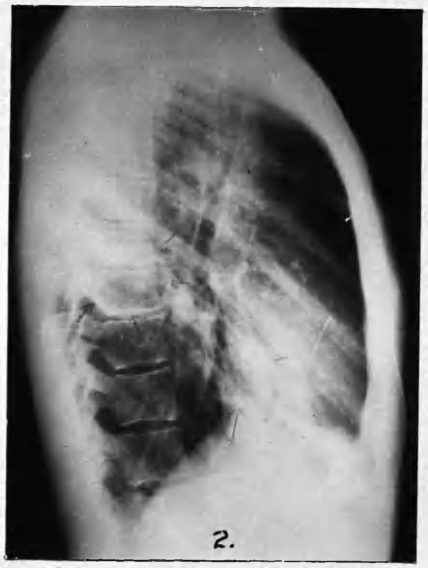

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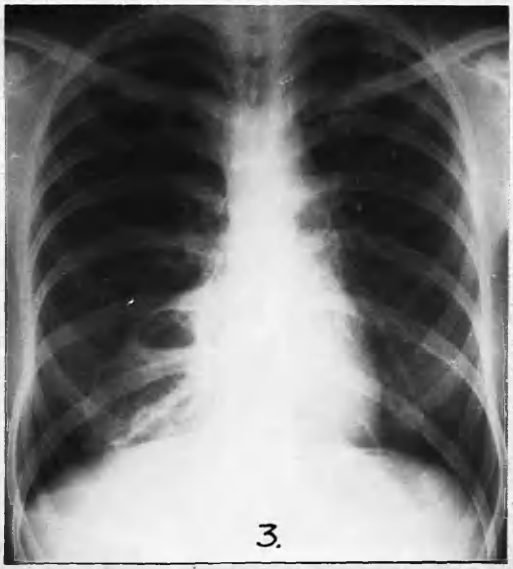

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CASE
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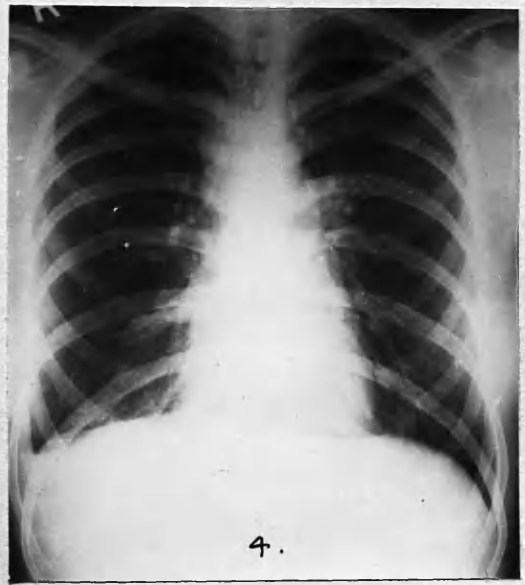

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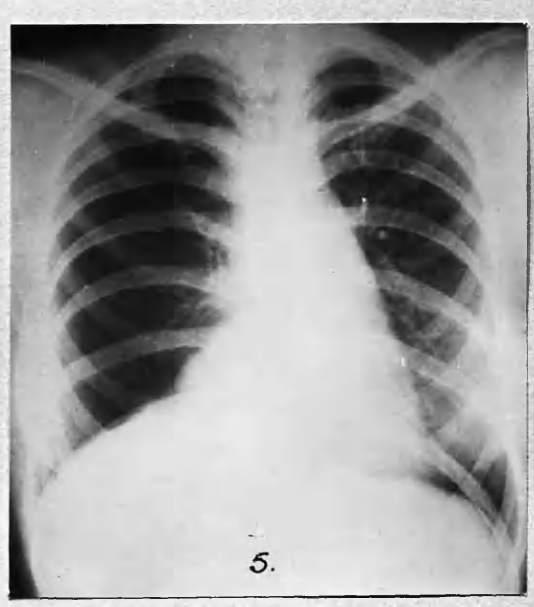

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Case 17:

M.L.D.G. Female. 26 years. Admitted 22/2/45. Discharged 29/5/45.

Insidious onset of symptoms during the 5 months preceding admission.

Examination showed dullness and impaired air entry with rales at the right posterior base.

X-Ray showed infiltration of the right lower zone with a fairly large mid-zone cavity with a well-defined wall (Case 17 - Fig. 1). The left lung was clear.

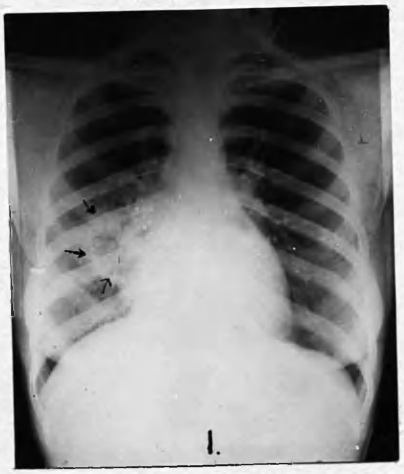
During the first fortnight rest only. Afebrile, very little sputum which was positive. B.S.R. 16.

Patient found it difficult to remain in bed and was anxious to leave hospital as soon as possible. Right pneumothorax induced on 7/3/45. After a month there was a good selective collapse of the right lower lobe but the cavity was still open (Fig. 2). No obvious adhesions were seen on the X-Ray. Right phrenic avulsion was carried out on 18/5/45 which produced a marked rise of the right dome of the diaphragm and rapid closure of the cavity (Fig. 3).

The patient then took her own discharge from the hospital and has continued refills at the clinic up to the present (nearly 4 years). General condition has remained good throughout. Some fluctuation in the degree of collapse has been noted from time to time and in July 1948, when a fair degree of re-expansion had been deliberately allowed, tomograms revealed a small cavity at the site of the original one and the lung was re-collapsed, since when the cavity has remained closed (Fig. 4).

She has recently (October 1948) been delivered of a child. More recently still (March 1949), a small effusion was noted and a specimen of this showed a clear fluid negative for tubercle bacilli.

CASE 17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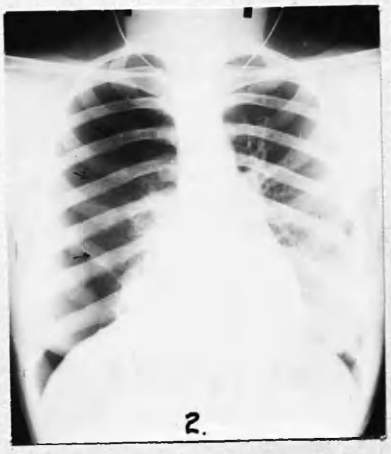
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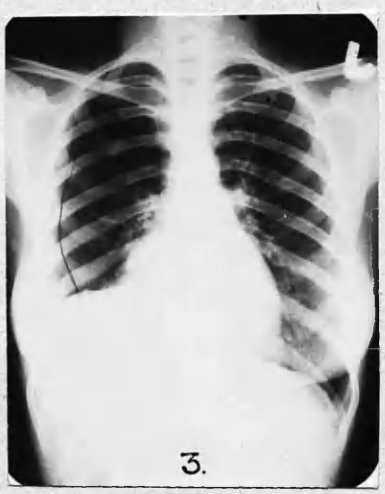
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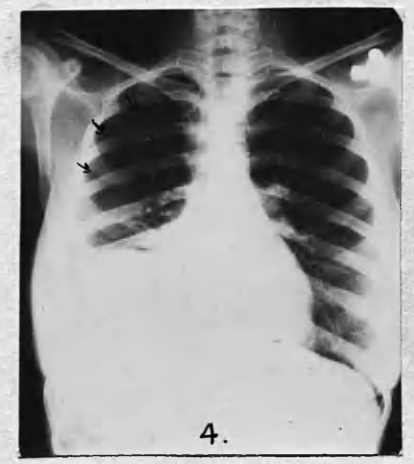
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Case 18: N.C.P. Male. 26 years. Admitted 15/5/46. Discharged November 1946.

This patient took ill with a "cold" and pain in the right lower axillary region of a pleuritic nature 2 months before his admission to hospital. He remained on duty, however, for about 6 weeks after the onset. Symptoms at the time of admission were: a little cough and sputum, night sweats, feverishness and pleuritic pain in the right side of the chest. Also loss of weight of 23 lbs. in the preceding two months, dyspnoea and hoarseness.

Examination revealed a localised area of bronchial breath sounds and rales between the 5th and 6th dorsal vertebral spines and the medial border of the right scapula. All other areas were clear. The larynx showed an area of congestion and oedema in the inter-arytenoid region. X-Ray: Fairly dense infiltration in the lower half of the right lung field with a moderately large hilar cavity (apex of lower lobe). The left lung was clear.

Treatment for the first month was bed rest and vocal rest with inhalations. The sputum was positive and the B.S.R. was 78. Right pneumothorax was then induced and a good concentric collapse was obtained, the cavity becoming smaller in the 2 months which followed but not closing.

Thoracoscopy was carried out on 13/8/46 and a thick short fleshy adhesion was found attaching the apical part of the lower lobe to the parietes in the paravertebral gutter. A small area of this was freed but complete section was not considered safe and the amount done was unlikely to affect the collapse. Right phrenic crush was carried out shortly after. By the time of discharge the cavity was no longer visible and the sputum was negative.

The patient returned to duty in February 1947 and refills were continued at the clinic. His health was good until June 1947 when he developed an acute febrile pleural effusion which required aspirating several times up to August, after which the fluid gradually dried up. This was followed by marked pleural thickening and some obliteration of the basal portion of the pleural space. Small refills at long intervals still continued up to March 1949 and the patient is in good health and devoid of symptoms. Except for a short period at the onset of the effusion, he has continued on duty as a warder.

Case 19: M.V. Female. 32 years. Admitted 25/6/45. Discharged 30/6/46.

This patient was examined as a contact of her sister who died of pulmonary tuberculosis in April 1944. X-Ray of the chest at that time was clear but, re-examined in January 1945, evidence of tuberculosis was found, although the patient had not at that time any other signs of ill-health. On admission the only complaint was very slight cough and she could raise a little sputum with an effort.

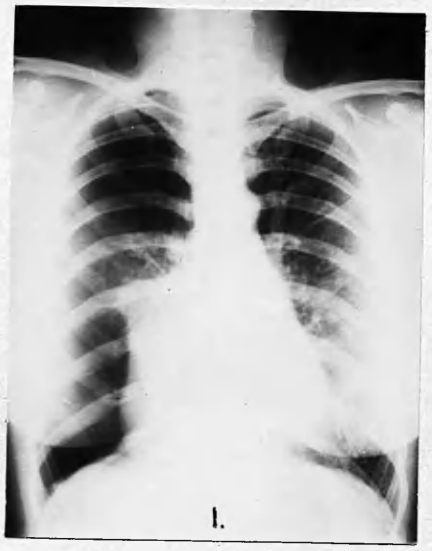
Examination generally revealed no abnormality but at the right posterior base there was an area of dullness on percussion with bronchial breath sounds. X-Ray on admission showed tuberculous infiltration and a small cavity at the right hilar region with a small area of infiltration in the left outer mid-zone (Case 19, Fig. 2).

Bed rest only was carried out for 2½ months. During this time she was afebrile and gained weight satisfactorily. However, she had a small amount of sputum which was positive and her B.S.R. remained raised. In addition, the X-Ray showed the right hilar cavity to persist unchanged and also a little more infiltration in the left mid-zone.

Right pneumothorax was, therefore, carried out on 19/9/45 and this caused a collapse of the right lower lobe without, however, cavity closure (Fig. 2). No obvious adhesions were seen, although these probably existed in the paravertebral gutter. No thoracoscopy was carried out but a right phrenic crush was performed on 16/11/45 and this led to a good rise of the diaphragm and disappearance of the cavity after about a month. A month later a small effusion developed with some febrile reaction but no aspiration was required and the fluid absorbed fairly quickly.

At the time of discharge the cavity was closed, the pleural space dry and there was no cough, sputum or signs of activity. A year later gradual obliteration of the pleural space commenced from below upwards (Fig. 3) and, although small refills at positive pressures were continued until early this year, the process is now (March 1949) almost complete. The patient remains in good health, without symptoms and there has been, so far, no sign of the cavity re-opening.

CASE 19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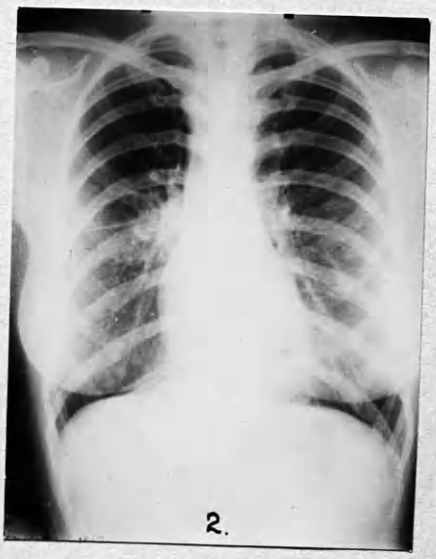
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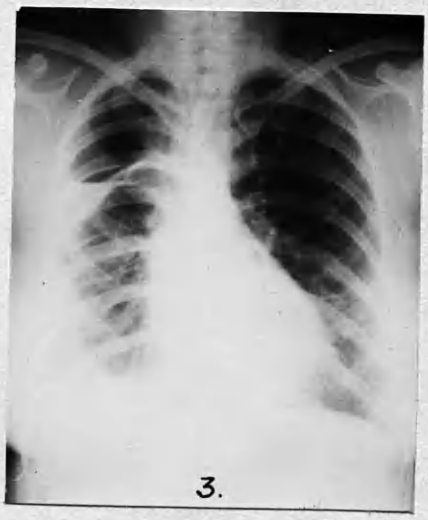
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Case 20:

E.E.K. Male. 21 years. Admitted 6/12/45. Discharged 17/12/46.

Insidious onset following measles 2 years before admission with episodes of pleuritic pain and small haemoptyses. X-Rays had failed to show any abnormality until a few months before admission.

On admission there were signs of involvement at the right posterior base and also in the upper zone. X-Ray at this time showed a fairly large right posterior basal cavity with a small area of infiltration in the second right anterior interspace. The left lung was clear.

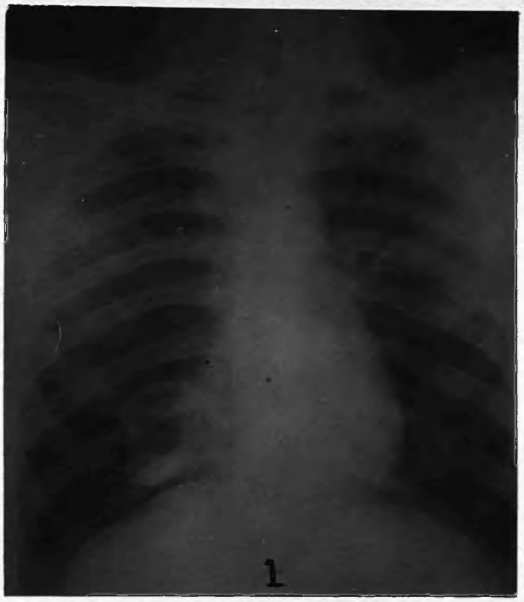
After a month of bed rest, the condition was unchanged. The B.S.R. was slightly raised and the sputum was positive. Pneumothorax was considered advisable.

Right pneumothorax was induced on 16/1/46 and a basal collapse obtained (Case 20, Figs. 1 and 2). After 2 months of refills the cavity still remained patent and thoracoscopy was carried out on 15/3/46. This showed a vascular adhesion to the diaphragm and a short adhesion to the apex of the lower lobe, neither being cuttable with safety.

Right phrenic crush was then done on 2/4/46 and 3 weeks later pneumoperitoneum was induced (Fig. 3). From then until discharge, the pneumothorax and the pneumoperitoneum were kept up and the cavity was not seen after August.

After leaving hospital he went to Cape Town where the phrenic crush was repeated and the pneumothorax abandoned. Thereafter pneumoperitoneum refills were kept going, refills being through a lower rib space. About September 1947 refills were inadvertently given above the diaphragm and a basal pneumothorax space established (Fig. 4). The cavity remained closed and refills have been given into this space up to the present (March 1949). (Fig. 5).

The patient is well, working and the cavity has remained closed since about August 1946.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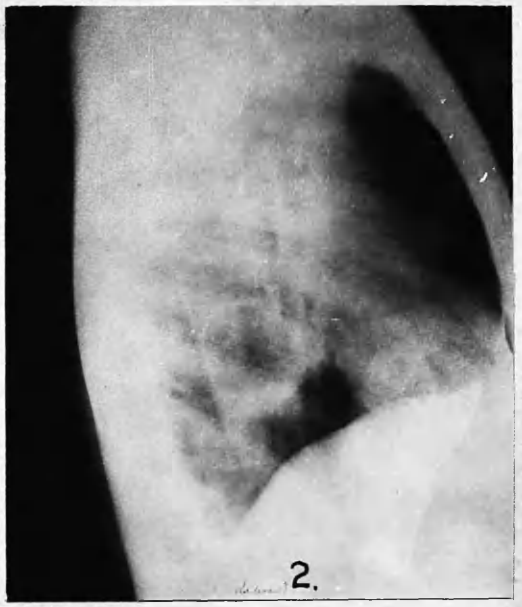
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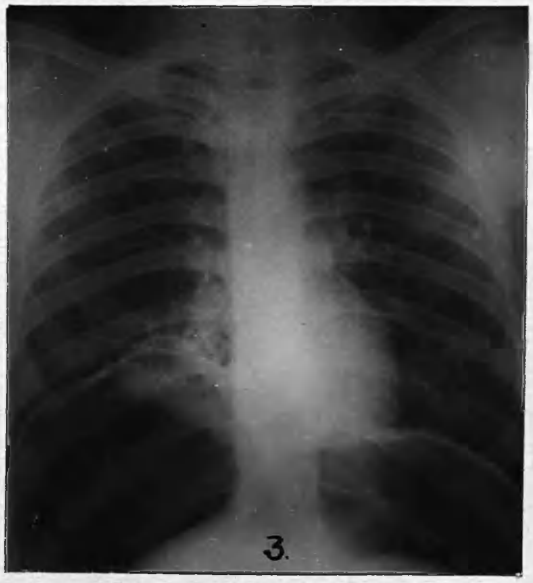
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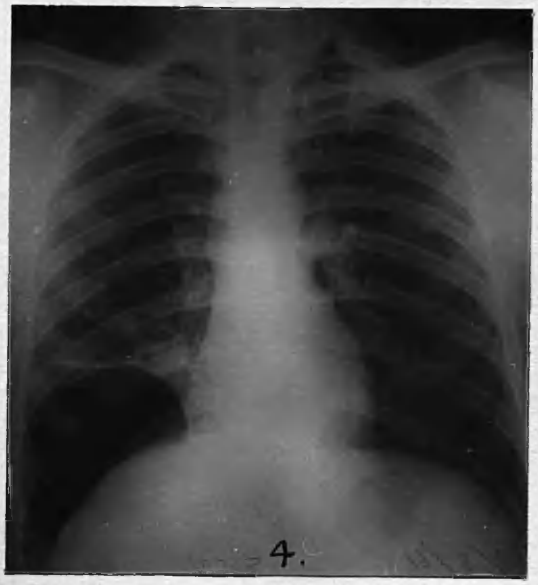
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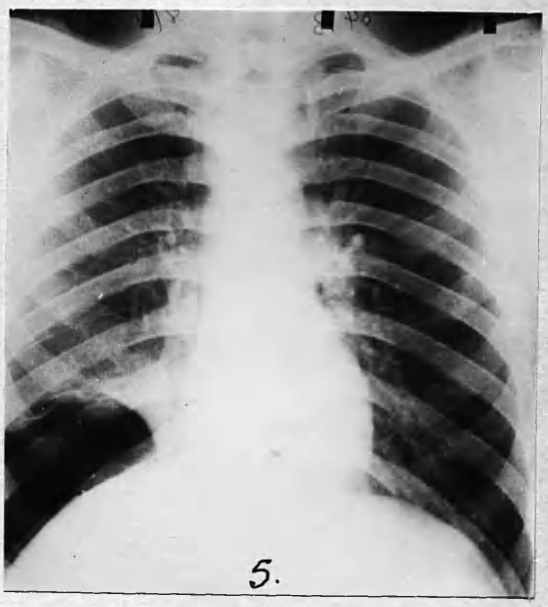
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Case 21:

A.J.B. Female. 22 years. Admitted 23/9/47. Took own discharge 11/12/47.

One year history of cough, sputum and pleuritic pain in the right chest. Bad family history. Father and husband have pulmonary tuberculosis and infant died of meningitis.

Physical signs - dullness to percussion at right base back and front with numerous rales.

X-Ray: Scattered infiltration throughout right lung-field with a large cavity lateral to the right hilum at the apex of the lower lobe and several smaller cavities in the upper third of the lung. Also scattered infiltration and a small cavity in the upper third of the left lung.

Rested for the first 6 weeks. Small daily rise of temperature. Sputum positive. B.S.R. 15. Slight weight gain. X-Ray unchanged at the end of this time.

Right pneumothorax induced 22/10/47. There was some collapse of the right upper lobe but the cavity became markedly distended. Small refills were continued for 4 weeks when an acute febrile pleural effusion occurred. The fluid was clear and serous but positive for tubercle bacilli. The pneumothorax was abandoned forthwith and the pleural space aspirated dry. Re-expansion took place promptly, leaving a small residual effusion but the patient refused any further hospital treatment.

Case 22:

E.H. v. W. Female. Admitted 24/12/47. Died 2/2/49.

Pleuritic onset 7 months before admission, at which time there were marked symptoms. Examination at this time showed quite marked wasting generally and signs of involvement of the left lung.

X-Ray showed a loss of translucency over the lower two-thirds of the left lung with a fairly large mid-zone cavity (below the apex of the lower lobe), probably some left basal effusion. The mediastinum was displaced towards the left and the left dome of the diaphragm was slightly elevated. There was also a moderate degree of infiltration in the right outer mid-zone (Case 22, Fig. 1).

Fairly far-advanced, acute disease, possibly pneumonic with a cavity which showed some tension element.

During the first 3 weeks further investigation showed a daily rise of temperature to 100, B.S.R. 79 and sputum positive. It was decided to induce a left pneumothorax to attempt closure of the left basal cavity and prevent further extension of the disease on the right.

This was done on 14/1/48 (Fig. 2). This was followed after a few days by an acute febrile episode with pain in both sides of the chest. The fever settled after 10 days but X-Ray on 28/1/48 showed a wedge-shaped area of consolidation replacing the infiltration previously noted on the right (Fig. 3). There was some ballooning of the cavity on the left side. Six weeks later the left pneumothorax was abandoned as the cavity was still further distended and cavitation had commenced on the right side. A small effusion had also appeared on the left.

Right pneumothorax induced on 31/3/48. Numerous adhesions were seen over the diseased area and it was not found possible to obtain enough collapse for thoracoscopy (Fig. 4). Refills, however, were continued for $2\frac{1}{2}$ months when a small effusion appeared on this side; the pneumothorax was abandoned.

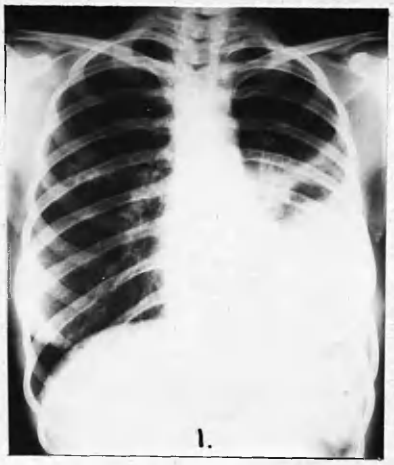
From this time on there was a gradual worsening in the patient's condition. By September, both lungs had markedly expanded and a large cavity was seen on each side and also bilateral effusion (Fig. 5). That on the left was aspirated once-- clear fluid - positive for tubercle bacilli. There was daily fever to 100 - 101, a large quantity of sputum and a high B.S.R.

Streptomycin was given in a dose of 1 Gm daily during the last month of life and there was a fall in temperature and reduced sputum output.

Note:

It seems that a better preliminary treatment would have been pneumoperitoneum with possibly left phrenic crush. The left pneumothorax could also have been abandoned more promptly when seen to be ineffective. The earlier use of Streptomycin - when the flare-up on the right side took place - might also have been advantageous.

CASE 22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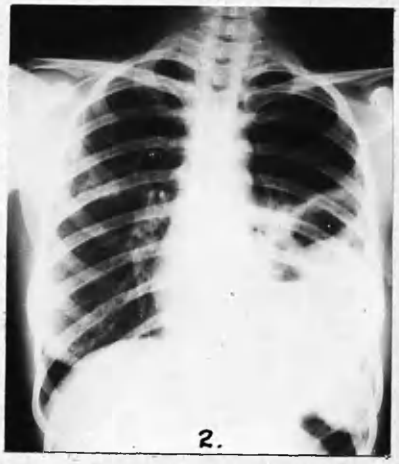
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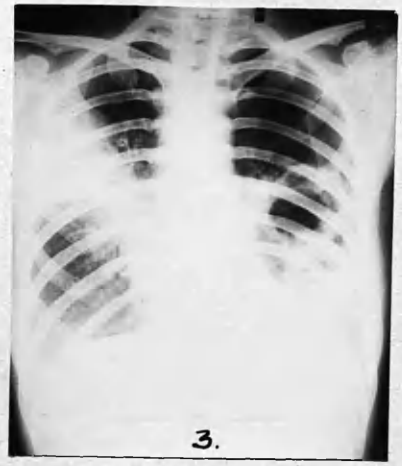
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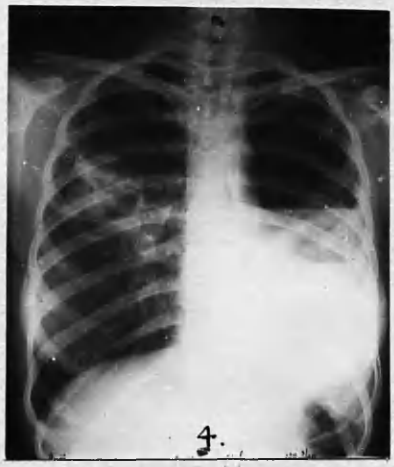
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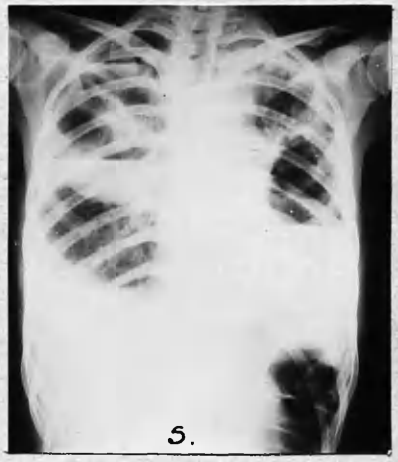
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The foregoing seven cases are detailed because they are all concerned with the treatment of cavitation in the lower lobe. Cases 16, 17, 18 and 19 had the ordinary types of cavity commonly encountered at the apices of the upper lobes. The treatment consisted firstly of pneumothorax and, as this was insufficient to effect cavity closure, phrenic crush was added and the rise of the diaphragm together with continued pneumothorax caused the cavity to close successfully. Only in Case 18 was thoracoscopy carried out and a short uncuttable adhesion to the apex of the lower lobe was found. Adhesions were probably present in the other cases also. Such adhesions are difficult to see on the X-Ray plate, even on the lateral view.

Cases 16 and 17 had cavities of tension type, yet no particular difficulty was encountered in closing them.

In Case 21 the cavity was of this type also but, on inducing pneumothorax, it became markedly distended and the collapse had to be rapidly abandoned.

Case 22 is another poor result for this form of collapse.

Case 20 was a true basal cavity. When the lung was collapsed by pneumothorax, the lower lobe was unable to contract concentrically because of uncuttable adhesions at its apex and base. Longitudinal relaxation was effected by phrenic crush causing a rise of the diaphragm.

Cavities at the apex of the lower lobe present the same problems as to closure as do those at the upper lobe apex. Concentric collapse seems to be somewhat easier to obtain as they lie, so to speak, at the hub of the wheel, the rim of which is formed by the apex of the lung and the diaphragm. Chest wall pull commonly has to be overcome, either by cutting the adhesions to the paravertebral gutter or by preventing the downward movement of the lung root by phrenic paralysis supplemented, if need be, by pneumoperitoneum.

Rapid concentric relaxation of the lung depends upon the absence of significant adhesions over the diseased area. As will be mentioned, adhesions were present in the majority of cases in the present series. In two of the cases where there were no adhesions, one had a small recent focus below the apex of the upper lobe and the other a small focus below the apex of the lower lobe. Rapid selective collapse was obtained in both with pneumothorax.

Case 23:

I.B. Female. 30 years. Admitted 2/8/46. Discharged 25/1/48.

Insidious onset about a year before admission following a cold. Fairly marked symptoms and signs of involvement of the left upper third on admission. X-Ray at this time showed a moderate degree of scattered infiltration throughout the left lung-field with a moderately large sub-apical cavity. In the right lung there was a small area of infiltration in the second anterior interspace.

As this patient had already had two months rest in hospital elsewhere, it was decided to induce left pneumothorax immediately. This was done on 7/8/46, but there were a good many adhesions holding up the apex and the collapse was mostly basal although the cavity became smaller. On 6/12/46 thoracoscopy and incomplete adhesion section was carried out. A month later there was complete atelectasis of the left lung and an effusion had developed which required aspiration. A month after this a further thoracoscopy was done but, as the pleura was thickened and sodden, no adhesion section was attempted. Thereafter attempts were made to expand the lung by withdrawing air and this was successful. A shallow collapse was continued until the effusion seemed to have settled down. The fluid aspirated was always thin and clear.

On 26/9/47 (thirteen months after induction) left extrapleural strip with fenestration was performed. Convalescence was uneventful; only one aspiration of bloody fluid was required. Thereafter, a good collapse of the lung was obtained with cavity closure and sputum conversion. The patient left hospital four months later and refills continue.

The foregoing case illustrates how successful extrapleural strip with fenestration can be, provided a good strip is obtained and no infection supervenes. Cases 11 and 12 show the unsatisfactory effects of infection of the pleural and extrapleural space.

Thoracoscopy and Adhesion Section in the present series:

Adhesions visible on X-Ray were present in practically all cases. In seven none were actually visible but four of these had cavities at the apex of the lower lobe, a condition associated in my experience with short adhesions in the paravertebral gutter which can often only be shown with difficulty by lateral or oblique views. Two others of these seven had each a focus of disease so small and so early (radiological evidence), that no adhesions had had time to form before collapse was instituted. Four patients refused to have thoracoscopy although adhesions were seen. Thoracoscopy was not attempted in fourteen patients as the nature of the adhesions present was such that any section seemed quite out of the question. One hundred and six thoracoscopies were carried out on seventy-five patients. In seven the pleural space was inspected only and no adhesion section attempted. For the others, success in closing the cavity was directly proportionate to success in cutting the adhesions which held up the diseased area of the lung.

Of thirty pneumothoraces in which adhesion section was complete, cavity closure was obtained in twenty-seven (although one of these developed empyema). The pleural space obliterated rapidly in another and slowly in a further case and the pneumothorax was given up because of empyema in another.

Of fifty pneumothoraces in which the adhesions could not all be completely divided, cavity closure was only obtained in twenty-five; in thirteen the cavity became smaller and in a further ten the cavity was unchanged; two developed empyema.

C O N C L U S I O N S

Artificial Pneumothorax has again been shown to be a useful form of treatment of pulmonary tuberculosis but only in so far as it is successful in effecting cavity closure and the conversion of the sputum.

If it cannot be made effective within a reasonable time, it should be given up or supplemented by some other procedure designed to bring about cavity closure. In certain cases where it is considered that the patient is unfit for major surgery, it may be justifiable to continue a partially effective collapse for some time in the hope that he may become fit for the major operation.

Pneumothorax should be used for those cases requiring a reversible collapse procedure. A re-expanded functioning lung with a healed lesion is the mark of success. An unexpandable or partially expanded lung with marked loss of function is, to a certain extent, a failure.

The field of usefulness of artificial pneumothorax is becoming smaller as the surgery of the chest improves and also as it is being more and more realised that pneumothorax is an ineffective and dangerous procedure in the presence of obstructive bronchial tuberculosis. It is likely to find a wider application as the disease is diagnosed at an earlier stage.

Tuberculous empyema is a complication with a serious prognosis, especially from the long-term point of view. In the present series it has been shown that a proportion of such cases are preventable by a better selection of cases for pneumothorax but, in certain cases, the risk of its occurrence must be taken if the patient is not to be denied effective treatment.

The treatment of cavitation at the apex of the lower lobe by pneumothorax is essentially the same as that at the upper lobe apex but is made more difficult by the fact that short uncuttable adhesions frequently occur and a combination of pneumothorax and phrenic paralysis is usually required before cavity closure can be effected.

The section of adhesions is a technical procedure depending for its success on good fortune in finding adhesions suitable for division and on the skill of the operator.

SUMMARY

1. The history of artificial pneumothorax treatment is outlined, especially the development of the indications for the use of this form of treatment.

2. The anatomy of the lung is described in some detail, emphasis being laid on the elasticity of the various pulmonary and pleural tissues.

3. The pleural and pulmonary dynamics are discussed with reference to artificial pneumothorax and stress is laid on the importance of the bronchial movements and calibre changes.

4. The pathology of pulmonary tuberculosis is touched upon at points which concern this work.

5. Cavities are classified and reference made to the various forms of cavity closure and healing.

6. The importance of the bronchial factor in the evolution, persistence, behaviour and closure of cavities is emphasised.

7. A short note on adhesions is inserted.

8. The rationale of collapse therapy is next discussed as are also the indications and contra-indications for pneumothorax treatment in the light of present-day knowledge. The difficulty of obtaining a satisfactory collapse by pneumothorax in the presence of active bronchial tuberculosis is noted.

9. The technique of collapse therapy by artificial pneumothorax is described. It is only permissible to use this form of collapse as a temporary collapse measure and emphasis is laid on the fact that this is a relaxation procedure and not a compression of the lung.

10. The complications pleural effusion and adhesions are discussed, stress being laid on the prevention and management of the former.

11. Various methods of assessing the results of pneumothorax treatment are next discussed. The results to date in a personal series of one-hundred cases having pneumothorax treatment are set forth. It is confirmed that artificial pneumothorax, in so far as it is effective in obtaining cavity closure, is a useful form of treatment but, when it is ineffective, it must be given up in favour of some other form of treatment.

12. A number of cases of empyema are discussed with special reference to the prevention, where possible, of this complication.

13. Certain further cases are described in some detail as throwing light on the handling of pneumothorax with tension cavity and also the difficulty of closing cavities in the lower lobe.

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