ON THE RESULTS OF TREATMENT OF PULMONARY TUBERCULOSIS BY ARTIFICIAL PNEUMOTHORAX.

BY

Charles Metcalfe Brown,

M.B., Ch.B., D.P.H.

ProQuest LLC.
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106 – 1346

ON THE RESULTS OF TREATMENT OF PULMONARY TUBERCULOSIS BY ARTIFICIAL PNEUMOTHORAX.

INTRODUCTION.

The aim of this thesis is to consider the modern Artificial Pneumothorax Treatment of Pulmonary Tuberculosis with special reference to a series of one hundred cases and to analyse these cases with a view to the assessment of the effects of this special treatment, and to compare these effects with those observed by other physicians and also with those obtained by purely expectant treatment alone.

In spite of all known forms of treatment, the vast majority of patients suffering from pulmonary tuberculosis are necessarily handicapped for the remainder of their lives and the great probability is that the disease will shorten and eventually terminate their existence. The chief method of treatment is undoubtedly the application of general rules of hygiene, which combined with continuous medical supervision of the activities of the individual patient, form the basis of sanatorium treatment.

The results of this method of treatment alone are disappointing; a few patients are discharged as quiescent and may eventually be classed as arrested, the majority are improved and return to their homes either to spend the remainder of their lives as semi-invalids or to deteriorate gradually to their former condition or worse. The remainder fail to respond to the treatment and either die in the institution or are discharged as unimproved or worse.

Artificial pneumothorax in carefully selected cases is a very real adjuvant to ordinary sanatorium treatment in that the disease is attacked locally and has the effect of hastening and fortifying improvements produced by treatment on general hygienic principles. Further, artificial pneumothorax may be expected to produce improvement in many cases where sanatorium treatment alone would fail.

The principle of artificial pneumothorax is essentially that of collapsing and maintaining the collapse of the whole or part of a diseased lung by means of the insertion of a gas between the parietal and visceral pleura of that lung. This collapse is designed to assist the healing of the pulmonary

lesions by limiting or abolishing the respiratory movement of the affected lung, by reducing the local circulation of blood and lymph and by approximating open lesions to each other and to surrounding structures.

This special treatment demands the exercise of patience and the exhibition of faith on the part of the patient and involves the expenditure by the physician of much time and care. The primary cause of this investigation was a desire to discover the average return on the outlay of human endeavour on this form of treatment in Middlesex.

This thesis is based on the cases of the first hundred patients who received this form of treatment at the County Sanatorium, Harefield, Middlesex. The majority of these cases were observed and treated by me between June 1925 and January 1928. The after-histories of patients were investigated by me in September, 1928 and in December, 1929 at the Sanatorium and at the Tuberculosis Dispensaries in the various districts of the County.

I am indebted to Dr. J. Tate, County Medical Officer of Middlesex and to Dr. J. R. Mc.Gregor,

Medical Superintendent at Harefield for allowing me facilities for collecting information.

HISTORICAL.

Treatment by pulmonary compression was first advocated by Carson of Liverpool in 1822 (1). His main theme was that if the diseased parts of a lung were kept in an undisturbed state by means of pulmonary collapse and the sides of a lung "abscess" placed in "salutary contact", favourable circumstances for healing would be achieved. Carson realised also the potential value of compression of the lung in the treatment of haemorrhage for he deduced that "a haemorrhage (sic) from one of the lungs..... if not immediately fatal, would be as certainly stopped by the collapse of that lung, as the flooding consequent upon parturition, by the contraction or rather the resilience of the womb". Carson, however, does not appear to have put into practice his recommendation that operative means should find a place in pulmonary tuberculosis treatment.

The technique of modern artificial pneumothorax treatment was first elaborated and published by Forlanini (2) his first publication on the possibilities of the treatment appearing in 1882.

Prior to Forlanini's publications, various

observers had noted that marked improvement resulted from spontaneous pneumothorax in patients suffering from pulmonary tuberculosis, and in 1885, Cayley of the Middlesex Hospital, London, had reported the treatment of a case of severe haemoptysis by pleural incision (3). About this time, interest in producing pulmonary collapse by mechanical means had been aroused in several countries, and in 1885 before the value of artificial pneumothorax had been proved, de Cérenville of Lausanne had performed his first thoracoplasty (4).

The treatment so far had been confined to Europe and more especially to Italy, but in 1898, Murphy of Chicago (5) published details of five cases and strongly advocated the adoption of the principle of surgical interference in pulmonary tuberculosis. Numerous other workers in many countries followed the path trodden by these pioneers, and the first artificial pneumothorax operation in England was performed in 1910 by Lillingston (6) who had himself derived considerable benefit from treatment by this method in a Norwegian sanatorium.

Since then the study and research of many

workers has improved the technique and popularised the treatment and since the war artificial pneumothorax has been accepted as one of the approved methods of treatment. In fact, in 1921, at the Royal Society of Medicine, Sir James Kingston Fowler said that "the induction of artificial pneumothorax is the only advance in the treatment of pulmonary tuberculosis since the introduction of sanatorium treatment as carried out at Nordrach". To-day, that statement requires considerable modification in view of the success of thoracoplasty and of Sanocrysin treatment, but there is no doubt that artificial pneumothorax is a method of treatment which unlike many alleged remedies now discredited, has stood the test of time.

ANATOMICAL AND PHYSIOLOGICAL CONSIDERATIONS.

The framework of the thorax consists of the dorsal section of the vertebral column, the ribs and the sternum. The anterior extremities of the ribs are attached to the sternum by means of the costal cartilages and these latter contribute very materially to the elasticity of the thorax. The ribs by virtue of their strength and relative narrowness also exhibit considerable elasticity. The breadth of the intercostal spaces is greater in front than behind and between the upper than the lower ribs. This is of importance when the site of election for pneumothorax puncture is under consideration.

The ribs form a series of levers, the fulcra of which are in the neighbourhood of the costo-transverse articulations. The power is supplied by the muscles of inspiration, chiefly the Serratus posterior superior so far as the ribs are concerned in ordinary quiet respiration. The necks of the ribs form the short sections of the levers so that when they move downwards a corresponding upward movement occurs at the anterior extremities, and because of the antero-posterior slope of the ribs, the upward

movement of the anterior extremities, with the sternum attached, is accompanied by a forward movement thus enlarging the thoracic cavity antero-posteriorly. The curvature of the ribs increases from above downwards and when the ribs are raised in inspiration the transverse diameter of the chest is consequently increased in the plane to which each pair of ribs is raised. A rise of the body of each rib also occurs relative to its extremities and as the body lies in a plane lower than that of the extremities this rise must also add to the increase in the transverse diameter on inspiration. Thus, by means of the rib mechanism, the thorax increases in size both laterally and antero-posteriorly and the depth is increased by the contraction and consequent descent of the diaphragm.

The lungs form an enclosed cavity without any natural outlet except the trachea. The interior of the thorax may be regarded as consisting of a central mediastinal cavity and two lateral pleural cavities. The mediastinal cavity contains all the thoracic viscera including the trachea and bronchi, and each lateral cavity contains a pleural sac

invaginated by a lung. In health, the pleural sac contains sufficient serous fluid to lubricate the pleural surfaces during the respiratory movements and the pleural space is a potential one only. The parietal wall of the pleural sac forms the inner lining of the chest wall; the visceral layer of the pleura forms the outer lining of the lung. In both cases the pleural membrane is firmly adherent to the underlying structures.

The trachea bifurcates to form the bronchi and these divide and sub-divide until the air tubes have multiplied vastly in number and diminished correspondingly in size. Each of these small bronchial tubes supplies a pulmonary lobule which consisting as it does of air-cells, blood-vessels, lymphatics, nerves and areolar tissues forms a miniature lung. The areolar tissue is chiefly composed of elastic tissue and it is on the elasticity of this tissue and on that of the thoracic wall already discussed that expiration depends. Inspiration is the only active part of ordinary quiet respiration; expiration is a passive elastic recoil.

At the end of expiration, the lung is at rest.

The air of the lung, supplemental and residual, being in communication with the outer air is at the prevailing atmospheric pressure and the lung tissue is therefore pressed from within against the thoracic wall, the lung being ballooned out to fill the available space, and the elastic tissue of the lung subjected to some degree of stretch. The outer air presses on the thoracic wall and so tends to drive it in, but this tendency is overcome by the rigidity of the chest wall supported by the atmospheric pressure from within. In other words, the external atmospheric pressure and the elasticity of the lung tend to cause movement inwards and these are resisted by the combined rigidity of the chest wall and the internal atmospheric pressure. There are thus two equal sets of forces acting in opposite directions and in consequence no actual movement takes place.

On inspiration, however, muscle power overcomes the external atmospheric pressure and the thoracic wall moves outwards. The diaphragm descends and pushes the abdominal viscera downwards and the abdominal wall moves outwards; indirectly, the diaphragm also has overcome atmospheric pressure. The opposing

sets of pressures no longer balance, and the lung tissue is caused to move outwards by the excess of internal pressure. The lung enlarges to fill the larger space and the elastic tissue of the lung stretches still further to allow the lung to accommodate more air. Inspiration is complete, the interchange of gases between the blood of the pulmonary capillaries and the air has taken place and expiration commences.

The diaphragm and the other inspiratory muscles relax, the elastic recoil of the chest wall and the abdominal wall takes place and so the thoracic capacity diminishes once more. The excess of force which caused increased stretching of the pulmonary elastic tissue is gone and the lung in turn recoils, the tidal air is expressed, the opposing forces are once more equal and expiration is complete.

If a wound be made through the chest wall including the parietal pleura, the external atmospheric pressure acts directly on the visceral pleura. The internal atmospheric pressure is thus neutralised and further elastic recoil of the lung

takes place, and air flows through the wound and converts the potential pleural space into an actual one. A pneumothorax is formed and a collapse of the lung commensurate with the amount of air admitted results. This collapse is somewhat retarded by the force of cohesion resulting from the apposition of the smooth pleural surfaces with only a thin layer of serous fluid between. This force however is gradually overcome, the layers of pleura peel apart and the pulmonary collapse eventually becomes complete.

The normal intrapleural pressure has been determined by several workers by experiment, and the results are of importance for comparison with the initial pressures recorded at the induction of artificial pneumothorax. Heynsius (7) quotes Donders as finding that at the end of quiet inspiration the intrapleural pressure was - 12.2 cm. of water and at the end of quiet expiration - 10.3 cm. Halliburton (8) states that the intrathoracic pressure varies from - 6.8 cm. to - 9.5 cm. of water at the end of expiration to - 40.8 cm. at the end of a deep inspiration. The average findings of these

and other workers appear to be from - 8 cm. of water at the height of quiet expiration to - 13 cm. at the height of quiet inspiration. Normally, therefore each pleural cavity has a negative pressure. This pressure falls still lower at each inspiration, owing to the enlargement of the thorax and the tendency of the lung to lag behind as it makes its inspiratory excursion. In obstructed expiration the intrapleural pressure is a markedly positive one. An accident that may occur during an artificial pneumothorax operation strikingly demonstrates this fact. If a patient coughs while a water manometer is in connection with a pleural cavity, by means of a rubber tube and cannula, the intrapleural pressure is made so markedly positive that, unless suitable precautions have been taken the water will, in all probability, be blown completely out of the manometer.

The question of the relationship of the pleural cavities has been investigated by Graham and Bell (9). These workers measured the intrapleural pressures in a fresh human cadaver and having raised the pressure on one side by inserting air, found that on the opposite side an almost equal rise of

pressure had occurred. Parallel results were obtained with a freshly killed dog, thus establishing the similarity of conditions in the dog and in man. With the living dog, the same experiments were performed with the same results. They thus concluded that from the standpoint of pressure relationship, the normal thorax may be regarded as one cavity instead of two and that no matter what change of pressure is made in one pleural cavity, an almost equal change results in the other. They estimate the resistance of the mediastinum to be equivalent to the pressure of 1 cm. of water except when the mediastinum has been made rigid by induration following chronic inflammation. This attitude is supported by Amberson and Peters (10) who state that "on the functioning side, the intrapleural pressure is actually raised and the expansibility of the lung is proportionately decreased". It must be added, however, that Amberson and Peters do not adduce any experimental evidence to support their opinion.

These American views are not endorsed by British authorities. Burrell and MacNalty (11) conclude "that in compressing one lung by artificial pneumo-

thorax one does not interfere with the pressure in the opposite pleural cavity, even if the mediastinum is displaced". The basis on which this opinion is founded, however, is somewhat doubtful in that bilateral pressures were taken in two cases only, one of which was a case of pleural effusion. Either or both of these cases may have had rigid mediastina following inflammatory processes.

Riviere (12) concurs with the conclusion of Burrell and MacNalty, but like Amberson and Peters, he fails to give evidence in support of his views.

The question is one which cannot be solved by experiments on the chest of a normal man for obvious reasons, but a study of the variations of intrapleural pressures in artificial pneumothorax is of assistance. It is frequently observed during a pneumothorax refill or soon after a refill, that there is a gradual but definite fall in the intrapleural pressure on the treated side. This is explained by the fact, as Emerson (13) demonstrates, that the thorax has considerable accommodative powers and is capable of counteracting increase of intrapleural pressure by an enlargement of the thorax

produced by unconscious elevation of the ribs and descent of the diaphragm. Further, in cases of artificial pneumothorax at Harefield, it was noted that the posture of the patient had a considerable influence on the pressure in the pneumothorax cavity. If the patient lay on the treated side, the pressure in the pneumothorax cavity was greater than if he lay on the sound side. This phenomenon was observed by Burrell (14), and has been proved by him by means of X-rays to be due to the movement of the intra-thoracic viscera under the influence of gravity. Also, the movement of the mediastinum under these conditions can usually be demonstrated by the shifting of the apex beat.

Since the movement of the mediastinum under the influence of gravity is capable of causing an increase of intrapleural pressure on the lower side, it follows that if in a normal chest, the influence of posture having been eliminated, the intrapleural pressure is raised on, for instance, the left side by the insertion of air, a movement of the mediastinum to the right side will take place, causing an

increase of intrapleural pressure on that side. Further it is reasonable to deduce that this increase will be negatived to some extent by the accommodative powers of the thorax. Similarly, on the left side the same process will take place and by virtue of its elasticity the mediastinum will tend to return to its original position so hastening the fall of pressure on the right side and retarding the fall on the left side. The net effect will be that the intrapleural pressure on the right side will fall markedly, possibly to normal, and the intrapleural pressure on the left side will also fall but to a less extent, producing a difference in intrapleural pressures.

These deductions accord with the conclusions of Graham and Bell in that, following insertion of air on one side an initial uniform intrathoracic pressure is postulated, and these authors also observed that there is a gradual fall in pressure in closed pneumothorax (9). The deduction that there should be a final difference of intrapleural pressures on the two sides, accords with the clinical observations of Burrell and MacNalty.

Finally, whether or not there is uniformity of intrathoracic pressure, the fact remains, proved by the experience of many physicians that the contralateral lung in artificial pneumothorax carries its extra load with efficiency and little or no respiratory embarrassment normally results.

PATHOLOGICAL CONSIDERATIONS.

In all forms of tuberculosis, the deposit of the Bacillus tuberculosis in a tissue produces sooner or later, the chronic inflammatory process known as a granuloma, the essential element of which in this disease, is the tubercle. As a result of the bacillary infection, local proliferative changes take place, producing highly vascular granulations in the centre of which develop typical multinucleated giant-cells and numerous mononuclear cells. A few tubercle bacilli are found in and amongst these cells. This tuberculous granulation tissue rapidly becomes non-vascular and forms a semi-translucent mass or gray tubercle, the centre of which undergoes a coagulation necrosis or caseation. At the same time, the connective tissue cells of the tubercle attempt to enclose the mass in a capsule of fibrous tissue. If the resistance of the infected tissue is sufficiently strong, this attempt succeeds and the bacilli and caseous material become encapsulated and the progress of the tuberculosis is checked. If however, these efforts at repair fail, owing to the weak resistance of the tissues relative to the

strength of the infection, the tuberculous process spreads, involving surrounding tissues and frequently fusing with other similar foci to form a large mass of tubercles with a yellow caseated centre. Particles of the infective debris may be carried by the blood or lymph stream to other parts of the affected organ or to other organs of the body and so the disease is disseminated.

In pulmonary tuberculosis, the site of the initial deposit is usually the wall of a smaller bronchiole, and the surrounding alveoli become filled with caseous material, and the alveolar walls show evidence of fibrosis except towards the centre of the diseased area where the alveolar walls are also involved in the general caseous degeneration. Beyond the area of fibrosis, the alveoli are subject to catarrh of the broncho-pneumonic type. Thus the diseased tissue consists of a central area where the tubercle bacilli have been victorious, a surrounding area of fibrous tissue formation of varying extent where the fight is still being waged, and beyond this, a skirmishing area where the toxins of the infection have irritated the surrounding healthy

tissue into an inflammatory protective state. The disease may spread by direct infiltration or by the blood or lymphatic streams and in some cases by aspiration of infective material into a healthy bronchiole.

In the acute caseous type of the disease, there is rapid formation of large areas of softening with little or no attempt at fibrous tissue formation. All stages of this softening may be found in the lungs and masses of the caseous material may be expectorated, leaving cavities with soft ragged walls.

More usually, in the fibro-caseous form of pulmonary tuberculosis, the healing and the degenerative processes are more nearly balanced and various stages of the disease are always present in the lung. The older lesions show evidence of considerable fibrosis, while the more recent lesions show marked caseation and are surrounded by tissues in a broncho-pneumonic state. Cavities are also found in this type of disease and they may be quiescent with smooth fibrous walls or have an outer fibrous wall and an inner ragged wall discharging pus. These cavities are sometimes trav-

ersed by trabeculae consisting of vessels or bronchi, and aneurisms of the blood vessels in the walls or in the trabeculae may exist. Rupture of such an aneurism causes haemoptysis. Fibrous tissue near a cavity tends to increase and adjacent pleura may be much thickened in consequence. In some cases, areas of caseous material may be surrounded by a thick fibrous capsule and so remain innocuous so long as the capsule is unruptured. The process may be carried further, and calcification of the caseous area may occur rendering it non-infectious.

Where a great excess of fibrous tissue is produced throughout the lung or a lobe of the lung, the affected area becomes contracted and firm and the pleura is much thickened and adherent. This is the fibroid type of the disease, and bronchiectasis may result from the pull of the contracting fibrous tissue on the weakened walls of the bronchi. By the same force, the mediastinum may be pulled over towards the affected side, and a compensatory emphysema may occur in the unaffected lung areas.

The pleura is constantly affected in pulmonary tuberculosis. Early pleurisy of the dry form occurs

and this may go on to considerable thickening with formation of adhesions between the lung and the chest wall. Adhesions are most constantly found over the older lesions, more especially over large superficial cavities. In apical cavity formation, marked pleural thickening and adhesions are commonly found as a result of the relatively large area of pleura in close proximity to the tuberculous lesion. In acute cases, the pleura may be studded with miliary tubercles leading to serous effusion and so frequently causing collapse of the whole or part of the lung. The tracheo-bronchial glands are invariably involved in pulmonary tuberculosis and may be caseous, fibro-caseous or calcified.

From the pathological processes that take place in the lung in cases of pulmonary tuberculosis, it is readily seen that nature makes strenuous efforts to eliminate or diminish the effects of the infection. Impenetrable barriers of fibrous tissues are raised in some cases, which form effective mechanical checks to the progress of the disease. Fibrous tissue contracts as it grows older and the effect of this contraction is to close vascular

communication between the diseased area and the rest of the body, thus localising infective material and harmful toxins. Pleuritic thickenings and adhesions over cavities are means of strengthening the walls of the cavity and so diminishing the risk of the rupture of the cavity into the pleural sac and the consequent infection of the latter. Pleural effusion might also be regarded as a means of collapsing the lung to give its diseased portions the rest necessary for the curative process.

THE RATIONALE OF ARTIFICIAL PNEUMOTHORAX TREATMENT.

When artificial pneumothorax is induced, whether cavitation is present or absent, the destruction of the vacuum in the pleural cavity is followed by elastic recoil of the pulmonary tissue and consequent contraction of the lung. The contraction of the lung is naturally followed by expulsion of the more fluid pathological products, and this is demonstrated by the initial increase of the daily quantity of sputum that frequently follows the induction of artificial pneumothorax. Thus the compression of the lung, at an early stage in treatment, assists in the removal of infective material from the lung.

Rest is essential to the healing of any lesion, for one cannot be other than impressed by the excellent results obtained in the treatment of bone tuberculosis, the essence of which is immobilisation. The perpetual contraction and expansion of the tuberculous lung, however, prevents the healing process and also causes the lung to act as a pump for the distribution of bacilli and toxins through the vascular systems to other areas. The chief result of the collapse of the lung by the insertion of air

into the pleural cavity is to bring the lung into a state of comparative rest, with consequent profound changes in the organ itself and in the thorax generally.

In chronic pulmonary tuberculosis, fibrosis is always present and this fibrosis, while tending to the limitation of the spread of the disease, is a factor which, in association with the normal state of pulmonary tension, prevents the collapse of cavities, the walls of which in most cases are exuding pus. Diagram A illustrates a cavity in the left lung showing how, in spite of the tendency of the fibrous walls of the cavity to contract and so produce obliteration or diminution of the contained space, this wall is pulled out by centripetal forces which are the direct result of the physiological vacuum that keeps the visceral and parietal pleura in apposition.

DIAGRAM A.

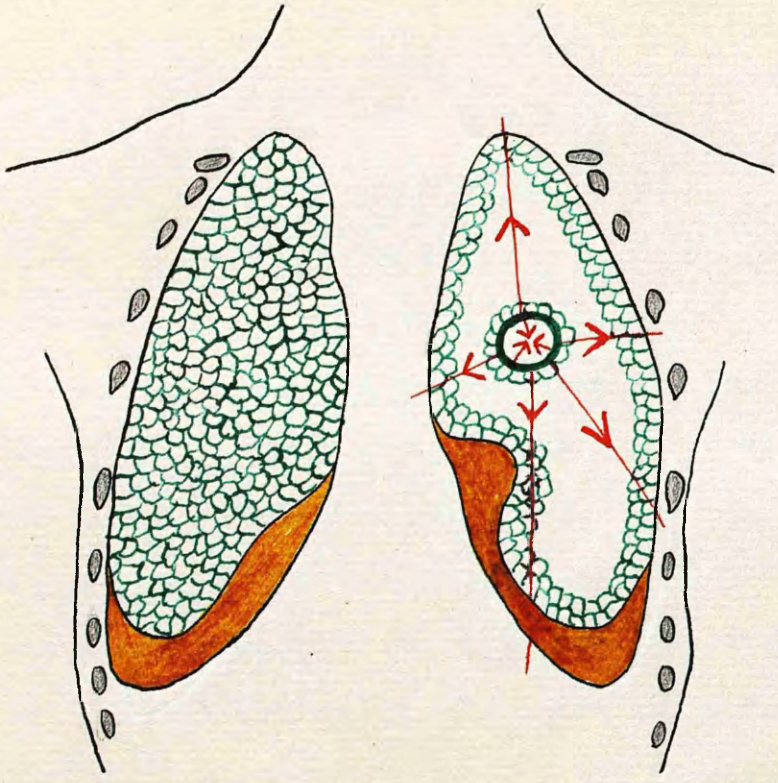
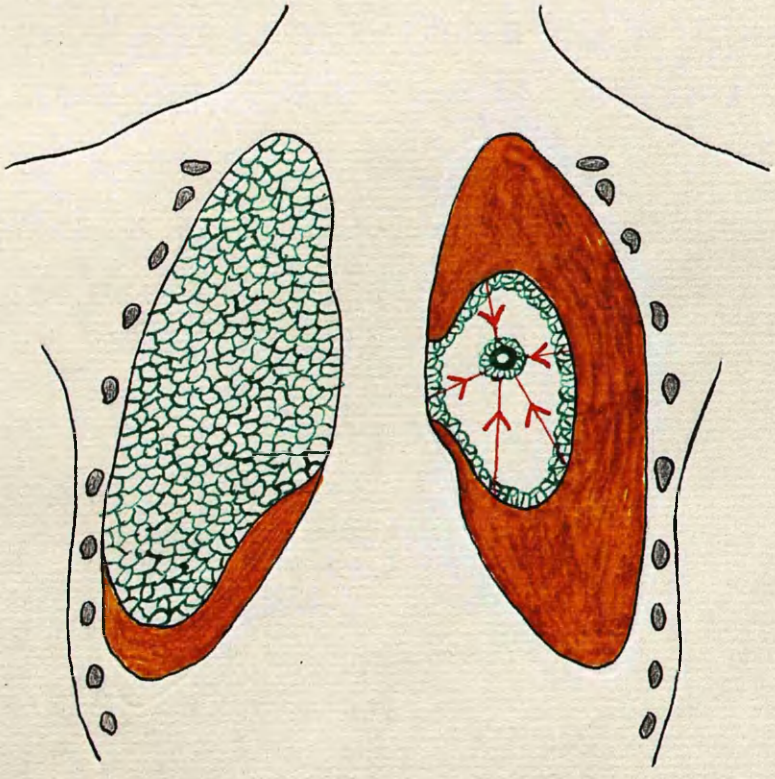


DIAGRAM B



When fibrosis of the lung is marked, fibrous strands radiate from the capsule of the cavity, and have their ultimate distal attachment either in thickened pleura or in the mediastinum. While it is true that neither the mediastinum nor the chest wall to which the pleura is applied is a fixed anchor for these fibrous strands, for it is well known that flattening of the chest wall and mediastinal displacement are results of fibrotic changes, it is also recognised that these deformities are rarely excessive, and even when excessive, the enormous force required to produce such marked changes must also exert a very great pull on the cavity wall. Thus, the tendency of the fibrous strands to contract assists the physiological forces in keeping the cavity or cavities patent.

Given that a length of elastic tissue has to stretch across a certain distance, if a portion of the length of elastic tissue loses its elastic properties, the remainder of the elastic tissue will be stretched to a greater extent than before. On this principle, Parry Morgan (15) has shown that as a result of the destruction of the elastic tissue in a tuberculosis focus, the lung tissue along any axis passing through the consolidated mass must be subjected to greater stretching than the remainder of the lung.

Since the cavity is essentially a consolidated mass, the centre of which has been evacuated, the same conditions must apply in the case of the axis passing through the cavity. Here, therefore, is an additional factor which may keep a cavity patent.

As shown in Diagram B, when the visceral and parietal pleurae are separated by the establishment of an artificial pneumothorax, the lung is no longer applied to the thoracic wall. The elastic and fibrous strands no longer pull the cavity wall towards the pleura; the pleura is now pulled towards the cavity. In the same way, the cavity wall is

no longer pulled towards the mediastinum, the whole lesion, wall and lumen, moves towards the mediastinum. The contracting force of the cavity wall is no longer counteracted by the centripetal forces and the internal surfaces of the cavity may eventually meet and coalesce, so producing healing. By the same process, tuberculous ulceration of the walls of the smaller respiratory passages may be healed by being pressed against other pulmonary structures.

Peters (16), having induced artificial pneumothorax in a rabbit, injected a quantity of lamp black into the animal's circulatory system. The uncollapsed lung was found to contain much more carbon than the collapsed lung, proving that the circulation of the blood in the collapsed lung is much smaller than that in the uncollapsed lung. By means of similar experiments, it has been shown that the flow of lymph through the lungs varies directly with the extent of the respiratory movements, so that the diminution of movement following the collapse of a lung is followed by stasis of the lymphatic fluid in that organ. Further, in post-mortem examinations, it is found that the veins and lymph-

atic channels in a collapsed lung are definitely dilated, and this must assist in the production of venous and lymphatic stasis. The effect of such stasis must be to retard the absorption and distribution of toxic products and to limit the entrance of tubercle bacilli into the general circulation.

A further effect of compression of the lung, as seen in post-mortem examinations, is the increased production of fibrous tissue in that lung. This is probably due to the diminution of movement of the organ, and is an effect frequently noted in other structures when subjected to disuse. The result of this overgrowth of fibrous tissue is to assist in the walling off of cavities, the constriction or obliteration of the vascular systems and the still further contraction of the lung.

When artificial pneumothorax is induced for the treatment of haemoptysis, the cessation of haemorrhage is probably due to the relative immobilisation of the lung and stasis of the blood rather than to compression. On the other hand, however, pulmonary haemorrhage in tuberculosis is frequently due to the rupture of a vessel in the wall of a cavity and

the arrest or diminution of such a haemorrhage is no doubt assisted by the contraction of the fibrous wall of the cavity, so compressing the ruptured vessel.

The functioning lung in artificial pneumothorax has to bear the whole burden of respiration, and this function is to some extent hampered by the partial relaxation of the lung due to the almost invariable displacement of the mediastinum towards the sound side. Respiratory embarrassment, however, is not a frequent symptom, probably because, as many workers have shown, one third or less of the normal total lung volume is sufficient for ordinary respiratory requirements.

Since the flow of blood in the collapsed lung is diminished, additional blood is circulated through the uncollapsed lung, and this is probably the cause of the oedema in the uncollapsed lung which occasionally supervenes, and must be constantly watched for. On the whole, however, the actual mechanical effects of artificial pneumothorax on the contra-lateral lung are so slight that they are not of very considerable importance.

THE SELECTION OF CASES.

The County Sanatorium, Harefield contains 280 beds for adult cases, and an average of 600 adults are admitted each year. Artificial pneumothorax was induced in the first hundred cases, all of whom were adults, over a period of four years, so that approximately four per cent of all adult admissions were eventually so treated. In dealing with patients in all stages of the disease, other workers report 3.5% to 10% as being suitable for artificial pneumothorax treatment. Children as such, were not debarred from artificial pneumothorax treatment but the occasion for so treating a child did not arise, probably because the number of children admitted was relatively small and these were, in the main, merely pretubercular or very early cases.

With the exception of a very few cases of acute pulmonary tuberculosis, all the admissions were cases of the chronic type of the disease in the early, intermediate or advanced stage. Collapse therapy is probably suitable for almost every case of pulmonary tuberculosis at some stage of the disease, and a greater percentage of patients would doubtless

have been offered this treatment but for the fact that few of these cases were under specialised medical observation at the time when operative interference would have been of great value. Further, many cases considered unsuitable from a clinical point of view for pneumothorax treatment, while receiving ordinary sanatorium treatment, would no doubt reach a stage after discharge when operative treatment would be highly beneficial. Clinical opportunity apart, however, there are several factors which prevent the application of this special treatment to otherwise suitable cases. A prospective pneumothorax patient must face the prospect of operative treatment for a period of two years or more, with the added irksomeness of strict hygienic supervision. Thereafter, if the results are very favourable, he will only be able to perform light work for a limited number of hours per day. In recent years, the question of cost is solved, so far as treatment is concerned, by the acceptance of the burden by the local authorities, but should the patient have family responsibilities, he may feel that he should attempt to work, in spite of his impaired and parlous physical condition, in

order to safequard the interests of his dependants. Artificial pneumothorax may thus be refused, to the detriment both of the patient, and of the interests he hoped to protect. The prospect of the discomfort and inconvenience of the frequent subsequent refills occasionally deters a patient from accepting the treatment and sometimes, even though a minor's consent has been obtained, the consent of the relatives is unreasonably withheld.

Most of the artificial pneumothorax cases at Harefield were of patients who had been under observation in the institution for a period of at least three months and who during that time had failed to respond to ordinary sanatorium treatment. The exceptions were, (a) patients who had been under dispensary and domiciliary observation by the Tuberculosis Officer and had been recommended by him for immediate operative treatment, (b) patients with haemoptysis which failed to yield to ordinary measures and whose pulmonary and general condition was deemed favourable for collapse therapy, and (c) patients with pleural effusion aspirated and replaced by air. In this latter type of case, the

pneumothorax was merely a method, when considered advisable, of preserving the collapse of the lung already produced by the effusion.

The best results obtained by artificial pneumothorax treatment are no better than the best obtainable by ordinary sanatorium treatment. It behoves the physician, therefore, to avoid operative measures until he is satisfied that ordinary treatment has failed or is likely to do so. The primary application of ordinary methods is also of value in that the physician has an opportunity of observing and recording the signs and symptoms of the patient over a reasonable period of time. If, at the end of this time, the patient has made satisfactory progress as shown by increase of weight, diminution of sputum, absence or abatement of toxic symptoms, improvement of cough, and evidence of cessation of spread of the disease as shown by clinical signs and X-ray examinations, then the idea of an artificial pneumothorax may for the time being be abandoned. On the other hand, if the symptoms are severe and the disease extensive and spreading in one lung, and if the other lung appears at all

capable of carrying on the respiratory function, then artificial pneumothorax should usually be attempted as a last resort. Between these extremes lie the cases which require the most careful exercise of clinical deliberation and judgement and it is impossible to lay down hard and fast rules in selection because each individual case is a problem in itself. Given, however, a case with definite moderately widespread infiltration in one lung and little or no disease in the other lung, and which shows after several months, neither progression nor retrogression of the disease, then artificial pneumothorax is indicated. Further, the given case would be suitable for treatment if the disease became or remained stationary and the symptoms subsided after a period of absolute rest, but recrudescence of symptoms and signs took place when the patient undertook graduated exercise or work.

Indications and Contraindications. For the purposes of the present discussion, pulmonary tuberculosis may be classified as follows:-

1. Acute Pulmonary Tuberculosis.
   (a) Acute Miliary Tuberculosis.
   (b) Acute Pneumonic Tuberculosis.
   (c) Acute Bronchopneumonic Tuberculosis.

2. Chronic Pulmonary Tuberculosis.

3. Fibroid Tuberculosis.

Treatment by artificial pneumothorax is unsuitable for cases of acute miliary tuberculosis of the lung owing to the widespread nature of the disease. No inductions were performed at Harefield in cases of acute pneumonic or bronchopneumonic tuberculosis chiefly because early death normally supervened in the cases of the very few admissions of this type. Other workers report a few unilateral cases of acute pulmonary tuberculosis treated with good results, but it is generally admitted that with or without treatment, the outlook is bad.

The majority of the cases treated by artificial pneumothorax belong to the chronic pulmonary tuberculosis group. The classical case for treatment may be regarded as one of definitely unilateral disease. Infiltration, caseation and cavitation can be successfully dealt with by collapse of the diseased lung, and the presence of some fibrosis is

all the more favourable in that the ultimate aim is to obtain extensive fibrosis in the collapsed organ.

So far as the contralateral lung is concerned in most cases, however, disease short of the involvement of more than one third of that lung is no contraindication if the disease appears to be non-progressive, and in this connection, it is noteworthy that healing may take place in a less affected lung following collapse of the worse lung. Cavitation in the contralateral lung is a definite contraindication, unless a thick fibrous capsule has formed and fresh infiltration is definitely absent. The presence of toxaemia is not a contraindication, as one of the first and most striking results of a successful collapse is a fall of the temperature to normal and a cessation of tachycardia. Artificial pneumothorax treatment in early chronic pulmonary tuberculosis is usually inadvisable because it is possible for the disease to be arrested with or without other methods and there is, too, some slight risk in the actual operations of induction and refill. Further, it is possible that

an early case, other methods of treatment having failed, may reach a stage when operative interference would be of definite promise. If an artificial pneumothorax has been performed too early and abandoned, the lung will have expanded and adhesions may have formed between the two layers of pleura, rendering, a re-induction impossible.

When the disease is of the chronic lobar type as opposed to the chronic lobular type, the whole or greater part of a lobe becomes a consolidated mass with loss of its normal elastic resiliency. In consequence, collapse is difficult or impossible to obtain, so that this type of case is unsuitable for treatment.

The disease in all the cases of collapse therapy was apical in character. Basal disease was occasionally encountered, but this was found to be associated with apical infection of various degrees of severity.

The border-line between the fibrotic type of chronic pulmonary tuberculosis and fibroid tuberculosis is very indefinite, in fact, the former is generally the forerunner of the latter and the

division between the two diseases is the artificial one of text-book classification rather than a clinical reality. A well-marked case of fibrosis of the lung is usually of slow progress, and cough and dyspnoea are distressing features. Artificial pneumothorax may be attempted if the contralateral lung is in reasonably good condition and if the dyspnoea, which in itself is a contraindication, is not too severe. Extensive adhesions are frequently found and may render even a partial collapse impossible and the lung may be so contracted, with associated traction of the mediastinum to the affected side, that further contraction of the lung may be unobtainable. On the whole, therefore, the treatment is not suitable for fibroid tuberculosis, but benefit may be reasonably expected in a few carefully selected cases.

Clive Riviere (12) cites a group of adult cases analagous to the hilum tuberculosis found in children. These cases are described as having extensive disease on one side, cavitation, if present, being found comparatively low down in the lung. The contralateral lung is devoid of stethoscopic signs, and

therefore in such cases artificial pneumothorax would appear to be of value. On examination by X-rays, however, he found what he terms definite peri-bronchial tuberculosis of a very chronic type. Should this disease be active, the apparently suitable case for collapse therapy becomes definitely unsuitable, although if the hilum tuberculosis is quiescent, he considers that it need be no bar to artificial pneumothorax treatment.

Riviere does not appear to receive support on this matter from other writers. Hilum tuberculosis is a well-recognised entity in children, but this general recognition has not been extended to adult cases. It may be that the peribronchial fibrosis, possibly a forerunner of tuberculosis in some cases, resulting from bronchial affections other than tuberculosis, may be diagnosed on X-ray examination as a so-called hilum tuberculosis of the adult. Apart from this however, X-ray diagnosis of tuberculosis of the lung without other clinical evidence in support, is generally speaking an unwise use of an excellent confirmatory diagnostic aid.

The induction of artificial pneumothorax in cases of haemoptysis has already been referred to in this section, in dealing with the exceptions to the practice of treating patients on ordinary sanatorium lines for a period of several months before considering the question of operative interference. When recurrent haemoptysis or large pulmonary haemorrhages have occurred, and ordinary measures have failed to control the loss of blood, the induction of artificial pneumothorax may be of infinite value if performed in time. Occasionally, a rapidly spreading bronchopneumonia occurs after a haemoptysis and when this happens collapse therapy is rarely helpful in averting the fatal issue. Normally, however, when the total loss of blood within a few days is more than 800 cubic centimetres, either as a result of frequent small haemoptysis or a large haemorrhage from the lung, induction should be performed. After a successful induction with cessation of haemoptysis, the physician must consider the advisability of maintaining the collapse. Should the contralateral lung be competent, the collapse should be maintained, but when it is not reasonably free from disease, the

lung should be allowed to expand about a month after haemoptysis has ceased. The lung from which blood is issuing is frequently difficult to determine, but the knowledge of the presence of cavitation in a lung, discovered previously by clinical and X-ray examination, is sometimes helpful, because a haemoptysis frequently results from the rupture of a vessel in the wall of a cavity. Where no cavitation is known to be present, other indications being absent, the more diseased lung should be collapsed, on the ground that the more disease there is present in a lung the more chance will there be of erosion of a vessel and consequent leakage. A very helpful and obvious sign of the approximate site of the haemoptysis which does not appear to have been mentioned though doubtless observed by other workers, is the bubbling sound caused by the presence of blood in the bronchioles. This sound is easily detected by stethoscopy and is occasionally heard by the observer standing by the bedside. A corresponding bubbling sensation is also usually felt and located by the patient himself. In desperate cases, it is wise to coll-

apse a lung rapidly and if the haemoptysis does not cease, and it is thought that the wrong lung has been collapsed, the other lung may be collapsed about sixteen hours later, as by the end of that time a good deal of the air originally inserted will have been absorbed by the pleura. Alternatively, the air originally introduced may be aspirated as soon as it is realised that the fifty per cent chance has failed, and a collapse may then be produced in the other lung.

From observation of the Harefield cases, the presence of tuberculosis in situations other than in the lung, with one exception, is not regarded as a contraindication. The exception is the presence of intestinal tuberculosis in any marked degree. This complication is usually associated with poor general condition and power of resistance and artificial pneumothorax is rarely of value. Laryngeal tuberculosis was found to be much relieved and in several cases complete healing of the local condition was observed, following successful collapse of the diseased lung.

Emphysema of the lungs and the associated dys-

pnoea is a contraindication, because collapse therapy has the effect of reducing still further the available respiratory surface, with consequent aggravation of dyspnoea to a distressing and dangerous extent. Artificial pneumothorax is also inadvisable in cases of true asthma and of chronic interstitial nephritis with associated dyspnoea and high blood pressure. Pulmonary tuberculosis is a frequent concomitant of diabetes and previous to the introduction of insulin, collapse therapy was regarded as definitely contraindicated. Now, however, with insulin readily available and with facilities for the control of its administration, artificial pneumothorax is worthy of trial, though the concurrent treatments are necessarily irksome to the patient and arduous for the physician. Valvular disease of the heart unless fully compensated is a contraindication as also is degenerative myocarditis.

Acute infectious diseases are obvious contraindications and operative interference in patients over sixty years of age must be regarded as an unnecessary burden to these usually very chronic patients, who have an excellent chance of reaching their allotted span in spite of their disease.

Patients of a highly neurotic tendency are not suitable cases for this form of treatment.

The applicability of partial collapse in the treatment of pulmonary tuberculosis has been advocated by Parry Morgan (17). He asserts that partial collapse will produce the advantages claimed for complete collapse, with the additional advantage that respiration will not be materially interfered with, so that bilateral pneumothorax may be performed. Anatomically and mechanically, he proves that along any radial axis in the lung containing a tuberculous focus, the elastic tension will be greater than along an unaffected axis. This increased tension is said to have the effect of producing a stretching of the focus along the axis and contraction in a transverse direction, and his conclusion is that the alternate contraction and relaxation of the tuberculous focus will cause a wide distribution of toxins and even of tubercle bacilli. By inserting a quantity of gas smaller than that required for a complete collapse, he maintains that the gas will settle over the area of increased tension, bringing that tension into line with the general tension of the lung and so abolishing the deleterious pumping action.

It is noted that Parry Morgan does not claim that partial collapse is any improvement on complete collapse except that partial collapse makes bilateral pneumothorax possible, and even this fact can scarcely be used as an argument in favour of partial collapse, since it is obvious that no one would contemplate complete collapse bilaterally. With regard to the pumping of the toxins and bacilli from a focus as a result of the increased elastic tension, there is no proof that the increase of tension will be sufficient to overcome the resistance of the tuberculous focus to deformity. The possible rigidity of the tuberculous focus may be very slight, but the increase of tension relative to the general normal elastic tension of the lung must also be very slight. Parry Morgan's statement in this respect, must therefore be regarded as an interesting theory rather than a proven fact.

With regard to the general aspect of deliberate partial or selective collapse, it is a fact that a large number of the cases of therapeutic collapse, as a result of the presence of adhesions, are cases of partial collapse only. In the Harefield series,

though the aim was complete collapse, 40% of the cases had only partial collapse and these were maintained when possible with a fair degree of ultimate benefit. Further, the more complete the collapse of the lung, the greater is the distance between the pleural surfaces and the smaller is the chance of their touching as absorption of air takes place. When partial collapse is the aim, dimpling of the lung in the region of the lesion will probably take place but the pleural surfaces in the healthier areas will no doubt meet at some time or other during the maintenance of the collapse, more especially towards the end of a period between refills. The meeting of the pleural surfaces may result in an adhesion which may gradually enlarge and ultimately involve the whole of the pleural surfaces with consequent termination of the pneumothorax. This gradual spread of adhesions is frequently the spontaneous and favourable termination of a collapse of the complete type of several years duration. Obviously, if the collapse is partial, this process will tend to occur earlier than in a complete collapse, and a premature abandonment of the treatment may be necess-

itated. The conclusion, therefore, is that in artificial pneumothorax, the aim should be complete collapse and if partial collapse only is obtainable, good results may still be obtained.

The use of X-rays has been referred to several times in discussing the selection of cases, but the full consideration of the value of radiology before, during and after artificial pneumothorax will be dealt with in a later section.

The selection of cases for operative treatment at Harefield was carried out on the lines already discussed, and statistics in connection with these cases are worthy of consideration.

Age-grouping (Table 1) demonstrates that 88 per cent of the cases induced were between the ages of 15 and 35 years and that only 3% were over 40 years of age. The youngest and oldest cases were 15 and 54 years of age respectively. The preponderance of cases amongst comparatively young patients is as it should be, because, as Hippocrates observed, fatal tuberculosis is more common between the eighteenth and thirty-fifth year.

T A B L E 1.

AGE.	WOMEN.	MEN.	TOTAL.
15 – 20	19	14	33
21 – 25	23	7	30
26 – 30	8	10	18
31 – 35	3	4	7
36 – 40	5	4	9
41 – 45	–	1	1
46 – 50	–	1	1
51 – 55	1	–	1

In Table 2, it is noted that in 76 per cent of the cases eventually induced, the onset of the disease was insidious, the early symptoms being malaise, progressive loss of weight and slight rise of temperature in the evening. 13 per cent of the cases commenced with marked haemoptysis and in 11 per cent the development of pleurisy with effusion was the first indication of the presence of the disease. The average estimated duration of disease before induction was 2.25 years.

TABLE 2.

	WOMEN.	MEN.	TOTAL.
INSIDIOUS ONSET.	46	30	76
ONSET WITH MARKED HAEMOPTYSIS.	7	6	13
ONSET WITH PLEURISY WITH EFFUSION.	6	5	11

Table 3 shows the classification of the cases according to the primary reason for induction. 53 per cent showed unsatisfactory or complete lack of response to ordinary treatment, 14 per cent had severe haemoptysis and the remaining 33 per cent were treated by artificial pneumothorax as a last resort. It is worthy of mention that only 3 of the 18 cases with onset of severe haemoptysis were later collapsed because of severe haemoptysis. 8 of the insidious cases and 3 of the cases which commenced with pleurisy with effusion were so treated.

TABLE 3.

REASON FOR INDUCTION.	WOMEN.	MEN.	TOTAL.
RESPONSE TO ORDINARY TREATMENT SLIGHT OR NIL.	33	20	53
SEVERE HAEMOPTYSIS.	7	7	14
PROGNOSIS BAD WITHOUT A. P.	19	14	33

In Table 4, the cases are divided according to the side treated by artificial pneumothorax and further sub-divided according to the presence or absence of disease in the other lung before induction. 41 per cent of the cases were induced on the right side and 62 per cent of all cases had unilateral disease before induction. The preponderance of left-sided inductions illustrates the widely-observed phenomenon that marked widespread disease is commoner in the left lung than in the right.

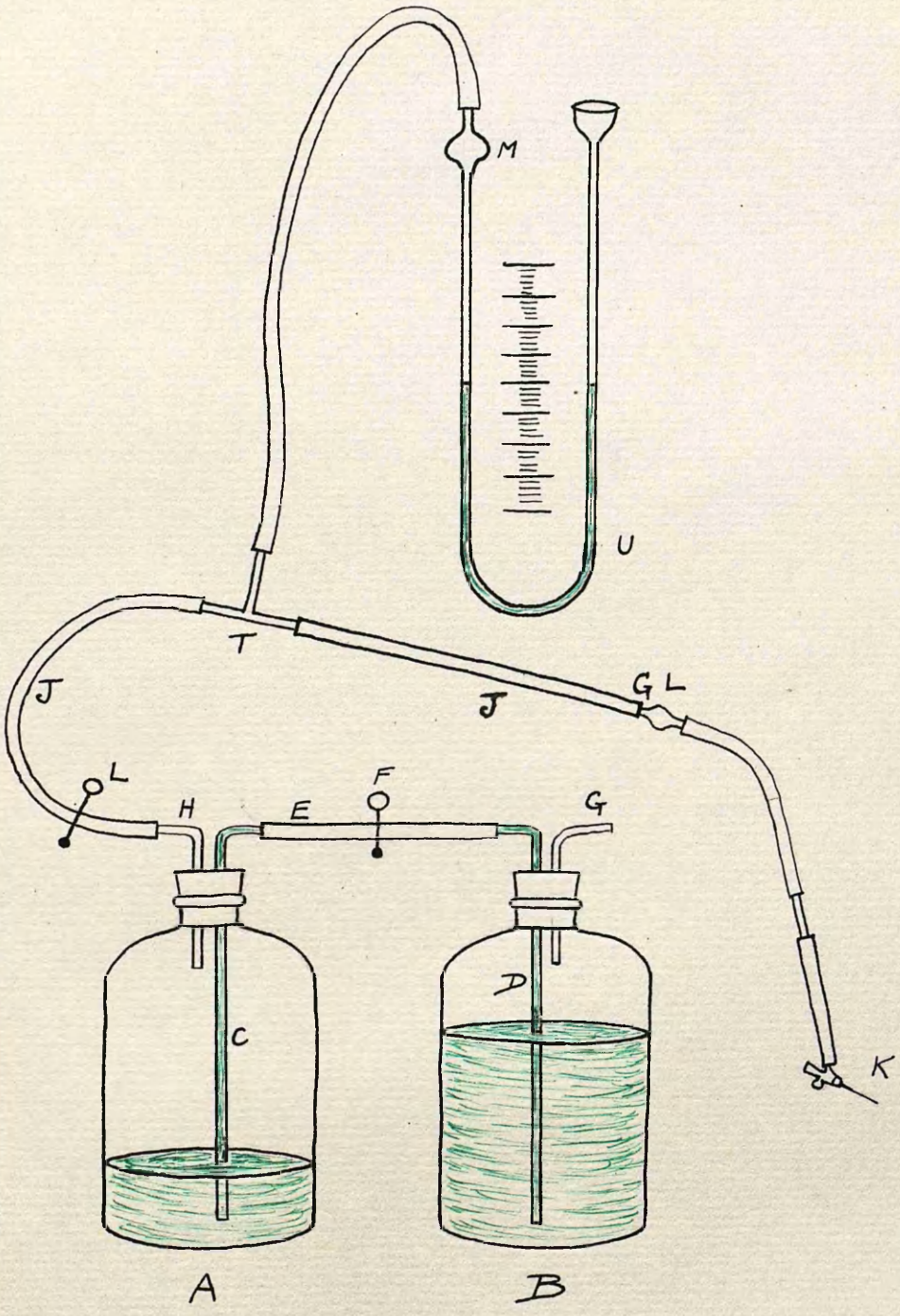
TABLE 4.

	WITH BILATERAL DISEASE.	WITH UNILATERAL DISEASE.	TOTAL.
RIGHT-SIDED A. P. CASES.	16	25	41
LEFT-SIDED A. P. CASES.	22	37	59
TOTAL.	38	62	100

THE TECHNIQUE OF ARTIFICIAL PNEUMOTHORAX.

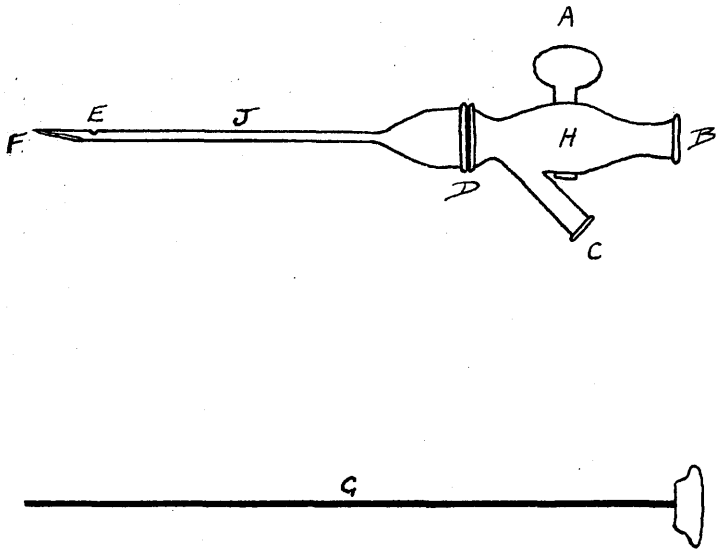
The apparatus used at Harefield is that devised by Lillingston and Pearson (Diagram C). It consists of two glass bottles closed by means of rubber stoppers. One bottle A, is graduated in terms of cubic centimetres and acts as an air reservoir, and the other bottle B, acts as a fluid reservoir, the fluid being 1-200 carbolic acid solution coloured by the addition of a small amount of methylene blue. Long glass tubes C and D, pierce the stoppers and reach to within one centimetre of the bottom of the bottles, and these glass tubes are connected by means of a length of rubber tubing E. The combined glass and rubber tubing when filled with fluid forms a syphon by means of which the fluid can be made to flow from the water reservoir into the air reservoir, and this flow is controllable by varying the relative heights of the bottles and also by a clip F, attached to the rubber tubing E. As fluid flows into bottle A, air is displaced through a short glass tube H to which is connected a rubber tube J, leading to the pneumothorax needle K. The amount of air which passes through the needle is the amount of air displaced

DIAGRAM C.



ARTIFICIAL PNEUMOTHORAX APPARATUS.

DIAGRAM D.



THE SAUGMAN NEEDLE.

from the air reservoir and this is measured by the amount of displacing fluid which has flowed into bottle A, as registered by the graduations thereon. The tube J has four interruptions. First, a clip L, is attached to the tube near the air reservoir by means of which the flow of air along the tube can be diminished or stopped. Secondly a glass T-piece T, is inserted in the tube by means of which a branch rubber tube is led to a U- tube U, half-filled with coloured water and graduated to form a water manometer measuring pressure in centimetres of water. At the top of the closed limb of the manometer, there is a bulbous enlargement M, which assists in preventing the aspiration of the fluid from the manometer in the event of a high negative pressure developing suddenly in the rubber tubing system. Thirdly, a glass bulb GL, packed with sterilised cotton wool, is interposed in tube J, in order to filter the air which passes to the needle. Fourthly, a short straight piece of glass tubing is inserted in tube J. This acts as a window through which the operator can observe any blood or serous fluid which may have passed

through the needle under pressure into tube J.

The needle used is the Saugman needle (Diagram D) of 1.2 millimetre bore. This needle consists of three separate portions, namely, the needle proper J, a three-way connection H, and a stilette G. The needle J has an oblique point F, and near the point there is a small hole E. The joint between needle J and connection H is made air-tight by means of a small leather washer D. The lumen of the connection between B and D can be closed by means of a tap A. When used for an induction or a refill operation, a rubber tube leading to the air reservoir is connected to C and the tap A is closed. Should the needle become blocked it may be cleared by inserting the end of the stilette into the inlet B, opening tap A and passing the stilette through the needle to point F. The opening E is useful in that the lumen of the needle is still connected to the pleural cavity, in the event of the point of the needle becoming blocked.

Most workers appear to use the Riviere trocar and cannula for inductions and the Saugman needle

for refills. This was the procedure at Harefield for the earlier cases, but for the later cases, the Saugman needle was used both for induction and refills. The Riviere trocar is, of necessity, blunter than the Saugman needle, and in dealing with a tough skin and a thin chest wall there is a danger, the skin having been pierced after a relatively great application of pressure, of the trocar travelling with considerable momentum through both layers of pleura and causing damage to the lung tissue. It is true that the instrument is fitted with a guard to prevent this accident, but it is difficult to estimate the thickness of a chest wall sufficiently accurately to set the guard safely. Further, the guard being movable may itself be moved by an accidental sudden entry of the trocar and cannula. The Saugman needle, on the other hand, being very sharp is thrust through the skin and chest wall under perfect control, the sensation of the actual puncture of parietal pleura is felt by the operator and the needle is safely thrust into the pleural cavity.
It may be argued that the Saugman needle being sharper than the Riviere cannula will more readily

injure the lung and visceral pleura when it comes in contact with these tissues, as it must occasionally do. In the cadaver, if one slowly and progressively pushes a Saugman needle against a serous membrane such as pleura, it is found that very definite resistance to the entry of the needle is felt, before actual puncture of the membrane takes place. Similarly, when one is practised in acupuncture, the resistance of the visceral pleura can be felt when this membrane is touched during an induction, and the operator is warned to penetrate no further. Slight injury to the visceral pleura short of actual puncture may be done, but this is trivial in comparison to the relatively great injury caused to the parietal pleura by puncture at induction and at each subsequent refill, with apparently no permanent damage.

With the point of the needle K in the pleural cavity and clip L closed (Diagram C), the pleural cavity is disconnected from bottle A, but is connected with manometer U. As already shown, the pressure in the pleural cavity varies with each respiration and this variation is shown and measured in centimetres of water by the oscillation of the

column of water in manometer U. If the level of the water in bottle B is made higher than that in bottle A, either by the presence of excess of water in bottle B or by raising bottle B by means of wooden blocks, and if clips F and L are open, water will flow from bottle B into bottle A and air will flow from bottle A to needle K. The manometer U will now register the variations of the pressure in the pleural cavity as modified by the pressure of the air in bottle A. At any time, by clipping tube J at L, the true pleural pressure can be read on the manometer scale so long as the needle K is not blocked.

The decision having previously been made that artificial pneumothorax should be induced, the patient, several hours after a light breakfast, receives a quarter of a grain of omnopon hypodermically, the induction being commenced about 40 minutes later. The administration of omnopon has a lulling effect on the naturally increased excitability of the patient and this drug is better than morphine in that it is less likely to cause vomiting. At Harefield, the actual operation takes place in the

patient's own cubicle which is cleared of unnecessary furniture, the patient being on his bed and the whole being suitably screened. By these means the disturbance of the patient is minimised and he is more likely to face that which he at first regards as an ordeal, with more equanimity.

The apparatus sits on a table drawn close to the bed, sterile bowls containing the sterilised instruments being placed on sterile towels. The needles are stored in absolute alcohol and when required for use are dried by being passed through the flame of a spirit lamp. The operator's hands are prepared as for any aseptic surgical operation.

The patient lies on his sound side facing away from the apparatus because it is found that if he is able to observe the manometer, on discovering that the oscillations correspond to his own respirations, his respiratory rhythm ceases to be natural. A well-stuffed pillow is placed below his chest so that his vertebral column is flexed laterally and the intercostal spaces widened on the side chosen for operation. The site

of the proposed puncture and a wide surrounding area is thoroughly cleansed with soap and water, dried with a sterile towel, and heavily painted with tincture of iodine by the nurse in attendance. The operator then surrounds the area with sterile towels.

The question of the selection of the site for puncture has been discussed by many writers and percussion, auscultation, radioscopy and radiography are advocated as important aids in deciding whether pleural thickening or adhesions are present. These methods are doubtless of considerable value but they may occasionally be very deceptive, and the only sure way of being certain that free pleural space is available is by actual trial. A careful puncture does no harm if it fails and other sites may then be tried, though the experience at Harefield is that each subsequent trial has a smaller chance of success, doubtless due to the fact that adhesions when present are usually numerous and fairly widespread. The usual site for puncture is in the fourth, fifth, or sixth interspace in the mid-axillary line. In this region, the chest wall

is relatively thin and in the underlying portion of the pleural sac, the lung makes a relatively large respiratory excursion, thus reducing the chances of adhesions. If two or three punctures fail in this area, a further attempt is made on the following day through the eighth or ninth interspaces about four inches from the vertebral spines. A good site for puncture is through the "Triangle of Auscultation" situated near the inferior angle of the scapula and bounded by the vertebral border of the scapula, Trapezius and Latissimus dorsi. If the patient bends forward at the same time folding his arms, the sixth and seventh ribs become subcutaneous at this point, offering an easy route for the needle. As a last resort the second interspace in the mid-clavicular line may be selected for puncture.

The chosen site is now anaesthetized by means of a proprietary preparation known as Eudrenine. Two cubic centimetres are normally used for each puncture, one cubic centimetre of the solution containing $\frac{1}{6}$th grain of benzamine hydrochloride and $\frac{1}{2000}$ th grain of adrenalin chloride in physiological

sodium chloride solution and preserved with chloretone. Benzamine hydrochloride is very rapid in producing local anaesthesia and even in 1 grain doses appears to be non-toxic. Its action is reinforced by the presence of the adrenalin chloride which produces a practically bloodless field and controls capillary bleeding.

Anaesthetization is commenced by raising a bleb by injecting a small quantity of the local anaesthetic intradermally. The needle is now withdrawn and with the hypodermic syringe vertical to the skin surface and with the needle point in the centre of the bleb, the needle is slowly advanced through the intercostal tissues, the anaesthetic being steadily injected the while. When the needle is felt to have reached the pleura, the remaining $\frac{1}{2}$-1 cubic centimetre of the solution is ejected from the syringe. The procedure is quite painless except for the initial intradermic prick.

A few minutes later, the Saugman needle is made to follow the track of the hypodermic needle. The resistance of parietal pleura is felt; the needle is thrust slowly through the membrane, the puncture

being accompanied by a snap which is always felt and occasionally heard by the operator. In favourable cases, the manometer at once shows negative oscillations through a range of at least five centimetres of water and the reading is duly recorded. With the fluid in the bottles level, the clips are opened and air is sucked into the pleural cavity. After 300 - 400 cubic centimetres of air have been allowed to enter, the clips are closed, the manometric readings, now less negative, are taken and the needle is withdrawn. The needle track is now broken up by deep massage in order to reduce the chance of escape of air and the needle wound is sealed by the application of a small particle of cotton wool soaked in collodium flexile.

Every puncture is not so successful as that described because the pleural cavity may not be entered owing to the presence of an adhesion. A puncture must then be made elsewhere. The needle may be blocked with blood or a small piece of fat or other tissue, in which case the stilette is used to clear the needle, in order to obtain free manometric swings. If good oscillations are obtained and only about 100 cubic centimetres of air are

sucked in, a further 200-300 cubic centimetres can be safely inserted by raising the level of water in bottle B about 5 centimetres above that of bottle A. Should the manometric pressure become rapidly positive under these circumstances, the needle is probably in a free pleural pocket and a partial collapse only is the best that can be hoped for. When severe haemoptysis is the reason for induction, 800 - 1000 cubic centimetres may be inserted, because rapid extensive collapse of the lung is essential if benefit is to be obtained. When the collapse is not urgent, however, the amount of air inserted at first should not exceed 400 cubic centimetres. Now, the pleura has considerable power for absorbing air, especially in the early stages of pneumothorax, and refills of air are necessary both to preserve and increase the collapse obtained at induction. During the twenty-four hours following induction, the condition of the contralateral lung, the position of mediastinum and general signs and symptoms are closely observed, and if these observations are satisfactory, the first refill of

about 400 cubic centimetres of air is given. The technique is the same as for induction except that there is usually no difficulty in obtaining satisfactory manometric oscillations. All being well, the second refill is given on the second day after the first refill, the third refill is given on the third day after the second refill and so on until the eighth refill is given on the eighth day after the seventh refill, the artificial pneumothorax being now a month old. The refills are then given weekly for several weeks and thereafter the interval between refills is gradually extended until at the end of six months the interval is from two to three weeks. As the interval increases, the amount of air inserted is increased up to 600-800 cubic centimetres, depending upon the size of the patient and the condition of the pleural cavity. It is rarely advisable to insert more than 900 cubic centimetres or to have an interval longer than six weeks. At the end of a year in an average patient with a good collapse, the usual refill is one of 800 cubic centimetres at an interval of

four or five weeks.

For three days after induction, the patient is kept at absolute rest. Thereafter, he remains in bed but the regimen is not so strict and at the end of three weeks he is allowed out of bed for gradually increasing periods so long as no untoward symptoms or signs develope. Eventually, the patient is given walking exercise and light work between refills, though a few hours rest in bed is prescribed after each refill.

The spacing and size of the refills must be governed by the condition of the lungs and pleura in each individual case. A study of the mean intrapleural pressures before, during and after each refill is of the greatest importance. Excessively high final mean pressures are to be deprecated, because it is noted that the incidence of fluid development is greater when high pressures are produced than otherwise, probably because some tearing of adhesions followed by pleurisy with effusion may be caused by a high intrapleural pressure. Further, if adhesions are present and a high pressure is injudiciously used with the idea of stretch-

ing or breaking the adhesions, the latter may be so stout that tearing of the lung will occur with a resultant spontaneous pneumothorax and a possible pyogenic infection of the pleural cavity.

A general rule followed at Harefield is that the amount of air inserted should be the smallest compatible with obtaining the collapse desired so long as the respiratory movements of the collapsed lung are reduced to a minimum. By so doing, gross mediastinal displacement and the other harmful effects of too high pressure are generally avoided.

Complete or partial collapse having been obtained, the question arises as to when the pneumothorax should be discontinued. In several of the Harefield cases, the pneumothorax was abandoned after the first or second refill on the ground that adhesions were so extensive that the collapse obtained was so slight that no benefit could be expected. It was considered advisable to discontinue a few cases because of the occurrence of faintness, dyspnoeic distress or pain subsequent to refill. In several cases, the treatment was abandoned when it became apparent that the subject was approaching

a moribund state in spite of treatment. Two cases developed tuberculous empyema, thus bringing the treatment to a close, and one case died of tuberculous meningitis, twenty-one months after induction and after twenty-six refills. In the large majority of cases, the problem of discontinuance was solved by a gradual spontaneous development of adhesions and a concurrent unpreventable expansion of the lung. In the cases of several patients, after a period of treatment of from two to four years, who had no symptoms and who were considered fit for work, the lung was allowed to expand simply by discontinuing the refills. Generally, in dealing with the chronic type of case, the aim was a minimum of two years treatment followed by abandonment of the pneumothorax when quiescence had been achieved.

Several of the Harefield cases were not induced by the method described above, the artificial pneumothorax being a sequel to air replacement of a pleural effusion. The refills and subsequent treatment in such of these cases as were considered to be suitable for continued pneumothorax were similar in all respects to the treatment already de-

scribed. The method of fluid replacement by air about to be described was also the method of refill in artificial pneumothorax cases complicated by the development of fluid, when it was deemed advisable to evacuate this fluid.

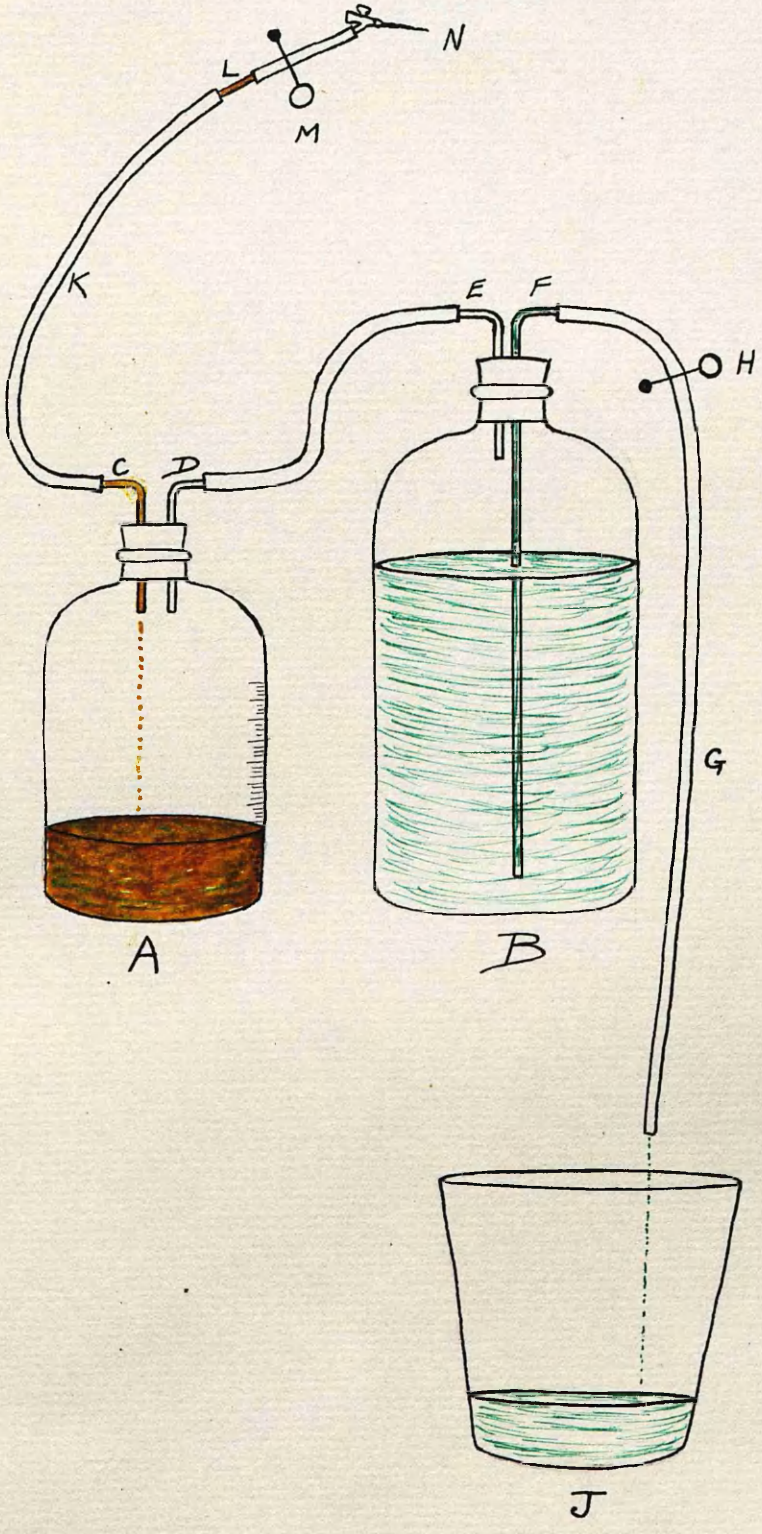
In fluid replacement, the patient assumes a sitting position in bed. After the usual preparations, the artificial pneumothorax needle connected with the apparatus (Diagram C), is inserted through the chest wall, in the axillary region, into the pleural cavity. If this needle reaches air in the pleural cavity, marked negative oscillations will be registered and the negative pressure may be so great as to aspirate the fluid from the manometer if the operator fails to prevent this accident by quickly pinching the rubber tube leading to the manometer. If the needle is in fluid, however, there will be no oscillation and the pressure may be negative or positive. If very markedly positive, the fluid will rush up the needle and be observed by the operator in the glass window near the needle, in which case the clips should be quickly closed.

Using a local anaesthetic as before, a large

bore Saugman needle is inserted through the eighth or ninth intercostal spaces in the mid-axillary line, the inclination of the needle being upwards. The arrival of the point of the needle in the fluid in the pleural cavity is indicated by the escape of fluid from the needle, sometimes under considerable pressure. The flow of fluid is stopped temporarily by fitting a piece of rubber tubing to the needle, the tubing being closed by means of a clip. The rubber tubing is connected to the aspirating apparatus (Diagram E), which is placed on the table near the bed.

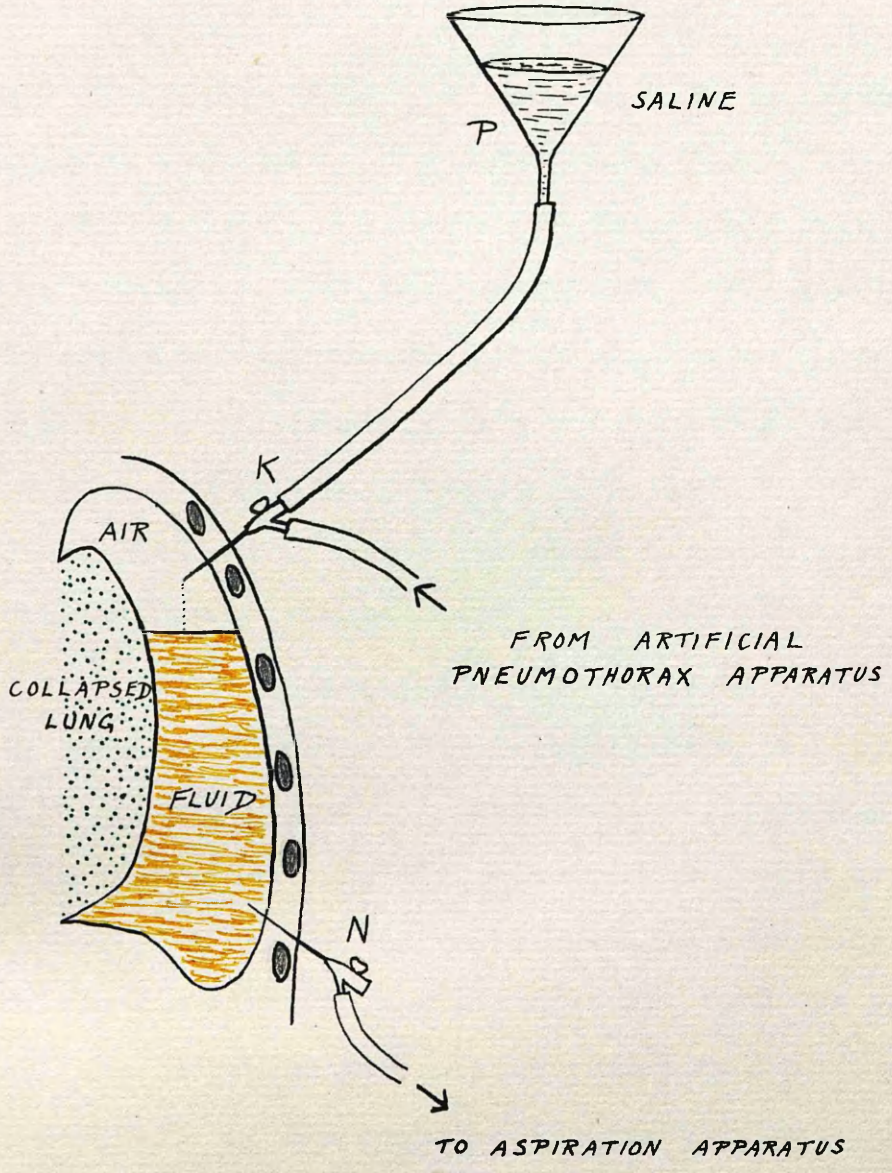
The apparatus consists of an empty graduated bottle A of about two litres capacity and a bottle B filled with about six litres of water. The bottles are stoppered and fitted with glass tubes C,D,E and F as indicated in the diagram. A long rubber tube G, fitted with a clip H, leads from glass tube F to a large open vessel J placed on the floor. Glass tubes D and E are connected by means of another length of rubber tubing. A rubber tube K leads to the Saugman needle N, a short piece of glass tubing L being inserted as a window. The

DIAGRAM E.



ASPIRATION APPARATUS.

DIAGRAM F.



METHOD OF REPLACING FLUID WITH AIR AND WASHING OUT PLEURAL CAVITY WITH SALINE.

clip M is already closed to stop the flow of serous fluid from the chest. The tubes F and G are filled with water and clip H closed. It is readily seen that with the point of the needle N in the pleural fluid and with clips M and H released, water flows from bottle B by siphonage into receptacle J, air flows from bottle A into bottle B to replace the siphoned water and the air in bottle A is replaced by fluid passing through needle N.

With both needles in the pleural cavity, the fluid is thus aspirated, air being allowed to flow into the pleural cavity, if free manometric swings have been obtained. If the pneumothorax needle is in fluid, however, no manometric oscillations will be obtained and sufficient fluid should first be aspirated to allow the level of the fluid to fall below the point of that needle. At frequent intervals during the operation, both sets of apparatus are clipped off in order to obtain a manometric reading. If the manometric pressure is markedly negative, the rate of inflow of air relative to the rate of outflow of fluid should be increased and vice versa if the pressure is marked positive. The final mean

pressure should be left at about zero.

It is occasionally advisable in cases of pyogenic infection of pleural effusion to wash out the pleural cavity with normal saline. This may be done by connecting a long piece of rubber tubing to inlet B of the pneumothorax needle (Diagram D), opening tap A, clipping off the pneumothorax and aspirating apparatus and pouring warm sterile normal saline into the funnel P and so through the needle K into the pleural cavity. The rate of flow is controlled by means of tap A. The pus and saline washings can then be aspirated and air replacements made under manometric control. The method is illustrated by Diagram F.

Dangers, Accidents and Incidents.

PLEURAL SHOCK. Many writers report the occurrence of pleural shock in a very small percentage of their cases of artificial pneumothorax or similar operations involving the puncture of the pleura. The condition appears to arise at the moment of the pleural puncture by the hypodermic or the pneumothorax needle, though isolated instances are recorded where the symptoms did not arise until an app-

reciable amount of air had been inserted into the pleural cavity. The signs and symptoms appear to include lividity of the complexion, faintness or complete collapse, imperceptible pulse and serious respiratory difficulty. Cases of sudden death without any monitory symptoms are mentioned in the literature on the subject. The cause of pleural shock has not been definitely ascertained and the accident has created some considerable controversy. Afferent reflex impulses to the medulla via the sympathetic system resulting from injury to the pleura has been suggested as a cause, these reflexes being cardio-inhibitory, or vasomotor with consequent rapid decline of blood pressure. Only one case of pleural shock occurred amongst the Harefield cases. A refill was about to be given on the right side of a female patient, age 30, who was suffering from advanced pulmonary disease and who was of very nervous disposition. Eudrenine was being injected and as soon as parietal pleura was reached, the patient suddenly became rigid, the pupils were dilated, the radial pulse could not be felt and respiration ceased. An intramuscular injection of pituitary extract was given

at once and was immediately followed by a gasp from the patient. Revival was at first slow, but recovery was complete in two hours. Approximately 4600 pleural punctures were made at Harefield in dealing with the first hundred cases, and the case described is the only one of pleural shock observed. Occasional complaints of faintness during refills were made by other patients, but these symptoms created no anxiety. If the reflex, as has been suggested, is of the vasomotor type, the presence of adrenalin in the local anaesthetic used, may be of value in minimising pleural shock in that adrenalin as a vaso-constrictor would counteract the fall of blood pressure caused by the reflex. Further, the habit of placing a quantity of local anaesthetic on the pleura before puncture may be of value in avoiding pleural shock.

CEREBRAL EMBOLISM. No cases of cerebral embolism occurred as a result of artificial pneumothorax operations at Harefield. Several workers, however, have recorded the occurrence of this accident. Cerebral embolism is probably due to the lodgement of an air bubble in an end-branch of one of the cerebral arteries, the air being carried via one

of the pulmonary veins into the general circulation, following accidental puncture of the lung tissue by the artificial pneumothorax needle. It is probable that air embolism would be entirely avoided by the observance of the following precautions. The pneumothorax needle should never be inserted before the air reservoir has been shut off by means of a clip. No air should be allowed to enter until reasonably free oscillations of the manometer are observed. The patient should be instructed to refrain so far as possible from coughing or breathing deeply while the operation is being carried on, or where cough is frequent and troublesome, the patient should be trained to warn the operator by means of a pre-arranged signal that he is about to cough, and on receipt of this signal the operator should temporarily stop the flow of air, leaving the needle in situ. However, so far as cerebral embolism in artificial pneumothorax is concerned, the position is well summed up by Rist (18) when he says,..... "we can repeat with Forlanini that cerebral embolism belongs to the historical period of pneumothorax therapy".

PUNCTURE OF THE LUNG. Adhesions between the parietal and visceral pleura are frequently accompanied by thickened pleura, and on inserting the pneumothorax needle, the operator on reaching parietal pleura may mistake the resistance of adherent lung for that of thickened parietal pleura. This being so, he pushes the needle on and instead of entering the pleural cavity, the needle punctures the lung. Such an occurrence is indicated by a slight negative pressure without oscillation of the manometer, followed by progressive increase of this negative pressure at each inspiration. The needle is finally blocked by blood clot, the manometer indicating a high stationary negative pressure. Several such accidents occurred at Harefield and in these cases, the needle was rapidly removed and apart from subsequent very slight haemoptysis, no harmful results were observed. The occurrence of this type of accident under the circumstances indicated is apparently unavoidable.

SURGICAL EMPHYSEMA. This accident occurs in the two forms, namely, deep and superficial. Deep

emphysema is the result of allowing air to escape before the needle has pierced the parietal pleura or after it has pierced both layers of pleura. The air travels external to the pleura sac, either internal to the chest wall or through the mediastinum and reaches the neck region causing pain, dysphagia and in a few cases, death from asphyxia. No cases of deep emphysema occurred at Harefield.

Superficial emphysema was, however, a frequent occurrence. After most refills, the final mean intrapleural pressure was either negative or very slightly positive. The final pressure was markedly positive in only a few "veteran" cases. It has been experimentally demonstrated that after a refill, the intrapleural pressure is definitely raised as soon as the patient sits up. An increase of pressure is also caused by the expansion of the inserted air as it is warmed by the enclosing tissues and a further increase is produced by the diffusion of carbon dioxide from these tissues into the pleural cavity. This diffusion is counterbalanced, to some extent however, by the absorption of oxygen by the pleura.

These factors, therefore, create a high intrapleural pressure several hours after refill, relative to the intrapleural pressure immediately after a refill. In addition, if a patient suffers from frequent and violent attacks of coughing, the intrapleural pressure becomes markedly high during the periods of obstructed expiration, and some of the pneumothorax air may be forced along the needle track into the subcutaneous tissues and musculature of the thoracic wall. This condition can to some extent be prevented by deep massage, as previously mentioned, of the needle track after withdrawal of the needle. Superficial surgical emphysema causes pain and stiffness in the region affected and gives rise to a crackling sensation on palpation. Treatment consists of placing a firm pad over the needle wound, held in position by means of strapping tightly applied. In such cases the patient should remain in the recumbent position for at least twelve hours after the refill.

NEEDLE ACCIDENTS other than puncture of the lung are relatively uncommon. In one case at Harefield,

during injection of the local anaesthetic, the hypodermic needle broke off at the hilt just as the needle point had reached the parietal pleura. The chest wall was thick and the fragment of the needle was completely embedded. It was soon located, however, by cutting down with a scalpel, which is always available at every chest operation, and easily extracted with forceps.

Sharp pain was occasionally experienced by patients, especially of the alar-chested type with narrow intercostal spaces, as a result of injury to the intercostal nerve running in the costal groove just internal to the lower border of the rib. Blood clot blockage of the needle may also follow injury to one of the intercostal vessels in the same situation, and subsequent bleeding along the needle track may be troublesome. These occurrences may usually be prevented by avoiding any tendency to upward inclination when inserting the needle. They are not usually, however, of serious consequence.

Septic infection of the needle track in ordinary induction and refill operations is

inexcusable and is prevented by the observance of strict aseptic precautions and the sealing of the skin puncture with cotton wool charged with collodium flexile.

PAIN is a frequent occurrence after artificial pneumothorax operations. The patient complains first of tightness and later of pain in the side treated. The pain is probably due to some stretching of adhesions and when present is often referred to the shoulder. Relief usually comes after several hours, but when severe, the pain may be treated by the administration of morphine or omnopon.

DYSPNOEA of slight severity is present in many cases, especially after the earlier refills. It is also frequently associated with mediastinal displacement which is dealt with in a later section. On rare occasions, severe dyspnoea has necessitated the aspiration of some of the air inserted.

A noteworthy incident is that which occurred during a refill, when as a result of an uncontrollable fit of coughing, the fluid of the manometer was completely blown out by the resultant sudden development of an extremely high intrapleural pressure.

An equally sudden and uncontrolled refill of air through the now empty manometer tubing was prevented by quickly closing the lumen of the rubber tubing near the needle.

To the inexperienced operator, an increase of cough and sputum after the first few refills may give rise to anxiety as indicating that the patient is definitely worse. These symptoms, however, are frequently noted and are the result of the collapse of the lung and the consequent expression of pathological products from the alveoli. When this temporary excess of material has been removed by coughing, the symptoms usually subside.

RADIOLOGY IN PNEUMOTHORAX TREATMENT.

It is now generally accepted that in the diagnosis and treatment of pulmonary tuberculosis there is something to be learned from X-ray examination in every case. When collapse therapy is under consideration, X-ray evidence is of still greater importance in that it is essential not only to know of the existence of the disease but to have detailed information as to the distribution and character of the disease in each lung and its pleural coverings. It is true that much of this information can be acquired by means of clinical examination, but X-ray results are valuable in that they confirm information gained by other methods and may also demonstrate additional facts undiscovered by these methods. Further, if X-ray findings always agreed with the results of ordinary examination of the chest, they would of of little value to the clinician, and it is this liability to disagree as well as to confirm that constitutes the value of radioscopy and radiography.

In the selection of cases at Harefield, X-ray examination always followed clinical examination.

Radioscopy gives information as to respiratory movements of the diaphragm, lighting up of the pulmonary apices on inspiration and the position and movements of the heart. Radiography, in addition to providing a permanent record of the condition and situation of the thoracic viscera and confirming or correcting results, is useful in locating adhesions, cavities and calcified lesions. Pulmonary consolidation and pleural effusion are clearly shown, and the presence of a hitherto unsuspected small spontaneous pneumothorax is occasionally revealed. It has already been shown that the presence or absence of disease in the better lung is of considerable importance in considering collapse therapy, and a radiograph frequently discovers the existence of deep-seated disease when other signs are absent. As a rule, X-ray examination demonstrates that disease in a lung is more extensive than the physical signs indicate, so that on the whole it appears that so far as the extent of the disease is concerned, radiography gives the more correct idea. The value of X-rays,

however, is considerably less than that of ordinary clinical methods in deciding upon the activity of pulmonary lesions, and for this, one must continue to depend upon symptoms and signs.

Print 1 is of a radiograph of Case 8 before artificial pneumothorax was induced. Fairly widespread disease, except at the apex of the upper lobe, is shown on the left side, the heart being displaced slightly to the left. Physical signs of active disease on the left side were found and there was a moderate degree of systemic disturbance. No physical signs were apparent in the right lung, but here the film shows some mottling and root shadows. The interpretation was that the right lung was under suspicion as being slightly affected, but clearly capable of carrying on the respiratory function. This case was therefore regarded for the purpose of artificial pneumothorax, as unilateral.

Radioscopy is most valuable after induction has been performed in observing the extent of the collapse obtained and in ascertaining after subsequent refills, the rate at which the lung tends to expand. The greater the collapse obtained, the denser is the

shadow of the collapsed lung and an effective collapse is one in which respiratory movements of the organ are absent or nearly so. The chest should be examined radioscopically, at least, soon after a refill and occasionally during that refill interval, so that the next refill may be given before the lung re-expands. Re-expansion can be completely prevented by this means, but it has been found that slight re-expansion as opposed to definite resumption of respiratory function is no disadvantage. In this way it is possible to estimate the most suitable interval, during the first few months of a pneumothorax, so that radioscopy may later be performed on less frequent occasions, chiefly for revising the length of the interval and for the observation of any complication that may arise. The presence of fluid in the pleural cavity is readily observed by X-ray examination, and it is noteworthy that the level of fluid in pneumothorax is horizontal, in contradistinction to the curved upper level of ordinary pleural effusion. Radioscopy in cases of hydropneumothorax demonstrates

delicate ripples on the surface of the fluid, when the patient is instructed to give a sudden jerk. Mediastinal displacement is also observed by radioscopy, and it is possible to remedy this condition, by varying the size and interval of refills, under visual control. Flattening and occasionally inversion of the diaphragm often follows insertion of air into the pleural sac, and this also is demonstrable by means of X-rays. Adhesions between the lung and the parietes are similarly detected, and also adhesions between the diaphragm and the mediastinum or the chest wall. X-ray observations at Harefield were usually made with the patient in the standing position, but occasionally, when a patient was very weak, it was necessary to radiograph him in the recumbent position.

A series of eight films were obtained of the chest of Case 8. This was the case of a female patient, aged 22, with moderately widespread left-sided disease and some systemic disturbance. The sputum was positive and the weight on induction was 9 stones 3 pounds. The reason for induction

was the poor response to ordinary sanatorium treatment. Print 1, showing the condition immediately before induction, has already been described. Print 2, taken after the second refill, shows partial collapse of the lower two-thirds of the left lung, the mediastinum being in the normal position, whereas before induction it inclined slightly to the left. Print 3, taken after the sixth refill, shows further collapse of the lung along the mediastinum, the apex remaining uncollapsed. Print 4, after twelve refills, shows some slight re-expansion of the lower part of the lung, the apex however now showing some collapse. The mediastinum is slightly displaced to the right. 600 cubic centimetres of air were being inserted at each refill, the interval being ten days. Print 5, shows the condition, four months after induction and immediately after the fifteenth refill. The mediastinum is markedly displaced to the right, the collapse of the left lung being good except for an adhesion in the region of the second rib. The diaphragm on the left side shows a concave thoracic surface instead of a convex one. These conditions are obviously the result of too high intrapleural pressure left after refill.

Print 6, a month later, shows the frequent result of excessively high pressure, namely, fluid. Print 7, taken six months after induction, shows the result of aspiration of the fluid and replacement with air. The amount of fluid removed was 1700 cubic centimetres and the final pressure was that of one centimetre of water (+1). Print 8, a year after induction, shows the result after aspiration of 900 cubic centimetres of fluid and leaving a pressure equal to that of five centimetres of water (+5). The adhesion in the region of the second rib is well shown in this film, and on comparison of Prints 1 and 8 it is seen that no radiographic signs of alteration of the amount of disease in the contralateral lung are present. Four aspirations were performed on Case 8, after which the fluid did not re-form. No other complications occurred. Artificial pneumothorax was abandoned two and a half years after induction, the lung having gradually re-expanded. Cough and sputum had disappeared, but extensive physical signs were still present on the left side. The

weight was eight stones four pounds, and the patient was fit for light work. The cough reappeared about six months after the cessation of the pneumothorax, symptoms became troublesome and the signs more extensive. The patient died fifteen months after cessation of artificial pneumothorax. Print 9 (Case 47), five weeks after induction, shows good collapse of the base of the right lung with marked pleural bulgings. The mediastinum is displaced to the left. The patient had no signs or symptoms two years after induction. Before induction, there was moderately advanced disease with definite systemic disturbance. Many tubercle bacilli were found in the sputum and slight haemoptysis was occasionally present. This case has since been lost sight of. Print 10(Posterior aspect - Case 37), shows a good collapse of the left lung against the mediastinum, two months after induction. A strong adhesion is present at the base, and at the junction of this adhesion and the lung, there is a ringed translucent area which is probably a cavity. The frequent occurrence of adhesions in association with superficial

cavities has already been mentioned. Print 11 (Case 23), taken seven months after induction, shows the left pleural cavity half full of fluid, the horizontal upper surface of the fluid being strikingly demonstrated. Print 12 (Case 20), taken five months after induction, shows the collapsed left lung attached to the cupola of the pleura by means of string-like adhesions. Extensive disease is seen in the contralateral lung, but this case was treated for severe pulmonary haemorrhage, the immediate results being good. Print 13 (Case 45), taken eight months after induction, shows good collapse of the left lung except for a strong conical adhesion stretching to the region of the first rib. At least one small cavity appears to be present in the base of this adhesion. The diaphragm on the treated side is seen to be depressed. On radioscopy, it was observed in this case, as well as in many others, that both sides of the diaphragm moved in the same direction on respiration, but that the movement on the treated side was less than that on the untreated side.

PSYCHOLOGICAL CONSIDERATIONS.

In dealing with pulmonary tuberculosis cases, undergoing sanatorium treatment at least, the classical "spes phthisica" is usually absent. Depression is very common indeed and is doubtless due to the fact that the patient is to some extent aware that his pulmonary affection renders his condition a precarious one. This consciousness is heightened as time goes on when he realises that many of his fellow patients are definitely on the down grade. His depression is further increased by the necessarily monotonous sanatorium regime of curtailed activity and by his enforced separation from his home, relatives and friends.

The induction or even the proposed induction of artificial pneumothorax brightens the picture. At last, some definite active steps are being taken to combat his disease. His fellow patients who have been successfully induced are improved, for, the immediate results of pulmonary collapse are usually good, and even those patients on whom induction has been attempted unsuccessfully are no worse than before. Some of the artificial pneumothorax cases

of long duration return to the sanatorium for subsequent refills and several of these are following their normal employment and doing well. Their cheerful outlook is noted by the prospective or early pneumothorax case with good results. The operation and subsequent refills improve the relationship between physician and patient, so that the patient's faith in his medical adviser increases, with a resultant beneficial anxiety to carry out the latter's instructions with regard to the details of treatment.

Thus, apart from physical results, it is clear that artificial pneumothorax treatment improves the mental condition of the patient, and although it is impossible to measure the value of this improvement, it cannot be gainsaid that in the treatment of most diseases, an improvement of the mental attitude is followed by beneficial general results.

RESULTS OF TREATMENT BY ARTIFICIAL PNEUMOTHORAX.

The results, both good and bad, will be considered in the chronological order in which they usually occur, from the time the lung collapse is produced to the time when the patient has returned to his normal environment.

The increase of cough and sputum which may immediately follow collapse has already been referred to. Some slight breathlessness may also be present and the patient generally feels more ill at ease than before induction. When, however, the excess of pathological products, which results from the diminished capacity of the collapsed lung, has been removed by expectoration, the unfavourable pulmonary symptoms subside, and after several refills, in favourable cases, the toxaemic manifestations begin to diminish. The temperature falls to normal, though some erratic fluctuations may be noted before collapse is complete, the pulse rate is lowered and night sweats, if previously present, are reduced in frequency and intensity or completely abolished. Most observers record some reduction in weight in the majority of cases induced, and in

many cases this loss of weight is never regained. The cause of this loss is inexplicable, though it is observed that a marked loss of weight frequently follows the production of a high intrapleural pressure with gross displacement of the mediastinum. Whatever the cause of the loss of weight observed, the fact remains that it is not of unfavourable portent.

In 70 of the Harefield cases, either partial or complete collapse was produced, and Table 5 shows these cases divided according to the weight on cessation of pneumothorax treatment relative to the weight on induction. For purposes of comparison, the 70 cases are divided according to the general condition immediately after abandonment of the pneumothorax relative to the condition on induction.

T A B L E 5.

Change in weight and in general condition of cases with successful collapse.

WEIGHT.		GENERAL CONDITION.	
Increased.	19	Improved.	35
Unchanged.	25	Stationary.	21
Decreased.	26	Worse.	14

It is readily seen that a number of cases which were definitely improved so far as symptoms and signs were concerned had nevertheless lost weight.

When fairly complete collapse of the lung is obtained, sputum usually progressively diminishes in quantity and may disappear. Concurrently, the number of tubercle bacilli found in the sputum diminishes and eventually the patient may have either no sputum or slight sputum free of the specific organism. In successful cases of collapse for haemoptysis, slight "staining" of sputum may be present for some days but when this disappears, no recurrence of pulmonary bleeding normally occurs. Table 6 shows an analysis of sputum results before and on cessation of collapse therapy of the 66 cases of successful collapse at Harefield in which the pneumothorax has now been abandoned.

T A B L E 6.

BEFORE INDUCTION.		ON CESSATION OF COLLAPSE THERAPY.
No Sputum.	Nil	15
T.B.Negative.	4	13
T.B.Positive.	62	33
Dead.	Nil	5

From these figures it is manifest that a marked improvement in sputum condition is an immediate result of collapse therapy.

All cases at Harefield Sanatorium are classified according to the Ministry of Health classification (Appendix A.) Of the 66 successfully collapsed cases of the Harefield series in which collapse therapy has been terminated, 4 belonged on induction to Group A, 1 to Group B1, 30 to Group B2, and 31 to Group B3. The numbers in Group A and B 1 are too small to enable one to draw any reliable conclusion with regard to these groups, but the sputum results of the cases in Group B 2 and B 3 immediately after cessation of artificial pneumothorax are given in Table 7 where they are compared with the sputum results of certain patients at the end of ordinary sanatorium treatment only. The latter cases were those of all adult patients discharged from Harefield Sanatorium during the year 1925 and who were classified in Groups B 2 or B 3 on admission. The first hundred cases of artificial pneumothorax at Harefield were induced between 1923 and 1927. The year 1925 is the middle year of that

period and was in no way an abnormal year, so that the ordinary patients discharged in that year may be considered as suitable controls for the pneumothorax cases, so far as the time at which treatment was given is concerned. Of these ordinary cases, 192 belonged to Group B 2 on admission and 112 to Group B 3. In order that the sputum results may be comparable, they have all been reduced to percentages.

T A B L E 7.

	SPUTUM RESULTS AFTER ORDINARY TREATMENT.	SPUTUM RESULTS ON CESSATION OF COLLAPSE THERAPY.
GROUP B 2.		
No Sputum	8 per cent.	27 per cent.
Sputum T.B.Negative	31 " "	20 " "
Sputum T.B.Positive	57 " "	50 " "
Dead	4 " "	3 " "
GROUP B 3.		
No Sputum	3 per cent.	16 per cent.
Sputum T.B.Negative	16 " "	20 " "
Sputum T.B.Positive	51 " "	51 " "
Dead	30 " "	13 " "

There appear to be two possible fallacies in the above comparison. The first is that the pneumo-

thorax cases are classified on induction which took place, on an average, several months after admission, while the ordinary cases are classified on admission. No pneumothorax case improved during the period between admission and induction, otherwise collapse therapy would not have been instituted. On the other hand many of the pneumothorax cases showed definite deterioration before induction, so that the condition of the pneumothorax cases as a whole was retrogressive. The average case of pulmonary tuberculosis shows at least a slight improvement during sanatorium treatment. Thus the prognosis in the average pneumothorax case before induction, excluding the possible effect of collapse therapy, was worse than that in the average case in the corresponding classification group, in which pneumothorax was not contemplated. Supposing, for the moment, that collapse therapy is neither beneficial nor harmful, one would therefore expect that the ultimate condition of the average pneumothorax case would be worse than that of the case which had been treated ordinarily, and the average sputum result of the former would similarly be worse than that of the latter. Any bias therefore,

in Table 7 which might result from the difference of the times of classification, is against rather than in favour of the sputum results of the artificial pneumothorax cases.

The second possible fallacy is that deaths have been included in computing percentages results, and it might well be argued that some of the deaths included in the group of ordinary cases had possibly occurred within the first few months after admission, and since artificial pneumothorax was induced in the average case several months after admission, some deaths might possibly have occurred in cases in which pneumothorax might otherwise have been performed and that in consequence there is a bias in favour of the pneumothorax sputum results in the above table. It is apparent, however, that even by reducing the number of deaths of ordinary treatment cases by as much as fifty per cent of these deaths to compensate for this hypothetical error and increasing the other figures of the ordinary treatment cases correspondingly, namely by $\frac{2}{98}$ in Group B 2 and by $\frac{15}{85}$ in Group B 3, that the sputum results of the pneumothorax cases remain

better than those of the ordinary treatment cases. Apart from these considerations, however, any error as a result of including deaths must be very small indeed, and even if all deaths were excluded from these statistics, the error would be very great because most of the cases in which death occurred would doubtless have positive sputa for some time before death, and some of these cases would therefore require to be added to the positive sputum group.

These possible fallacies having been considered, one may proceed to draw conclusions from Table 7. In group B 2, 39 per cent of ordinary treatment cases were discharged in a non-infectious state, while of the pneumothorax cases, 47 per cent were non-infectious on cessation of treatment. In group B 3, 19 per cent of the ordinary treatment cases were non-infectious on discharge as compared with 36 per cent of the pneumothorax cases on cessation. So far as the individual case is concerned, this difference appears to be of little importance, but relative to the safeguarding of the public health, it indicates that artificial pneumothorax may be regarded as another weapon in the armoury of preventive medicine.

Displacement of the mediastinum towards the sound side is a frequent result of collapse therapy; in fact it probably occurs at some stage of treatment in every case of artificial pneumothorax. The degree of displacement is usually slight and no discomfort is normally experienced by the patient. On occasion, however, even with comparatively low pressures, considerable displacement is observed and dyspnoea may be very marked. This dyspnoea is probably the result of compression of the contralateral lung with consequent further diminution of the already diminished total respiratory surface. The displaced mediastinum, too, may conceivably press on and diminish the lumen of the bronchus or of a bronchiole of the opposite lung, and so cause respiratory embarrassment. So far as possible, displacement of the mediastinum is avoided and when it does occur an adjustment of the size and of the interval of the refills, consistent with maintaining a suitable collapse of the lung, is made.

In discussing the selection of cases, the presence of pleural adhesions has already been noted as a possible cause of failure in producing collapse.

Adhesions may also follow a serous effusion into the pneumothorax cavity though these are generally more easily dealt with than the stronger adhesions of possibly several years duration. Pleural adhesions constitute one of the chief causes of failure in artificial pneumothorax treatment, in that failure to find free pleural space may result from the close welding of the parietal and visceral surfaces, and if free space is found, the collapse obtained is only partial. When partial collapse is obtained, the collapse may only involve the diseased portions of the lung and the highly favourable condition of collapsed diseased tissues and uncollapsed healthy tissues be obtained. Too frequently, however, the reverse is the case, because the irritation of underlying diseased tissue is usually the primary cause of pleural inflammation with subsequent adhesion formation, the result being that a useless collapse of healthy tissue only is obtained. No attempt to deal with adhesions was made at Harefield, other than by temporarily increasing the intrapleural pressure above that required for satisfactory collapse, in the hope of stretching the weaker type of

adhesions. This was carried out under the most careful X-ray control and in some cases an improved collapse was obtained by so stretching or breaking adhesions. Great care was taken however, to refrain from increasing the intrapleural pressure to such an extent as to risk rupture of the lung by tearing the visceral pleura at the junction of an adhesion. In other words, obviously thick adhesions, as observed by radioscopy, were not attacked, and stretching attempts were soon abandoned in the case of the apparently weaker adhesions which failed to respond readily to moderate pressures. Now, it is obviously the stronger type of adhesion that is the common cause of failure to obtain good collapse, and the figures of the Harefield cases, to be discussed later, demonstrate that the chief cause of some of the unsatisfactory end-results was failure to deal successfully with strong adhesions.

Tudor Edwards (19) describes two methods of division of adhesions, namely, (a) division of bands of adhesions by means of a fine tenotome introduced through an intercostal space and (b) division of

bands by electric cautery under observation with a thoracoscope. The second method appears to be the better in that the cautery divides and to some extent seals the cut surface simultaneously, so that risks of haemorrhage, wounding of lung tissue, and air embolism are diminished. Edwards sums up his article by quoting Jacobaeus who says "Although it has been impossible to obtain so high a percentage of clinically improved cases as in cases of simple uncomplicated pneumothorax without adhesions, the procedure ought to be of permanent value in perhaps a limited number of pneumothorax cases with string-like or membrane-like adhesions". Excellent as this treatment appears to be, it is apparent that the complicated operation of cauterization can only be undertaken by a surgeon, practised and highly skilled in this type of work, and for this reason it is unlikely that this method will ever be widely used in sanatoria and similar institutions. When the result of artificial pneumothorax treatment is poor in consequence of limitation of collapse by adhesions, the alternative, if treatment is to be continued, appears to be the performance of thora-

coplasty in a suitably equipped and suitably staffed hospital for chest diseases.

The following is a summary of the degree of collapse obtained in the first hundred cases of artificial pneumothorax at Harefield Sanatorium:-

Good Collapse	- no adhesions.	25
Good Collapse	- a few adhesions.	17
Useful partial collapse	- adhesions present.	28
Useless partial collapse	- adhesions present.	13
No collapse	- adhesions present.	14
Treatment abandoned early-extraneous causes.		3

Deducting the 3 cases of the last group from the total number of cases, it is seen that only 26 per cent of cases were apparently free from adhesions and that in 28 per cent, there was failure to obtain useful collapse because of adhesions.

The results of other workers, quoted by Burrell (11), with regard to the failure of pneumothorax owing to adhesions are given below. The figures in the second column include those in the first column. The corresponding Harefield figures are added.

	NO COLLAPSE.	NO USEFUL COLLAPSE.
Burrell	13 per cent.	24 per cent.
Riviere	20 " "	37 " "
Saugman	11 " "	22 " "
Zink	24 " "	27 " "
Keller	12.5 " "	37.5 " "
Harefield series	14 " "	28 " "

The discrepancy between these figures is possibly due to a difference in the type of cases dealt with by the various workers, although it is noteworthy that there is only a small difference between Burrell's results and the Harefield results, and even this small difference may be explained by the fact that the Harefield cases on induction were rather more advanced than those observed by Burrell.

Pleural effusion is a very frequent result of artificial pneumothorax treatment, but the necessity for operative interference for the relief of this condition is fortunately much less frequent. There are several causes of pleural effusion, and one of the chief of these is no doubt the irritation re-

sulting from the formation of fresh tubercles on the pleura, with consequent tuberculous pleurisy. It must be admitted that in uncollapsed lungs affected with tuberculosis, pleural tubercles undoubtedly develop with but rare resultant wet pleurisy, but the conditions are altered when a pneumothorax cavity is present. The formation of tubercles on the pleural coverings of an uncollapsed lung tends to be limited by the formation of adhesions between the visceral and parietal pleura. When these are not contiguous as in pneumothorax, no adhesions can form, the pleurisy is therefore not limited and effusion readily occurs. Similarly, the breaking down of adhesions in artificial pneumothorax may create a pleurisy with effusion. Further, the apposition of moist pleural surfaces is lost in pneumothorax, and it requires no great stretch of imagination to assume that, the force of cohesion which prevents the downward drainage of the moisture having been lost, the fluid will slowly gravitate to the lower part of the pleural sac, the pleura will continue to secrete fluid to replace that which has drained, and so a passive hydrothorax will result. High intrapleural

pressures frequently cause the development of fluid, and it is possible that this is due to the compression of the vessels in the hilum of the collapsed lung, producing an unusual degree of stasis in the lung with consequent increased permeation through the bounding visceral pleura. In this connection, it is interesting to note that Halliburton (8) states that stomata exist in many serous membranes by means of which lymphatic vessels communicate directly with cavities formerly supposed to be closed, so that a serous cavity may be regarded as a widening out of the lymph-capillary system. If this is so, a lymph stasis in the lung, as a result of compression of the lymphatic main vessels in artificial pneumothorax, would be followed by an outpouring of fluid through the stomata into the pleural cavity, producing a hydrothorax.

From observation of the Harefield cases, pleural effusions appear to be divisible into four groups, namely (a) small serous effusions with little or no constitutional disturbance and which clear up without treatment, (b) serous effusions commencing with moderate temporary pyrexia and gradually increasing

in quantity so that aspiration is necessary, (c) serous effusions accompanied by serious progressive spread of pulmonary disease terminating in death and (d) serous effusions secondarily infected by pyogenic organisms, the outlook being grave. In groups (b) and (c), the serous effusion becomes purulent as time goes on, the pus however, being of the sterile tuberculous variety.

Serous effusions probably occur in the majority of cases, though only 46 per cent of the successfully collapsed Harefield cases were observed to have developed fluid which persisted for a period of weeks. It was noted, however, during radioscopic examinations, that a small quantity of fluid was occasionally observed in the phrenicocostal sinus, this fluid being undetectable by clinical examination. Daily radioscopy showed that this fluid frequently disappeared in a few days. A reasonable assumption, therefore, is that an undiscovered small quantity of fluid may be present at some time or other in most cases. However, in dealing with the results of artificial pneumothorax, one is mainly concerned with effusions which give rise to definite signs and symptoms and

which may materially affect the ultimate result of the case.

The fluid was examined in every case in which pleural effusion occurred, and tubercle bacilli were found in all the specimens examined with the exception of a small number of clear colourless samples. These exceptional samples, perhaps, were taken from cases of passive hydrothorax occurring as a mechanical result of the pneumothorax rather than as a result of a tuberculous process.

The development of fluid is detected by ordinary clinical examination and by radioscopy. Succussion splash, audible to both physician and patient, is probably the easiest and most reliable diagnostic sign in hydropneumothorax. No treatment is necessary for many cases of pleural effusion, but in those Harefield cases in which distressing symptoms occurred as a result of pressure of the fluid, and in which systemic disturbance was persistent, aspiration was carried out. In two cases, tuberculous pus which recurred after frequently repeated aspiration was finally disposed of by washing out the pleural cavity with normal saline as described in a previous section.

If pleural effusions are neglected for a long time, it is found that the pleural thickening, which always follows effusion to some extent, becomes very marked and adhesions may develop, necessitating the abandonment of collapse therapy. The thickening of the visceral pleura may be so great that the lung is permanently preserved in a state of collapse, but there is insufficient evidence to indicate whether this event is to be regarded as favourable or not. An obvious disadvantage is the chest deformity which follows the slow obliteration of the pleural cavity when the lung is incapable of expanding. Only one case of secondary pyogenic infection of pleural effusion occurred, and this was probably due to spontaneous perforation of the lung. In spite of treatment, the condition of this patient progressively deteriorated and death resulted.

Of the Harefield series of 70 successfully collapsed cases, 32 developed fluid. These are divided as follows:-

Serous Fluid. Treatment unnecessary.....28%

Purulent Fluid. Treatment unneccessary.....22%

Purulent Fluid. Aspiration performed.....47%

Secondary pyogenic infection of fluid.....3%

It is readily seen that treatment by aspiration was unneccessary in half of the cases in which fluid developed.

Few opportunities occurred for post-mortem examination on cases treated by artificial pneumothorax, but in the autopsies which were performed the collapsed lung was found to be a fleshy mass in close apposition to the mediastinum. The shape of this mass was found to vary according to the degree of collapse obtained. In complete collapse, the lung has a shape similar to that of a Rugby football, the longer axis being vertical. The shape in partial collapse depends upon the nature and situation of adhesions, and sometimes as a result of apical and basal adhesions, the lung is only compressed laterally the length from apex to base being unchanged. Air-spaces are few or entirely absent in the compressed lung and a marked widespread overgrowth of fibrous tissue is the most striking anatomical feature. Many cavities are found to have been obliterated and those which have persisted are usually found to have thick fibrous walls, the contents if any are dry and calcification is occasionally present.

The layer of air intervening between a collapsed lung and the parietes renders attempts at clinical examination of that lung practically valueless so far as estimating the amount and activity of disease in that lung is concerned. On expansion, however, at the end of artificial pneumothorax treatment, vesicular or broncho-vesicular breathing is heard on auscultation, the latter type being common in those cases in which disease on induction was severe and widespread. No signs of active disease are found when the collapse has been complete and of long duration, but uncollapsed or partially collapsed portions of a lung may still be found to show signs of activity. In this latter event, the prognosis has not been improved by artificial pneumothorax, for the treatment is practically useless unless the whole of the disease in the treated organ is rendered quiescent. A lung, partly healed and partly the site of active disease, will probably revert to its original state of widespread disease within a few months after re-expansion. The importance, therefore, of obtaining a complete collapse of diseased tissues is apparent, if a disease-free lung in which healing is likely to be permanent is to

be procured.

The effects of artificial pneumothorax on the contralateral lung are of importance, because if disease is present in that lung even though the worse lung has been successfully treated, the future of the patient is still precarious. As previously observed, the circulation in the contralateral lung increases after collapse of the worse lung, and practically the whole of the respiratory function is thrust upon the uncollapsed organ. Whether or not these changes assist or retard the spread of disease in the contralateral lung is difficult to determine, and the question remains a subject of controversy amongst artificial pneumothorax workers. The pathological products that are expressed from the worse lung as it is collapsed, endanger the sounder lung in that some of the infective material may be aspirated by way of the bronchus and may thus give rise to fresh foci of infection. This disadvantage of pulmonary collapse, however, is probably counterbalanced by the fact that at a later stage of treatment, the now collapsed lung is no longer the source of infectious discharges into the bronchi that it

was before collapse therapy was commenced. Pulmonary tuberculosis tends to increase progressively, without and sometimes in spite of treatment, so that apart from the possible effects of collapse of the worse lung, disease if present in the contralateral lung has a natural tendency to spread. In any individual case, the sum of the effect of these varying factors must determine the spread or absence of spread in the contralateral lung, and while one can observe the total effect, it is impossible to compute the actual effect of each contributory factor.

Of the 70 successfully collapsed cases at Harefield, 46 had unilateral disease before induction and in 32 of these the disease remained unilateral throughout treatment, the remaining 14 becoming bilateral. Of the 24 cases with bilateral disease on induction, 15 showed spread of disease in the contralateral lung, 6 showed no change and 3 showed improvement of the contralateral lung. In other words, 30 per cent of cases unilateral on induction became bilateral and 62 per cent of the bilateral cases showed spread of disease after induction. Of the cases bilateral on induction, only 12 per cent

showed improvement of the contralateral lung.

Oedema of the contralateral lung, with consequent cough and dyspnoea, is an infrequent result of displacement of the mediastinum caused by injudicious use of high intrapleural pressures. Moist crepitations are heard at the base of the lung on auscultation when this complication is present. This condition was but rarely found in the Harefield series of cases, and the remedy consisted of reduction of the intrapleural pressure.

In a previous section on the indications for collapse therapy, mention has been made of the excellent effect on laryngeal tuberculosis of the collapse of a diseased lung. Several of the pneumothorax cases suffered from hoarseness or aphonia, pain and troublesome cough before induction. Ulceration and oedema of the laryngeal structures were present in these cases, and in one case the ulceration involved the edges of the vocal cords. In three cases in which these signs and symptoms were present before induction, the local disease healed completely after production of practically complete collapse of a lung. The ulcerated vocal

cords in the case mentioned showed some cicatrization after healing, but in spite of this deformity, vocalisation was only slightly impaired. Apart from the cases mentioned in which complete healing took place, other cases showed improvement of the laryngeal condition after artificial pneumothorax. It should be added that complete silence was enforced in these cases of laryngeal tuberculosis, but the progressive and frequently complete disappearance of laryngeal symptoms in artificial pneumothorax cases is rarely met with when silence treatment alone is undertaken.

The marshalling of figures relating to the results of artificial pneumothorax treatment is a matter of considerable difficulty. It is impossible for any single observer to collect and organise the results of his cases in the form of statistical tables to show definitely what may or may not be expected from collapse therapy, because no individual, even after a life devoted to this treatment, could possibly observe a sufficiently large number of cases to make his statistics dependable. Further,

it is unfortunately impossible to obtain a reliable series of control cases which would form a basis for comparison with the cases treated by artificial pneumothorax. Rist and Hirschberg (20) have attempted to estimate the percentage of cures in a series of cases treated by artificial pneumothorax by means of a comparison between the end-results of these cases with those of cases not so treated but in which the chest condition was nearly similar. All cases had positive sputum, and unilateral disease affecting at least half a lobe was present. By the eighth year of observation, 90 per cent of the controls were dead, the figures for the treated cases being less than half this percentage. 36 per cent of the cases treated by artificial pneumothorax were cured.

While these figures demonstrate the value of artificial pneumothorax, the attempt to establish "control" cases introduces an element of fallacy in that no two cases of pulmonary tuberculosis can be said to be alike or even nearly alike, and at Harefield under the same kind of ordinary sanatorium treatment, no two apparently similar cases have

ever been observed to follow the same subsequent course.

In view of the fact, therefore, that one can neither compare the results of collapse therapy with untreated cases nor gather a large series of cases alone, all that one can hope to do is to record and arrange one's own results, and trust that in years to come all the results of the many workers on this form of treatment may be gathered together to form informative and reliable statistics.

The following table shows the results of artificial pneumothorax relative to the condition on induction in cases classified in Groups B 2 and B 3. These groups and the terms "quiescent" and "arrested" are defined in Appendix A, the only other term requiring definition being "much improved". This latter term is applied to cases in which the general health was good, physical signs not necessarily cleared up though much diminished and tubercle bacilli not necessarily absent from the sputum. The figures include 4 cases still under treatment.

TABLE 8.

Condition of cases in which complete or partial collapse was obtained (Group B 2 and B 3).

	On cessation of collapse therapy.	In September 1928.	In December 1929.
Quiescent or Arrested.	5	4	9
Much improved.	27	13	11
Stationary.	19	13	5
Worse.	9	5	3
Dead.	5	29	36
Lost sight of.	-	1	1

The average period between abandonment of collapse therapy and the final observation in December 1929, is 3½ years. 20 of the 66 cases are very definitely improved and when it is remembered that most of these 66 cases, after a period of sanatorium treatment, were considered to be definitely on the down grade, the fact that 30 per cent of them either show no signs of disease or are in good health at the end of a period of years, demonstrates that in these cases, at least, the results of collapse therapy are remarkably good. Table 8 further shows that the number of definitely improved cases increased slightly

during the fifteen months subsequent to September 1928, and since all the cases had returned to their home environment in 1928 there is apparently no expectation of the number of improved cases becoming smaller in future years. The diminishing numbers in the "stationary" group show that the disease is unlikely to remain stationary, and the large number of patients who have died relative to the number improved demonstrates that the few cases in the "stationary" group in 1929 are more likely to end in death within the next few years than to show improvement. The mortality of 55 per cent is very high and it is of interest to make some comparison with the mortality after ordinary sanatorium treatment. The pulmonary tuberculosis mortality after sanatorium treatment at King Edward Vll Sanatorium, Midhurst has been investigated by Bardswell and Thompson (21). In Groups 2 and 3 at Midhurst, corresponding to Groups B 2 and B 3 of the Ministry of Health classification, there were discharged 848 and 399 patients respectively. 376 patients in Group 2 and 285 in Group 3 had died 3 to 4 years after discharge. Now it happens that there were 35 cases in Group B 2 and

30 cases in Group B 3 in the Harefield series, and the Midhurst series may be made approximately comparable to the Harefield series in respect of the distribution of the total number of patients between the two groups, by halving the number of patients and of deaths in Group 2, making 424 patients and 188 deaths. By adding the figures of Group 3 to these "corrected" figures, we have a total of 823 patients and 473 deaths in Groups 2 and 3 of the Midhurst series. It happens, fortuitously, that the number of years between discharge or cessation of treatment and the final observation, is the same in both the Harefield and the Midhurst series. From the above figures, it is calculated that 57 per cent of the Midhurst cases who had received ordinary treatment had died at the end of a period of 3 to 4 years after discharge, as compared with 55 per cent of the Harefield cases after cessation of artificial pneumothorax treatment. In other words, the mortality in the two series is approximately the same. It should be added, however, that the social condition of the Midhurst cases was relatively good, the "working or industrial classes" to quote Bardswell,

not being eligible for treatment at Midhurst. Working-class patients, however, form the bulk of the cases treated at Harefield, and to quote again from Bardswell and Thompson's report, "the better the facilities for living under good conditions, the more hopeful the prospects of recovery". The Harefield cases, therefore returned to worse conditions than the Midhurst cases, so that the conclusion is that the mortality, after artificial pneumothorax treatment, of patients returning to relatively bad home conditions is similar to that, after ordinary sanatorium treatment, of patients returning to relatively good home conditions. That is to say, other things being equal, the mortality after artificial pneumothorax treatment is somewhat less than that after ordinary sanatorium treatment.

An excellent index of the result of any treatment of pulmonary tuberculosis is the fitness for work of patients, several years after the cessation of treatment. Table 9 deals with the fitness for work in 1929, of all cases at Harefield in which artificial pneumothorax was attempted. The cases

are divided into four groups, namely, cases with good collapse without adhesions, cases with good collapse adhesions being present, cases with partial collapse the result of adhesions, and cases in which adhesions prevented any collapse being obtained.

TABLE 9.

	Good collapse, no adhesions.	Good collapse. Adhesions.	Partial collapse. Adhesions.	No collapse. Adhesions.
Fit for work.	13	3	6	2
Unfit for work.	1	4	6	4
Dead.	11	10	15	20
Lost sight of.	-	-	1	4
Total	25	17	28	30

Owing to the fact that a comparatively small number of cases have been divided into many different groups, it is difficult to draw any reliable general conclusions from the above table. It is obvious, however, that more than half the cases in which good collapse without adhesions was obtained are fit for work at the moment. On the other hand,

two-thirds of the cases, in which no collapse was obtained owing to the presence of adhesions, are dead, and only 2 are fit for work. There is little to choose between the results in the two central columns, but it is noteworthy that relative to the results in the first column, the fitness for work as indicated in these central columns is less and the mortality is greater, and that relative to the fourth column, the fitness for work is greater and the mortality less.

Saugman and Gravesen (22) give an analysis of 211 "third-stage" cases, two to twelve years after discharge, and Table 10 shows their results arranged similarly to the Harefield results in Table 9.

T A B L E 10.

	Good Collapse. No adhesions.	Good collapse. Adhesions.	Partial collapse. Adhesions.	No collapse. Adhesions.
Fit for work.	33	14	5	9
Unfit for work.	1	-	-	3
Dead.	12	28	40	63
Lost sight of.	1	-	-	2
Total.	47	42	45	77

On comparison with Table 9, it appears that the mortality in the series of Saugman and Gravesen was higher than that in the Harefield series, though in the first column they obtained a greater percentage of cases fit for work. The higher mortality may be explained by the facts that the Danish cases were more advanced on induction and that the final observation took place from two to twelve years after discharge, as compared with from one to five years in the Harefield series. The larger number of patients fit for work as recorded in the first column in Table 10 is possibly due also to the longer period between treatment and final observation, for it has already been noted in Table 8 that the number of definitely improved Harefield cases increased slightly during the fifteen months subsequent to September 1928. It is possible, then, that within definite limits the number of improved cases may continue to increase slightly as time goes on.

From Tables 9 and 10 it is clear that, the greater the number of adhesions present and in consequence the smaller the collapse obtained, the

less chance there is of fitness for work being attained and the greater chance there is of death occurring.

Table 11 shows the first hundred cases of artificial pneumothorax cases grouped according to the condition on induction, divided according to the degree of collapse obtained, and further sub-divided according to the change in condition in 1929 relative to the condition on induction. Four cases lost sight of are included in Table 11 and one such case in Tables 12 and 13, the condition of each of these cases being recorded as that observed on the occasion of the last examination.

TABLE 11.

	A.	B1.	B2.	B3.	Total.
GOOD COLLAPSE.					
Improved.	-	-	13	2	15
No material improvement	1	1	4	-	6
Dead.	-	-	7	14	21
PARTIAL COLLAPSE.					
Improved.	2	-	4	1	7
No material improvement	1	-	3	2	6
Dead.	-	-	4	11	15
NO COLLAPSE.					
Improved.	-	-	2		2
No material improvement	-	-	4	4	8
Dead.	2	-	8	10	20

The figures relating to Groups A and B1 are very small, but it is noteworthy that both cases in which no collapse was obtained are now dead, and that no deaths occurred in the five cases in which either complete or partial collapse was obtained. Of the remaining groups, the better results are found in Group B2, especially so when good collapse has been achieved. The results in Group B3 are very poor, the mortality being very heavy, but it is doubtful if any

of the B3 cases would be alive now if artificial pneumothorax had not been instituted. Two-thirds of the cases in which no collapse was obtained are dead, the mortality in the successfully collapsed cases being considerably less. This demonstrates that while artificial pneumothorax fails in many cases, it is of very definite value in many others.

THE FATE OF CASES WITH BILATERAL DISEASE ON INDUCTION AS COMPARED WITH THAT OF CASES OF UNILATERAL DISEASE IS SHOWN IN TABLE 12. THE FINAL OBSERVATION WAS IN DECEMBER 1929, AND ONLY SUCCESSFULLY COLLAPSED CASES ARE DEALT WITH.

T A B L E 12.

	Bilateral disease.		Unilateral disease.	
	No.	Per cent.	No.	Per cent.
Improved.	6	25	16	35
No material improvement.	3	12.5	9	19
Dead.	15	62.5	21	46

These figures, as one would expect, show that cases with unilateral disease give better results with artificial pneumothorax treatment than do the bilateral cases.

Theoretically, the effects of pleural effusion may be beneficial in that the fluid assists in keeping the lung collapsed so that risk of expansion during the refill intervals is diminished. Further,

a frequent result of pleural effusion is pleural thickening which may strengthen the mediastinal partition and so prevent harmful mediastinal displacement. A further advantage of fluid development is that fewer refills at less frequent intervals are necessary. On the other hand, there are definite disadvantages such as dyspnoea due to pressure of the fluid, systemic disturbances and the risk of secondary infection of the fluid. Saugman (23) found that cases without pleural effusion did very slightly better than the cases in which fluid developed. Of his cases with effusion, 37 per cent were fit for work and 58 per cent were dead, while of the cases without effusion 40 per cent were fit for work and 53 per cent were dead. The following table shows the results in 1929 of the successfully collapsed cases at Harefield, divided according to the presence or absence of effusion.

TABLE 13.

	With Effusion. No.	Per cent.	Without Effusion. No.	Per cent.
Improved.	7.	22	15	39
No material improvement.	3	9	9	24
Dead.	22	69	14	37

It is clear that in the Harefield cases, the development of fluid during artificial pneumothorax had very definite detrimental effects on the results of treatment. Although the Harefield cases are not strictly comparable with those of Saugman, it is obvious that while little difference in the final condition of Saugman's cases is noted relative to presence or absence of fluid in these cases, there is a marked difference between such Harefield results.

The explanation, possibly, is that Saugman has included in his "with effusion" results, the results of those cases in which a transient effusion occurred. As mentioned earlier in this section, it is probable that most artificial pneumothorax cases develop a slight amount of fluid which persists for a few days only. Such transient effusions in the Harefield

cases are disregarded, so far as classing cases according to the presence or absence of fluid is concerned. That is to say, many of Saugman's favourable cases would be classed as having had effusion, while of the Harefield series similar favourable cases are classed as not having had effusion.

A summary of the first hundred artificial pneumothorax cases at Harefield Sanatorium is given in Appendix B. This summary deals with the salient features of each individual case, before, during and after treatment. The following cases are described in detail in order to illustrate so far as possible the various conditions, complications and results met with in artificial pneumothorax treatment.

The case numbers correspond with those of the general summary in Appendix B.

CASE 7. Female. Aged 25 years on admission on 10. 9. 25. Clerk. She had been discharged from Harefield Sanatorium 2 years earlier after ordinary sanatorium treatment. She had been working in the interval but before re-admission had lost weight and complained of cough, breathlessness, chest pains and night sweats. Haemoptysis in 1919 was the first

symptom of pulmonary tuberculosis.

On re-admission, the general condition was poor. Tubercle bacilli were found in the sputum. In the right lung, dullness was present throughout and tubular breathing and coarse crepitations were heard in all lobes. At the left apex, the abnormal signs were slight dullness, harsh inspiratory sound and fine crepitations.

Seven weeks later, the signs were, dullness specially marked at right apex, medium crepitations in all lobes of the right lung and most numerous in upper lobe. The left lung was apparently clear except for a group of fine crepitations in the region of the first left sternocostal articulation. Radiography showed widespread disease in right lung, the apex of left lung, too, being definitely involved. The temperature was oscillating, on an average, between 100°F. and 102°F.

Without collapse therapy, the prognosis was bad. Induction was performed on the right side on 3. 11. 25, followed by 11 refills. The intrapleural pressures before inserting air, the amount of air inserted and the final intrapleural pressures are

given below. Pressures are given in centimetres of water and the amount of air in cubic centimetres.

DATE	INITIAL PRESSURES.	AIR.	FINAL PRESSURES.
3. 11. 25.	- 27 - 10 cm.	350 c.c.	- 16 - 5 cm.
5. 11. 25.	- 21 - 5 cm.	340 c.c.	- 11 - 1 cm.
7. 11. 25.	- 18 - 5 cm.	400 c.c.	- 10 - 2 cm.
10. 11. 25.	- 15 - 4 cm.	400 c.c.	- 7 - 0 cm.
14. 11. 25.	- 15 - 3 cm.	400 c.c.	- 9 - 0 cm.
19. 11. 25.	- 12 - 1 cm.	400 c.c.	- 4 - 0 cm.
26. 11. 25.	- 13 - 1 cm.	400 c.c.	- 4 - 0 cm.
5. 12. 25.	- 15 - 1 cm.	550 c.c.	- 6 + 2 cm.
19. 12. 25.	- 10 + 1 cm.	350 c.c.	- 2 + 6 cm.
9. 1. 26.	- 9 + 2 cm.	900 c.c.	- 3 + 3 cm.
19. 1. 26.	- 7 + 3 cm.	200 c.c.	- 0 + 10 cm.
25. 1. 26.	- 3 + 6 cm.	100 c.c.	0 + 8 cm.

The development of fluid was discovered on 8. 12. 25. The accumulation of this fluid caused the higher initial pressures found at refills subsequent to that date. On 9. 1. 26, 1700 cubic centimetres of fluid were aspirated and replaced with 900 cubic centimetres of air, but fluid continued to accumulate.

CHART 1.

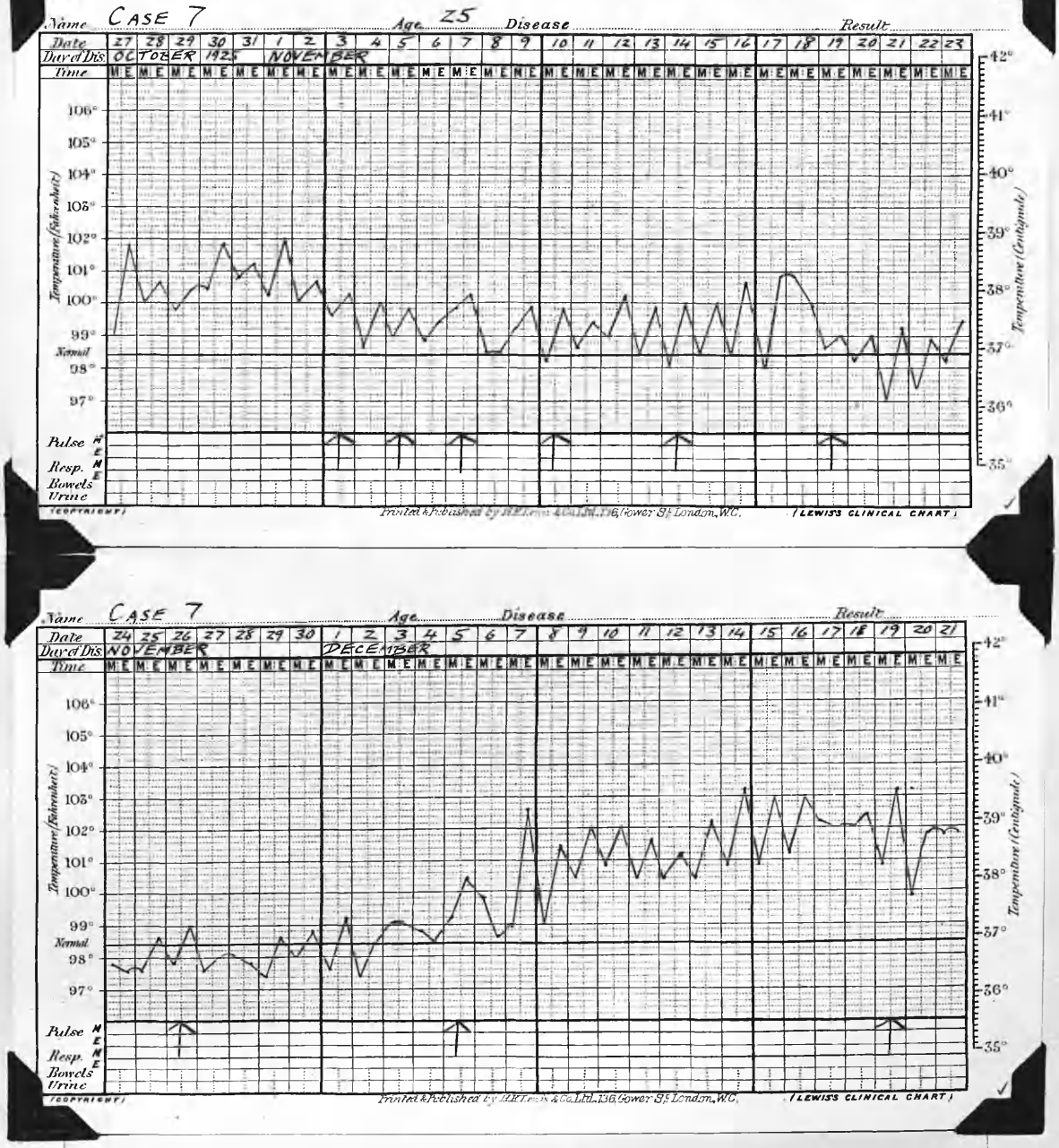


Chart 1. shows a fall of temperature at the end of the first three weeks after induction, the temperature becoming practically normal. The arrows indicate the days on which induction or refill was performed. An attack of pleurisy with effusion followed the refill on 5. 12. 25, the temperature rising to 100.4°F, that evening and later rising

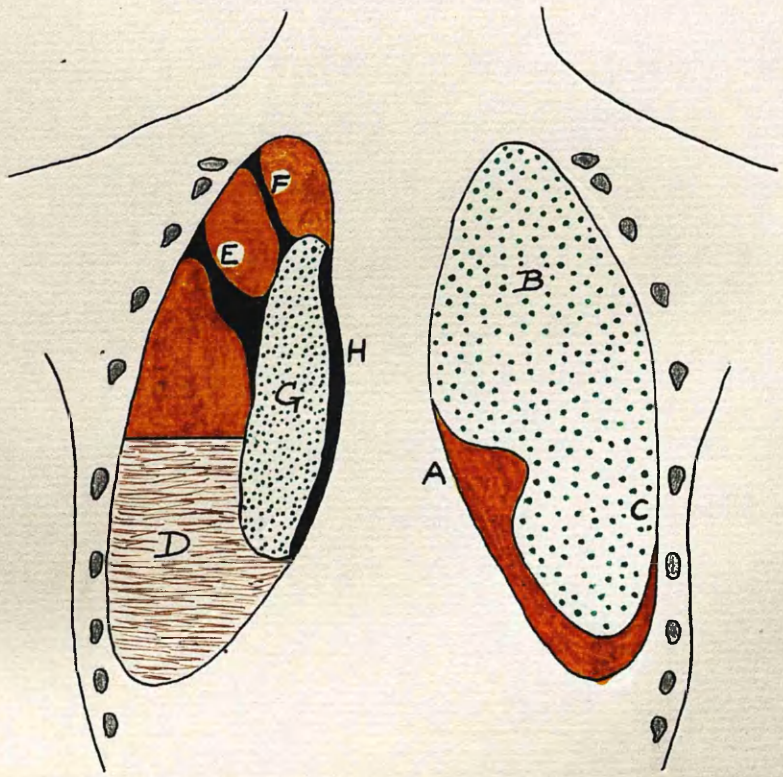
much higher. Thereafter, the patient became progressively worse and died on 13. 2. 26. A chill contracted at the time of the refill operation, the patient being in a very weak condition, may have caused the pleurisy on 5. 12.25, or it may be that the increased final pressure on that date as compared with previous pressures, was the cause of a pleural irritation followed by pleurisy with effusion. Whatever the cause, the pleurisy doubtless caused the death of the patient who before the onset of this complication had showed some signs of benefiting from collapse therapy.

An autopsy was performed and Diagram G illustrates the condition of the lungs post-mortem. The artificial pneumothorax collapse was practically complete, and the right pleural cavity was half full of thin yellowish-green turbid fluid. No organisms, other than tubercle bacilli, were found in this fluid. A core of lung persisted in the conical adhesion almost as far as the attachment of the latter to the parietal pleura. The collapsed lung was very extensively infiltrated with tuberculosis and multiple small cavities containing tuberculous pus were found.

DIAGRAM G.

CASE 7.

POST-MORTEM FINDINGS.



A. PERICARDIAL EFFUSION. NO TUBERCLES.

B. EXTENSIVE PERI-BRONCHIAL TUBERCULAR INFILTRATION INVOLVING WHOLE UPPER LOBE.

C. LOCALISED DISEASE IN LOWER LOBE.

D. PLEURAL EFFUSION.

E. CONICAL ADHESION.

F. SLENDER BAND ADHESION.

G. COLLAPSED LUNG.

H. DENSE ADHESIONS IN POSTERIOR MEDIASTINAL CAVITY.

The whole mediastinum was displaced towards the left side. Considerable fibrosis had already occurred in the collapsed lung and most of the alveolar spaces had been obliterated. The development of fibrous tissue in the lung after only 15 weeks of collapse is interesting, because one of the primary aims of pulmonary collapse is to encourage fibrosis in the diseased lung, and the earlier fibrous tissue develops and the wider it spreads, the greater chance there is of satisfactory healing taking place.

The presence of lung tissue in the conical adhesion shows that treatment of adhesions by division is not without danger, because if this particular adhesion had been divided, lung tissue would have been injured and a secondary infection of the pleural cavity would probably have resulted. Haemorrhage from the wounded organ, too, might have been very considerable and possibly fatal.

CASE 9. Female. Aged 23 years on admission to Harefield Sanatorium on 27. 7. 25. Shop assistant. She had an attack of bronchitis in 1921. Right artificial pneumothorax was induced at Brompton Hospital in 1922 for bronchiectasis. Tubercle bacilli

were found in the sputum in 1923. Refills were discontinued in 1925.

On admission, general condition was good, the temperature normal, slight sputum and dyspnoea present. The weight was 6 stones 7 pounds. Dullness and medium crepitations were found in the left upper lobe and signs of old pleurisy at the base of each lung. On 16. 11. 25, the cough was troublesome, an average of 30 cubic centimetres of sputum were expectorated daily and the sputum was occasionally blood-stained. The pulmonary signs were unchanged. On 16. 2. 25, the weight was 7 stones 3 pounds. Persistent "staining" had been present for four weeks. In addition to signs already found, post-tussive crepitations were heard at both bases, more numerous at the left. With a view to obtaining cessation of "staining", a low pressure artificial pneumothorax was considered necessary. It was impossible to decide which lung was responsible for the "staining", but as the left lung was more extensively diseased than the other, this lung was chosen for collapse.

On 13. 4. 26, pneumothorax was induced on the left side. The average evening temperature rose to

99°F. for a fortnight; thereafter it was normal. Blood-staining of the sputum ceased six weeks after induction. On 29. 6. 26, crepitations were still audible at the right base. The weight was 7 stones 2 pounds, the patient was able to take walking exercise and on the whole, the progress was satisfactory. On 28. 9. 26, the temperature was normal, "staining" had recurred and had been persistent for three weeks. The sputum was slight in amount. The apex of the lung was well collapsed, the base remaining uncollapsed. Activity was still present in the right base. On 26. 12. 26, the weight was 7 stones 3 pounds, "staining" was absent and the patient was again taking walking exercise. Crepitations were found at both bases. On 8. 3. 27, she was discharged as unimproved, but instructed to return at intervals for refills. On 11.8. 28, refills had to be discontinued because the left lung had re-expanded. In December 1929, symptoms had disappeared and the patient was fit for work. Signs of old pleurisy were found at both bases, but there were no signs of active disease.

In this case, right-sided bronchiectasis had

been successfully treated by artificial pneumothorax at Brompton Hospital. The presence of activity at both bases made it impossible to decide accurately the source of the blood in the sputum. Subsequent events proved that the choice of the left lung for collapse was justified. If by chance, the right lung had been chosen for collapse, blood-staining of the sputum would probably have continued and possibly a more severe pulmonary haemorrhage would have occurred from the left lung. In this event, the collapse of the right lung would have been abandoned and collapse of the left lung instituted.

An unusual incident occurred during a refill in this case. After 200 cubic centimetres of air had been inserted into the left pleural cavity, the patient complained of sudden pain in the left side and stated that she had experienced a sensation of something snapping inside the chest. The air reservoir was cut off, and it was found that the mean intrapleural pressure was lower than the initial mean intrapleural pressure had been. The refill was continued and it was found necessary to

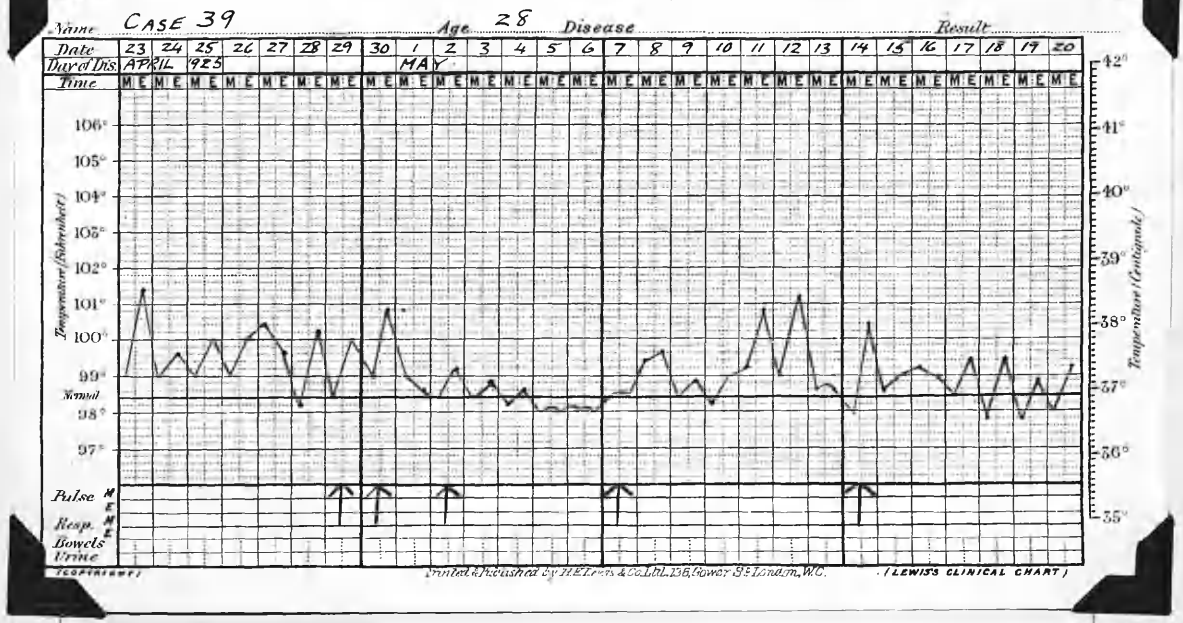
insert more air than at previous refills in order to obtain a mean final pressure similar to previous mean final pressures. The conclusion is that an adhesion was preventing collapse of a portion of lung and had given way during the refill, and so the size of the pleural cavity was increased and the intrapleural pressure reduced. It is probable that this adhesion had been subjected to progressive stretching as refill after refill was given, and the breaking point having been reached, a little more air was sufficient to snap the adhesion.

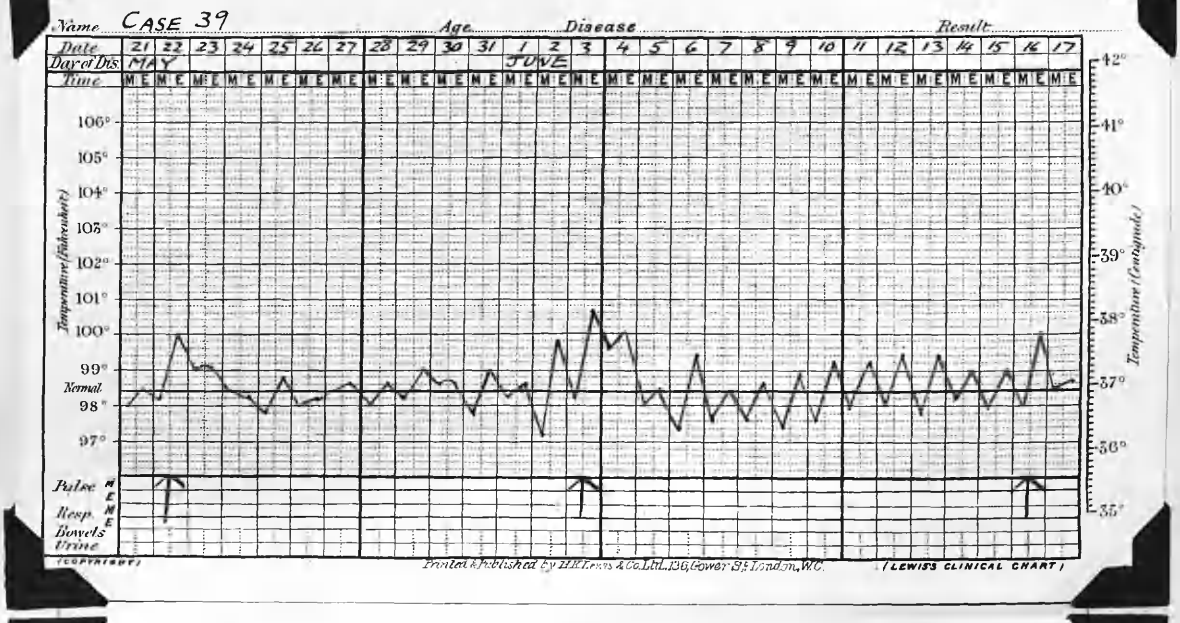
CASE 39. Female. Aged 28 years on admission to Harefield Sanatorium on 29. 1. 25. Clerk. Mother had died of pulmonary tuberculosis. The first symptoms were noticed in November 1924, and the disease was diagnosed in January 1925. On admission, the general condition was only fair, night sweats were present and cough was troublesome. 80 cubic centimetres of sputum were expectorated daily, the evening temperature was 99°F. - 100°F., and the pulse rate was 88 - 95 per minute. From the apex to the base of the left lung posteriorly and from the apex to the fifth rib anteriorly dullness,

medium crepitations, and fine friction sounds were found. No abnormal signs were discovered in the right lung. Many tubercle bacilli were found in the sputum on admission and also during the first two months of artificial pneumothorax treatment, though in diminishing numbers. Thereafter, the sputum was negative. Ordinary sanatorium treatment was given for four months and at the end of that time no progress had been made. Blood-stained sputum was occasionally noted.

Artificial pneumothorax was induced on the left side on 29. 5. 25. At the end of three months, the temperature was normal, cough was much improved and the amount of sputum per day was slight.

CHART 2.


Name CASE 39
Age 28
Disease
Result
Date 23 24 25 26 27 28 29 30 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20
Day of Dis. APRIL 1925 MAY
Time M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E
106° 105° 104° 103° 102° 101° 100° 99° Normal 98° 97°
Temperature (Fahrenheit)
42° 41° 40° 39° 38° 37° 36° 35°
Temperature (Centigrade)
Pulse M E
Resp. M E
Bowels
Urine
(COPYRIGHT)
Printed & Published by H.K. Lewis & Co. Ltd. 136, Gower St. London, W.C.
(LEWIS'S CLINICAL CHART)


Name CASE 39
Age
Disease
Result
Date 21 22 23 24 25 26 27 28 29 30 31 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17
Day of Dis. MAY JUNE
Time M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E
106° 105° 104° 103° 102° 101° 100° 99° Normal 98° 97°
Temperature (Fahrenheit)
42° 41° 40° 39° 38° 37° 36° 35°
Temperature (Centigrade)
Pulse M E
Resp. M E
Bowels
Urine
(COPYRIGHT)
Printed & Published by H.K. Lewis & Co. Ltd. 136, Gower St. London, W.C.
(LEWIS'S CLINICAL CHART)

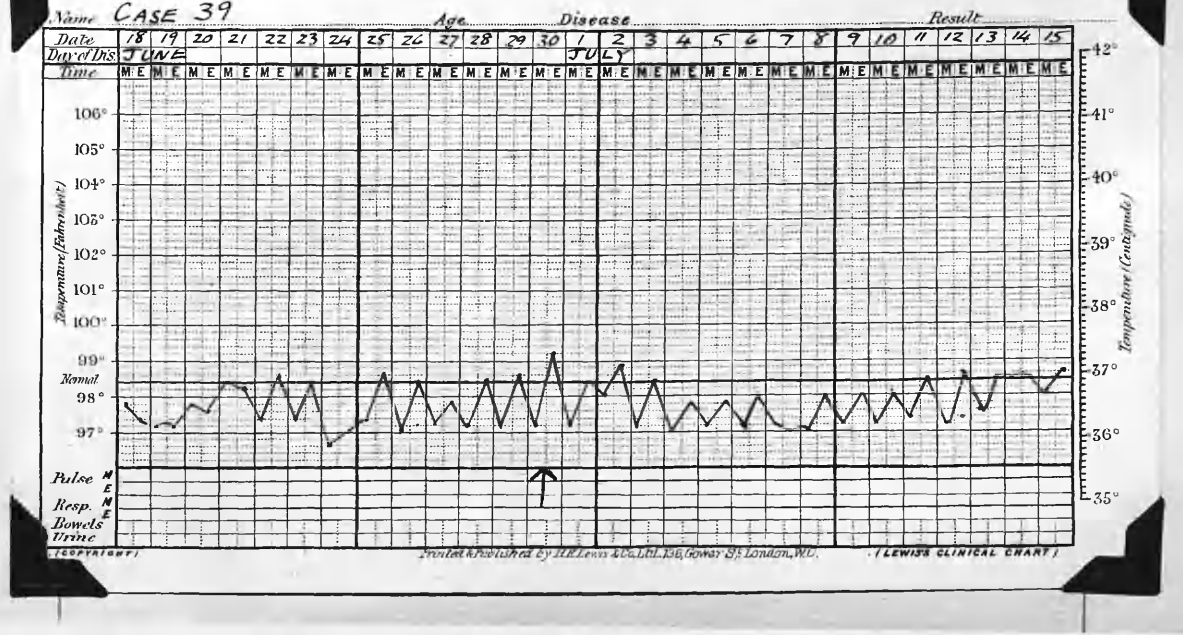

Name CASE 39
Age
Disease
Result
Date 18 19 20 21 22 23 24 25 26 27 28 29 30 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15
Day of Dis. JUNE JULY
Time M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E
106° 105° 104° 103° 102° 101° 100° 99° Normal 98° 97°
Temperature (Fahrenheit)
42° 41° 40° 39° 38° 37° 36° 35°
Temperature (Centigrade)
Pulse M E
Resp. M E
Bowels
Urine
(COPYRIGHT)
Printed & Published by H.K. Lewis & Co. Ltd. 136, Gower St. London, W.C.
(LEWIS'S CLINICAL CHART)

Chart 2 shows the high fluctuating temperature before induction and the definite fall of temperature after the first few refills. A rise of temperature on the evening following a refill is consistently noted, and this is no doubt due to autoinoculation. Later, however, when good collapse of the lung had been obtained, these temporary elevations of temperature ceased. The fact that weight rarely increases during artificial pneumothorax treatment has already been commented on, but this case shows definite increase of weight, as shown below.

29. 5. 25.	7 stones	8 pounds.	(On induction)
18. 7. 25.	7 "	10 "	
23. 8. 25.	8 "	2 "	
12. 9. 25.	8 "	5 "	
6.10. 25.	8 "	8 "	
16. 2. 26.	8 "	12 "	
15. 9. 27.	8 "	12 "	(On cessation)

Signs of active disease in the upper lobe of the right lung were found on 8. 11. 25, and a few rhonchi were heard in both lungs. A gradual expansion of the left lung necessitated the abandon-

ment of pneumothorax on 15. 9. 27. The general condition was much improved, some disease was present in the right lung, but no abnormal signs were detected on the left side, except that breath sounds were broncho-vesicular and some friction was present. The patient was now working as a clerk. In September 1928, the weight was 7 pounds less than on cessation of treatment, but the general health was good. Activity was still present in the right upper lobe and rhonchi in both lungs. About this time the patient married, against medical advice, but her new home conditions were excellent. She became pregnant and died a few weeks after child birth, the pulmonary disease having spread with great rapidity.

This tragic case illustrates how artificial pneumothorax treatment rendered an advanced case of pulmonary tuberculosis fit for ordinary work. While unmarried, the patient took good care of herself and was apparently holding her own. Pregnancy, however, as it so frequently does, lit up the pulmonary disease, and the good results of many months of patient work were lost.

CASE 46. Female. Aged 18 years on admission to Harefield Sanatorium on 11. 3. 24. Laundry worker. Her brother had died of pulmonary tuberculosis. She had complained of winter cough for three years.

On admission, cough and sputum were present and the temperature was normal. The weight was 7 stones 9 pounds. Tubercle bacilli were present in the sputum. The chest expansion was poor. At the left apex, dullness, harsh inspiratory murmer and post-tussive crepitations were present. The right lung was clear. Radioscopy showed general haziness on the left side, lighting-up on deep inspiration being good. No abnormal shadows were noticed on the right side, the diaphragmatic movement was good and the position of the heart normal. On 29. 7. 24, cough was troublesome, 10 cubic centimetres of sputum were expectorated daily, the temperature was normal and the pulmonary signs unchanged. In other words, there had been no response to four and a half months of ordinary sanatorium treatment.

Artificial pneumothorax was induced on the left side on 31. 7. 24. and good collapse was eventually obtained, the average refill being 600 cubic

centimetres of air. No tubercle bacilli were found in the sputum on 1. 9. 24. and cough and sputum had disappeared by March 1925 and have never recurred. This patient is still having artificial pneumothorax refills and no complications have occurred during this special form of treatment. Against medical advice, she has been working as a laundress for three years and her general health has remained good. The weight varies between 8 stones 9 pounds and 9 stones.

The absence of complications, as in this case, during more than five years of treatment, is quite unusual. No improvement was obtained with ordinary treatment, yet eight months after induction, symptoms had disappeared and later, collapse therapy enabled the patient to undertake with impunity, the heavy duties of a laundress, working under bad conditions.

CASE 61. Male. Aged 17 years on admission to Harefield Sanatorium on 31. 5. 26. Carpenter. Definite family history of tuberculosis was elicited, his mother and brother having suffered from the disease. The earliest symptoms appeared about six months before admission and haemoptysis occurred for the first time on 6. 5. 26.

On admission, the general condition was fair. The weight was 10 stones 11 pounds, and the height 6 feet. The sputum contained tubercle bacilli throughout treatment. 15 cubic centimetres of sputum were expectorated daily. The evening temperature was 99°F. On examination, no abnormal signs were found in the right lung. Dullness and crepitations were present on the left side from the apex to the level of the sixth rib anteriorly. In addition, signs of cavitation were found in the left nipple area. Radiography showed disease apparently affecting the whole of the left lung, and some mottling was present at the apex of the right lung.

On 29. 8. 26. in the early morning, 280 cubic centimetres of blood were coughed up, followed by a rise of temperature. The right lung being apparently clear, the left lung was regarded as the source of haemorrhage, especially as signs of cavitation were present. Left artificial pneumothorax was at once induced, 350 cubic centimetres of air being inserted.

CHART 3.

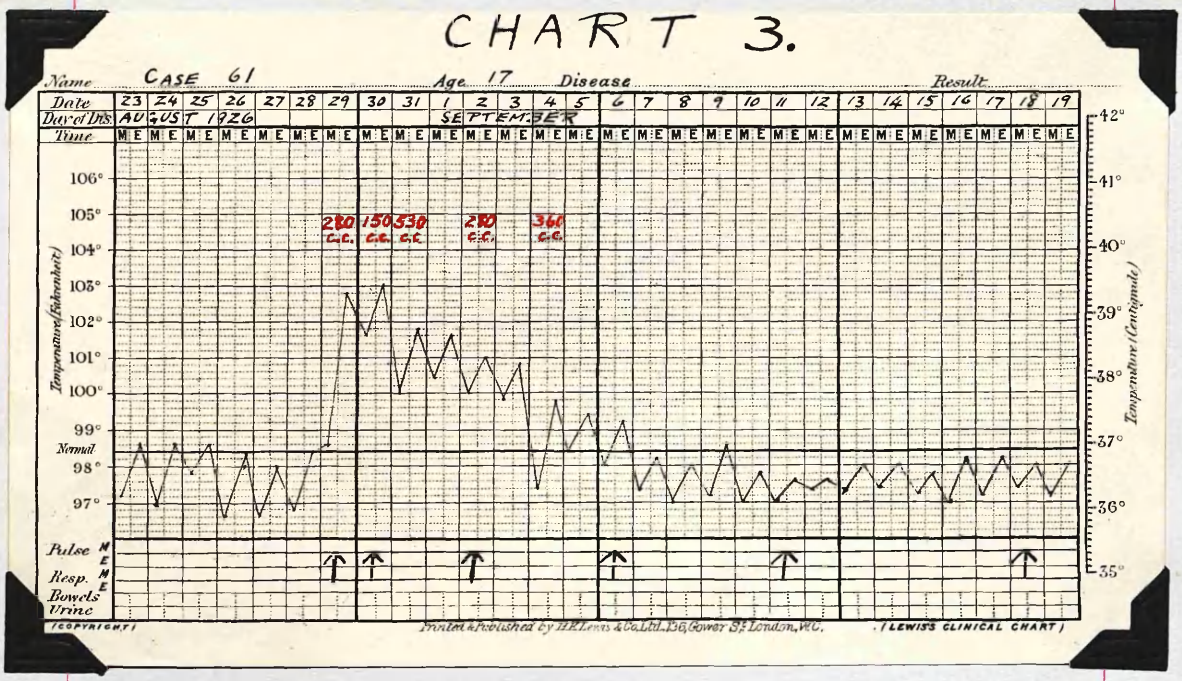


Chart 3 shows the rapid rise of temperature and the gradual fall to normal over a period of one week. The red figures show the amount of haemorrhage on the days indicated. The initial intrapleural pressures are indicated below.

DATE.	INITIAL PRESSURES.	AIR.	FINAL PRESSURES.
29. 8. 26.	- 4 0 cm.	350 c.c.	0 + 4 cm.
30. 8. 26.	- 12 - 2 cm.	350 c.c.	+ 12 + 16 cm.
2. 9. 26.	- 10 0 cm.	200 c.c.	+ 2 + 8 cm.
6. 9. 26.	- 12 0 cm.	130 c.c.	0 + 8 cm.
11. 9. 26.	- 12 0 cm.	200 c.c.	+ 2 + 8 cm.
18. 9. 26.	- 18 0 cm.	150 c.c.	+ 2 + 10 cm.
26. 9. 26.	- 10 + 3 cm.	75 c.c.	0 + 10 cm.
6. 10. 26.	No oscillations.	Nil.	-------

Adhesions formed rapidly and on 6. 10. 26, no free space was found because the lung was adhering to the parietes, necessitating abandonment of collapse therapy. It is seen that at the last two refills, smaller quantities of air produced practically the same final pressures, no doubt due to the gradual contraction of the pleural space by the adhesions.

Haemoptysis did not recur after the fifth refill and the temperature remained normal throughout subsequent sanatorium treatment. Signs of slight affection of the right apex were found four weeks after induction. The patient was discharged at his own request on 8. 10. 27, in practically the same condition as he was admitted. No change in his condition was noted in September 1928 nor in December 1929. He was then unfit for work. There had been no recurrence of haemoptysis since discharge.

This case illustrates the value of artificial pneumothorax in the treatment of sudden haemoptysis. A total of 1600 cubic centimetres of blood were lost in one week, and the pulmonary haemoptysis, without collapse therapy, might easily have proved fatal. The rapidity of spread of adhesions is

interesting, though from the rapid rise of intra-pleural pressure following the insertion of a small amount of air, one concluded that adhesions were present before induction.

CASE 95. Male. Aged 19 years on admission to Harefield Sanatorium on 22. 6. 25. Clerk. No family history of tuberculosis. The onset of the disease was insidious. On admission the general condition was good, cough and sputum were present and the temperature was normal. The weight was 9 stones 8 pounds. There was occasional blood-staining of sputum. The chest expansion was found to be deficient on the right side. Fine crepitations were heard at both apices and at the right base. Tubercle bacilli were found in the sputum for the first time, two months after admission.

"Staining" of the sputum continued for more than a year after admission. One haemoptysis occurred in December 1925, 100 cubic centimetres of blood being lost. Calcium chloride solution, injected intramuscularly, failed to stop the "staining". On 31. 3. 26. considerable spread of disease in the left lung was noted to have occurred, the whole

of the left upper lobe being affected. No spread had occurred in the right lung. On radioscopy, however, dense shadows were seen in the upper half of the right lung as well as in the greater part of the left lung. On 8. 7. 26, the temperature varied from 99°F. to 100°F. and "staining" was still present. On 30. 9. 26. a large haemorrhage occurred, 800 cubic centimetres of blood being lost. Since disease was more widespread in the left lung, induction on the left side was performed, 700 cubic centimetres of air being inserted. Refills were given every day for three days, and a further 1700 cubic centimetres of blood were coughed up during that period. Thereafter, severe haemorrhage ceased but "staining" of the sputum continued for three weeks. Meanwhile, the temperature had fallen to normal. Good collapse of the left lung had been obtained and refills were continued. On 1. 12. 26. there was a recurrence of "staining", the temperature rose and oscillated between 100.6°F. and 103°F. and the patient died on 26. 2. 27. of bronchopneumonia secondary to pulmonary tuberculosis.

This was a case in which the most persistent

symptom was blood-staining of the sputum. Treatment other than artificial pneumothorax was given, but failed to stop the "staining", and the disease advanced. When a severe haemorrhage occurred, collapse therapy was tried with some success at first, but the onset of bronchopneumonia, probably due to aspiration of blood into the bronchioles, caused the death of the patient.

SUMMARY AND CONCLUSIONS.

1. Artificial pneumothorax treatment is of definite value in cases in which ordinary sanatorium treatment has failed. Very advanced cases, on the whole, do not do well, but the average results in intermediate cases are good, especially when complete collapse is obtained. The mortality is very heavy, but is slightly less than that after ordinary sanatorium treatment.
2. Pulmonary collapse is an excellent measure when grave pulmonary haemorrhage has occurred.
3. The dangers of artificial pneumothorax operations can be reduced to a minimum by care on the part of the operator, the use of a small-bore Saugman needle for all operations and the liberal injection of a non-toxic local anaesthetic such as a solution of benzamine hydrochloride associated with adrenalin chloride.
4. In the treatment of pulmonary tuberculosis by artificial pneumothorax, laryngeal tuberculosis, if present, is cured or much improved in cases

in which good pulmonary collapse is obtained.

5. Good results in artificial pneumothorax treatment are obtained in inverse ratio to the number and extent of adhesions in the side treated. More than half of the cases in which good collapse without adhesions was obtained are now fit for work, but less than a sixth of the other cases of artificial pneumothorax are able to follow any occupation.
6. Other factors being equal, the percentage of cases in which as a result of collapse therapy, the sputum is absent or non-infectious is greater than that of cases in which sanatorium treatment alone has been given.
7. In cases in which disease was bilateral before induction and in cases in which pleural effusion occurred during treatment, the results of collapse therapy are much below the average.

BIBLIOGRAPHY.

1. Carson, J. Essays, Physiological and Practical, Wright, Liverpool, 1822, 56 and 62.
2. Forlanini, C. Gazz. Deg. Osp., 1882, iii, 537.
3. Cayley, W. Trans. Clin. Soc. London, 1885, xviii, 278.
4. de Cérenville, Rev. Méd. de la Suisse Rom., 1885, V, 441.
5. Murphy, J.B. Journ. Amer. Med. Assoc., 1898, xxxi, 342.
6. Lillingston, C. Practitioner, 1913, xc, 129.
7. Heynsius, A. Arch. F.D. Ges. Physiol., 1882, xxix, 267.
8. Halliburton, W.D. Handbook of Physiology, 1928, 261 and 312.
9. Graham, E.A. and Bell, R.D., Amer. Journ. Med. Science, 1918, clvi, 839 and 859.
10. Amberson, J.B., and Peters, A., Thoracic Surg., Lilienthal, 1925, ii, 324.
11. Burrell, L.S.T. and Macnalty, A.S., Med. Research Council, Special Report No. 67, 1922, 24 and 30.

12. Riviere, C. Pneumothorax and Surg, Treat. of Pulm. Tub., 1927, 14 and 153.

13. Emerson, C.P., Johns Hopkins Hosp. Reports, 1903, xi, 386.

14. Burrell, L.S.T., British Medical Journal, 1927, i, 135.

15. Morgan, W.P. Lancet, 1913, ii, 18.

16. Peters, A. Thoracic Surg., Lilienthal, 1925, ii, 325.

17. Morgan, W.P. Quart. Journ. Med., 1917, xi, 1.

18. Rist, E. Quart. Journ. Med., 1913, vi, 276.

19. Edwards, A.T., Medical Annual, 1925, 474.

20. Rist, E. and Hirschberg, F., Paris Méd., 1928, LXV11, 17.

21. Bardswell, N.D., and Thompson, J., Med. Research Comm., Special Report No. 33, 1919, 7 and 18.

22. Saugman, C. and Gravesen, J., British Medical Journal, 1923, ii, 506.

23. Saugman, C. Lancet, 1920, ii, 687.

24. Ministry of Health, Tuberculosis (Eng.), Mem. 37/T, 1925, 2.

A P P E N D I X A.

The Ministry of Health (24) has suggested that patients suffering from tuberculosis should be classified in accordance with the following scheme. All cases at Harefield, including those treated by artificial pneumothorax, are classified according to this system.

Classification. - Patients diagnosed as suffering from Pulmonary Tuberculosis are placed in the following categories:-

Class T. B. minus (Group A), viz., cases in which tubercle bacilli have never been demonstrated in the sputum; and

Class T. B. plus (Group B), viz., cases in which tubercle bacilli have at any time been found. It should be noted that a patient originally in Class T.B. minus must be transferred to Class T.B. plus at any stage in the course of treatment if and when tubercle bacilli are found; while, on the other hand, a patient who is once placed in Class T.B. plus can never revert to Class T.B. minus. Class T.B.plus is further subdivided into three groups as follows:-

Group 1 (Group B 1.) Cases with slight con-

stitutional disturbance, if any, e.g. there should not be marked acceleration of pulse nor elevation of temperature except of very transient duration; gastro-intestinal disturbance or emaciation, if present, should not be excessive.

The obvious physical signs should be of very limited extent as follows:- Either present in one lobe only and in the case of an apical lesion of one upper lobe not extending below the second rib in front or not exceeding an equivalent area in any one lobe; or where these physical signs are present in more than one lobe they should be limited to the apices of the upper lobes and should not extend below the clavicle and the spine of the scapula.

No complication (tuberculous or other) of prognostic gravity should be present. A small area of dry pleurisy does not exclude a case from this group.

Group 3 (Group B 3). Cases with profound systemic disturbance or constitutional deterioration, with marked impairment of function, either local or general, and with little or no prospect of recovery.

All cases with grave complications, whether tuberculous or not, are classified in this group, e.g., diabetes, tuberculosis of larynx or intestine, etc.

Group2(Group B2). All cases which cannot be placed in Groups 1 and 3.

Quiescent. Cases which have no symptoms of tuberculosis and no signs of tuberculous disease except such as are compatible with a completely healed lesion, and in which sputum, if present, is free from tubercle bacilli.

Arrested. In pulmonary cases the term "arrested" is applied only to cases which have been "quiescent" for a period of at least two years.

In non-pulmonary cases the term "arrested" is used as soon as there is reason to believe that the disease is unlikely to recur.

A P P E N D I X B.

Summary of Artificial Pneumothorax Cases at Harefield Sanatorium.

Key to Summary.

COLUMN.

1. Case Number.

11. Sex. M:- Male. F:- Female.

111. Age in years.

1V. Type of Onset of Disease.

I:- Insidious Onset.
P:- Onset with Pleurisy with effusion.
H:- Onset with marked Haemoptysis.

V. Duration (in years) of disease before induction.

Vl. Classification on Induction (Ministry of Health 1925).

Vll. Reason for Induction.

N.R. Little or no response to ordinary Sanatorium treatment.
N.R.H. Same as N.R. but some Haemoptysis also present.
H. Severe Haemoptysis.
P.B. Prognosis bad without A.P. treatment.
P.B.H. Same as P.B. but some Haemoptysis also present.

Vlll. Side Treated. L:- Left. R:- Right.

1X. Condition of Untreated Lung.

P. Disease Present.
A. Disease Absent.

X. Success of A. P. Treatment.

S.C. Good Collapse. No Adhesions.
A.S.C. Good Collapse. Few Adhesions.
S.P. Useful Partial Collapse. Adhesions.
U. Useless Collapse. Adhesions.
F. No Collapse. Adhesions.

Xl. Number of A. P. Punctures.

Xll. Average volume of air introduced at each refill in cubic centimetres.

Xlll. Development of Pleural Effusions.

P. Fluid Present.
A. Fluid Absent.

XlV. Spread of disease during A. P. treatment.

P. Spread Present.
A. Spread Absent.

XV. Classification on Cessation of A. P.

XVl. Condition on cessation of A. P. relative to condition on Induction.

A. Arrested. Q. Quiescent.
M.I. Much Improved.
S. Stationary. W. Worse.
D. Dead.

XVll. Period (in years) elapsing between cessation of A. P. treatment and observation in September 1928.

XVlll. Classification according to observation in September 1928.

XlX. Condition according to observation in September 1928 relative to condition on Induction.

S.U.T. Still under treatment.
L.S.O. Lost sight of.
Other contractions as in column XVl.

XX. Classification according to observation in December 1929.

XXI. Condition according to observation in December 1929 relative to condition on Induction.

XXII. Fitness for work in December 1929.

F. Fit for Work.
U. Unfit for Work.

Note. A. P. Artificial Pneumothorax Treatment.

I.	II.	III.	IV.	V.	VI.	VII.	VIII.	IX.	X.	XI.	XII.	XIII.	XIV.	XV.	XVI.	XVII.	XVIII.	XIX.	XX.	XXI.	XXII.
1	F	17	I	1¼	B2	NR	L	A	SC	60	400	A	P	B2	S	0	B2	S	B1	MI	F
2	F	33	I	3¼	B3	NRH	R	A	U	6	400	A	A	B3	S	3½	B3	W	B3	W	U
3	F	19	P	2½	B2	NR	L	A	F	2	0	A	A	B2	S	2¾	-	D	-	D	-
4	F	23	I	1	B2	NR	R	A	SC	66	800	A	A	B1	MI	-	-	SUT	-	SUT	F
5	F	39	I	15	B3	NR	L	P	SP	33	400	P	P	B2	MI	2¾	B3	W	B3	W	U
6	F	21	I	¾	B2	NR	R	P	SC	17	500	P	A	B3	W	3	B2	S	B1	MI	F
7	F	25	H	6½	B3	PB	R	P	ASC	11	400	P	P	-	D	0	-	D	-	D	-
8	F	22	I	¾	B2	NR	L	A	ASC	51	400	P	A	B2	MI	1¼	-	D	-	D	-
9	F	23	H	4	B2	H	L	P	SP	70	500	A	A	B2	S	0	B2	S	B1	Q	F
10	F	18	I	1	B3	PB	L	A	SP	35	300	P	A	B1	MI	½	-	D	-	D	-
11	F	31	P	1½	B2	H	R	A	SC	57	500	A	A	B1	MI	0	B1	MI	B1	MI	F
12	F	31	I	½	B3	NR	L	P	ASC	41	300	P	A	B2	MI	2	-	D	-	D	-
13	F	37	I	1	B2	H	L	A	SC	48	600	A	A	B1	Q	1½	B1	Q	B1	A	F
14	F	18	I	3¼	B3	NR	L	P	SP	35	400	P	P	B3	S	½	-	D	-	D	-
15	F	22	I	½	B3	PB	L	A	SC	21	200	P	P	B3	S	2	-	D	-	D	-
16	F	16	I	½	B3	PB	L	A	SP	15	400	A	A	B3	S	¼	-	D	-	D	-
17	F	19	I	4½	B2	NRH	R	P	SC	59	500	A	P	B2	S	-	-	SUT	B1	MI	F
18	F	23	I	1½	B2	NR	L	A	ASC	46	600	A	P	B1	MI	2¾	B2	S	B1	MI	F

I.	II.	III.	IV.	V.	VI.	VII.	VIII.	IX.	X.	XI.	XII.	XIII.	XIV.	XV.	XVI.	XVII.	XVIII.	XIX.	XX.	XXI.	XXII.
19	F	19	H	2¼	B3	PBH	R	A	ASC	33	800	A	A	B1	MI	2	-	D	-	D	-
20	F	37	I	1	B2	H	L	P	ASC	28	700	P	P	B2	S	2¼	B3	W	-	D	-
21	F	18	H	2	B3	NRH	L	A	ASC	42	800	A	A	B1	MI	2¼	B1	Q	B1	A	F
22	F	38	I	¾	B3	PB	L	P	F	3	0	A	P	B3	W	2¾	-	D	-	D	-
23	F	22	I	4	B2	NR	L	A	SP	27	500	P	P	B2	S	3¼	B2	S	B1	Q	F
24	F	25	I	2½	B2	NR	L	P	U	15	200	A	A	B3	W	¼	-	D	-	D	-
25	F	24	P	1	B2	H	L	P	SC	62	700	A	A	B3	W	¼	-	D	-	D	-
26	F	21	I	1¾	B3	NR	R	P	SP	40	300	A	P	B3	S	1¾	-	D	-	D	-
27	F	25	I	½	B3	PB	L	P	SP	8	200	P	P	-	D	0	-	D	-	D	-
28	F	30	I	½	B3	PB	R	A	SP	34	400	P	P	B3	S	¼	-	D	-	D	-
29	F	39	H	8	B2	NRH	R	A	U	8	200	A	A	B2	S	1	B3	W	-	D	-
30	F	28	I	½	B2	NR	R	A	F	9	0	A	A	B2	S	-	-	LSO	-	LSO	-
31	F	22	I	4	B3	NR	L	A	SP	34	300	P	A	B2	MI	1½	-	D	-	D	-
32	F	25	I	1	B3	PB	R	A	U	2	200	A	A	B3	S	1½	-	D	-	D	-
33	F	16	I	¾	B3	PB	L	A	SP	25	400	P	A	B1	MI	2	B3	S	-	D	-
34	F	25	I	1½	B3	NRH	L	P	SP	30	400	P	P	B3	S	¾	-	D	-	D	-
35	F	17	H	1¼	A	NR	L	A	SP	29	300	A	A	A	S	2	A	S	A	S	U
36	F	30	I	2	B3	NR	R	P	U	7	300	A	A	B3	S	3¼	-	D	-	D	-

I.	II.	III.	IV.	V.	VI.	VII.	VIII.	IX.	X.	XI.	XII.	XIII.	XIV.	XV.	XVI.	XVII.	XVIII.	XIX.	XX.	XXI.	XXII.
37	F	30	I	1/2	B2	PBH	L	A	SP	33	300	P	A	B1	Q	1 1/2	-	D	-	D	-
38	F	16	I	1/2	B3	PB	L	P	F	2	0	A	A	B3	S	1/4	-	D	-	D	-
39	F	28	I	1/2	B3	NR	L	A	SC	51	400	A	P	B2	MI	3/4	B2	MI	-	D	-
40	F	30	I	1	B2	H	L	A	SP	21	300	A	A	B1	MI	3/4	B2	S	B2	S	U
41	F	23	H	1 3/4	B2	NRH	R	A	ASC	16	700	A	A	B1	MI	3	B1	MI	B2	W	U
42	F	16	I	1/2	B3	PB	R	A	SC	41	400	A	A	B1	MI	-	-	SUT	-	SUT	F
43	F	16	I	1 1/2	B2	NR	L	A	SC	39	500	A	A	B1	Q	1	B1	Q	B1	Q	F
44	F	16	I	1/4	B3	PB	L	A	U	8	200	A	A	B3	S	2 3/4	B3	S	B3	S	U
45	F	24	P	1/2	B2	NR	L	A	SC	20	600	A	P	B3	W	1/2	-	D	-	D	-
46	F	18	I	2	B2	NR	L	A	SC	70	600	A	A	B1	Q	-	-	SUT	-	SUT	F
47	F	23	I	1	B2	NRH	R	A	SP	25	500	A	A	B1	MI	-	-	LSO	-	LSO	-
48	F	54	I	1 1/4	B2	NR	L	P	F	2	0	A	A	B2	S	-	-	LSO	-	LSO	-
49	F	21	P	2 1/2	B2	H	R	A	SC	86	600	P	A	B1	Q	0	B1	Q	B1	Q	F
50	F	18	I	3/4	B2	NR	L	A	U	2	100	A	A	B2	S	2	B3	W	B3	W	U
51	F	25	I	1/2	B3	PB	L	P	F	3	0	A	P	-	D	0	-	D	-	D	-
52	F	25	I	1/4	B3	PB	R	P	SC	12	600	A	P	B3	W	1/4	-	D	-	D	-
53	F	26	I	1 1/2	B2	PB	L	A	SP	69	300	P	A	B1	MI	-	-	SUT	B1	Q	F
54	F	16	I	2	B3	NRH	R	P	SP	17	200	P	P	B3	S	1 1/2	B3	S	B3	S	U

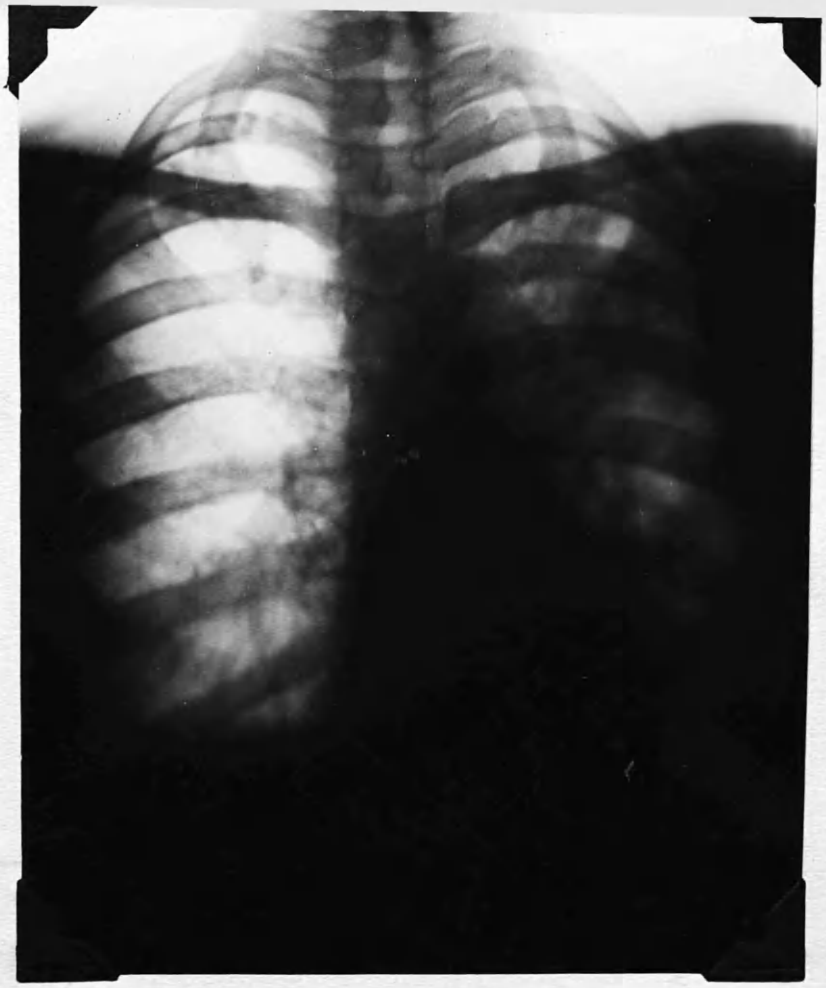
I.	II.	III.	IV.	V.	VI.	VII.	VIII.	IX.	X.	XI.	XII.	XIII.	XIV.	XV.	XVI.	XVII.	XVIII.	XIX.	XX.	XXI.	XXII.
55	F	17	I	¾	B3	NR	R	A	SC	33	500	P	P	B3	S	¾	B3	W	-	D	-
56	F	22	P	3¾	B2	NR	L	P	SP	70	400	A	P	B1	MI	¾	B1	MI	B1	MI	F
57	F	30	I	1¼	B3	PB	L	P	SC	19	500	P	A	B2	MI	1¼	B2	MI	-	D	-
58	F	22	I	¾	B2	NR	R	A	F	3	0	A	A	B2	S	2	-	D	-	D	-
59	F	18	I	⅛	B3	PB	R	P	F	3	0	A	A	B3	S	⅛	-	D	-	D	-
60	M	21	I	⅛	B3	PB	R	A	U	4	200	A	A	B3	S	0	-	D	-	D	-
61	M	17	I	1	B2	H	L	A	SP	8	200	A	P	B2	S	1¾	B2	S	B2	S	U
62	M	35	I	5	B3	PBH	R	P	SC	30	700	P	P	B3	S	¼	-	D	-	D	-
63	M	26	H	1	A	H	L	P	F	5	0	A	A	A	S	¾	-	D	-	D	-
64	M	29	P	7	A	NR	L	A	SC	23	600	A	A	A	Q	3¼	A	W	B2	S	F
65	M	15	I	1	B3	PB	L	A	ASC	29	500	A	A	B3	S	¼	B3	S	-	D	-
66	M	26	I	7	B2	NR	R	P	SC	30	600	A	P	B2	S	0	B2	S	B1	MI	F
67	M	31	I	14	B2	NR	R	P	U	7	200	A	A	B2	S	3½	B1	A	-	D	-
68	M	28	I	2¼	B2	NRH	L	A	SP	13	200	P	A	B3	W	⅛	-	D	-	D	-
69	M	43	I	¼	B3	PB	L	A	U	3	200	A	A	B3	S	¼	-	D	-	D	-
70	M	18	I	⅛	B2	NR	R	A	ASC	18	500	A	A	B1	MI	1	-	D	-	D	-
71	M	27	P	¾	B1	NR	L	A	SC	33	600	A	A	B1	S	2½	B1	S	B2	W	U
72	M	17	I	¾	B3	PB	R	A	ASC	44	400	P	P	B3	W	¾	-	D	-	D	-

I.	II.	III.	IV.	V.	VI.	VII.	VIII.	IX.	X.	XI.	XII.	XIII.	XIV.	XV.	XVI.	XVII.	XVIII.	XIX.	XX.	XXI.	XXII.
73	M	18	I	½	B2	NR	L	P	SC	24	500	A	A	B3	W	¼	-	D	-	D	-
74	M	19	I	½	B2	NR	R	A	ASC	37	700	A	A	B1	MI	-	-	SUT	-	SUT	F
75	M	29	I	5	B3	PB	L	A	F	3	0	A	P	B3	S	-	-	LSO	-	LSO	-
76	M	25	I	4	A	H	R	P	U	4	600	A	A	A	S	-	-	D	-	D	-
77	M	27	I	8	B2	H	R	P	F	6	0	A	A	B2	S	¾	-	D	-	D	-
78	M	33	I	2	B3	PB	L	P	SP	66	300	P	P	B3	W	¾	-	D	-	D	-
79	M	43	I	1	B2	H	L	A	SC	33	500	P	A	B1	MI	½	B1	MI	B1	MI	F
80	M	21	I	1¼	B2	NR	R	A	F	3	0	A	A	B2	S	1	B2	S	B1	MI	F
81	M	34	P	7	B3	NR	R	A	SP	52	600	P	A	B3	S	¾	-	D	-	D	-
82	M	20	I	3	B3	NRH	L	A	SP	62	400	A	P	B2	MI	1¼	B2	MI	B2	MI	F
83	M	22	H	1	B2	NRH	R	A	SP	16	400	P	A	B1	MI	¼	-	D	-	D	-
84	M	18	I	¼	A	PB	R	P	SP	24	300	P	A	B2	MI	3	B2	MI	B2	MI	U
85	M	19	I	4	B3	PBH	R	P	U	3	400	A	P	-	D	0	-	D	-	D	-
86	M	43	I	1½	B2	NR	R	A	SP	45	200	P	P	B2	S	1½	B3	W	-	D	-
87	M	49	I	1¾	B2	NR	L	A	F	3	0	A	A	B2	S	3¼	-	D	-	D	-
88	M	17	I	½	B2	NR	L	A	ASC	51	600	A	A	B1	MI	¼	B2	S	B2	S	U
89	M	23	I	1¼	B2	NR	L	A	F	4	0	A	A	B2	S	3¼	B3	W	-	D	-
90	M	24	P	½	B2	NR	R	A	U	15	100	A	A	B2	S	3¼	B1	Q	B1	Q	F

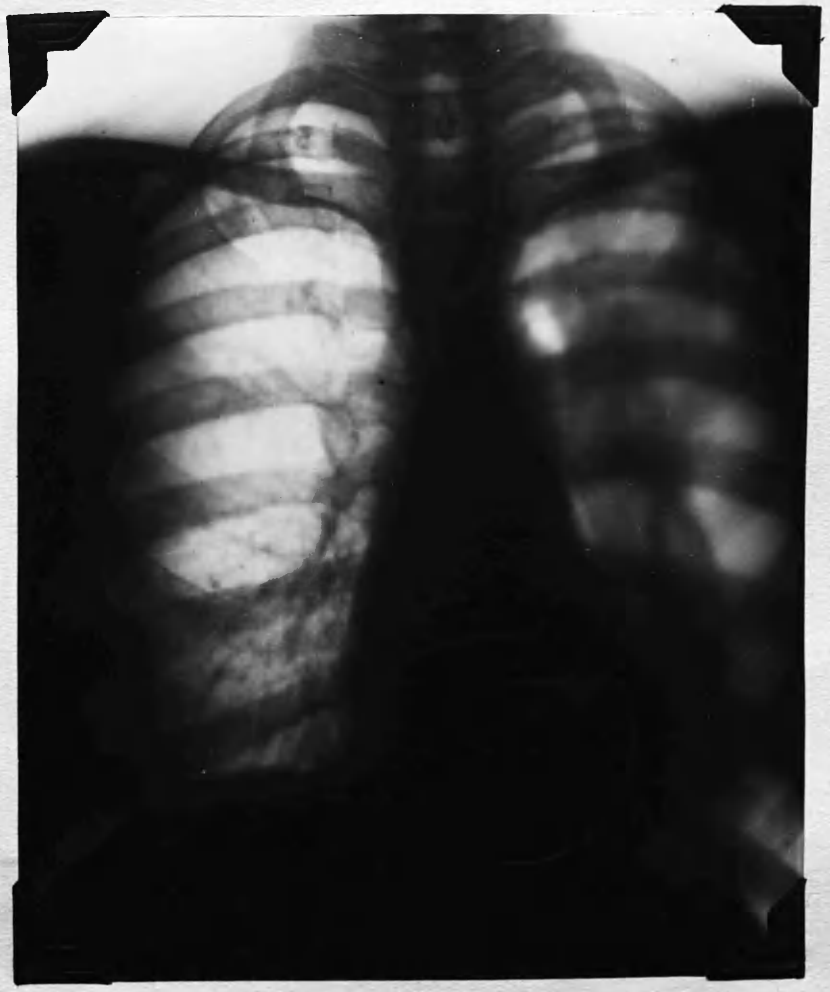
	I.	II.	III.	IV.	V.	VI.	VII.	VIII.	IX.	X.	XI.	XII.	XIII.	XIV.	XV.	XVI.	XVII.	XVIII.	XIX.	XX.	XXI.	XXII.
91	M	23	I	¾	B3	PB	L	P	U	5	300	A	P	B3	W	¼	-	D	-	D	-
92	M	45	H	1	B2	H	L	A	ASC	64	600	P	A	B1	MI	-	-	SUT	B2	S	U
93	M	19	H	I	B3	PBH	L	A	SC	24	500	A	A	-	D	0	-	D	-	D	-
94	M	28	I	7	A	NR	R	A	SP	23	400	P	A	A	MI	2½	A	Q	A	Q	F
95	M	19	I	2	B3	H	L	P	SC	18	500	A	A	-	D	0	-	D	-	D	-
96	M	20	I	1	B2	PB	L	P	ASC	36	600	A	P	-	D	0	-	D	-	D	-
97	M	29	H	5	B3	PB	R	P	ASC	25	500	A	A	B3	W	0	-	D	-	D	-
98	M	28	H	6¼	B2	NRH	L	P	U	10	200	A	A	B2	S	-	-	LSO	-	LSO	-
99	M	20	P	2	B2	NR	L	A	ASC	28	500	A	P	B1	MI	¼	B1	MI	B3	W	U
100	M	36	I	½	B3	PB	R	A	U	14	200	A	A	B3	S	3	B3	S	B3	S	U

APPENDIX C.

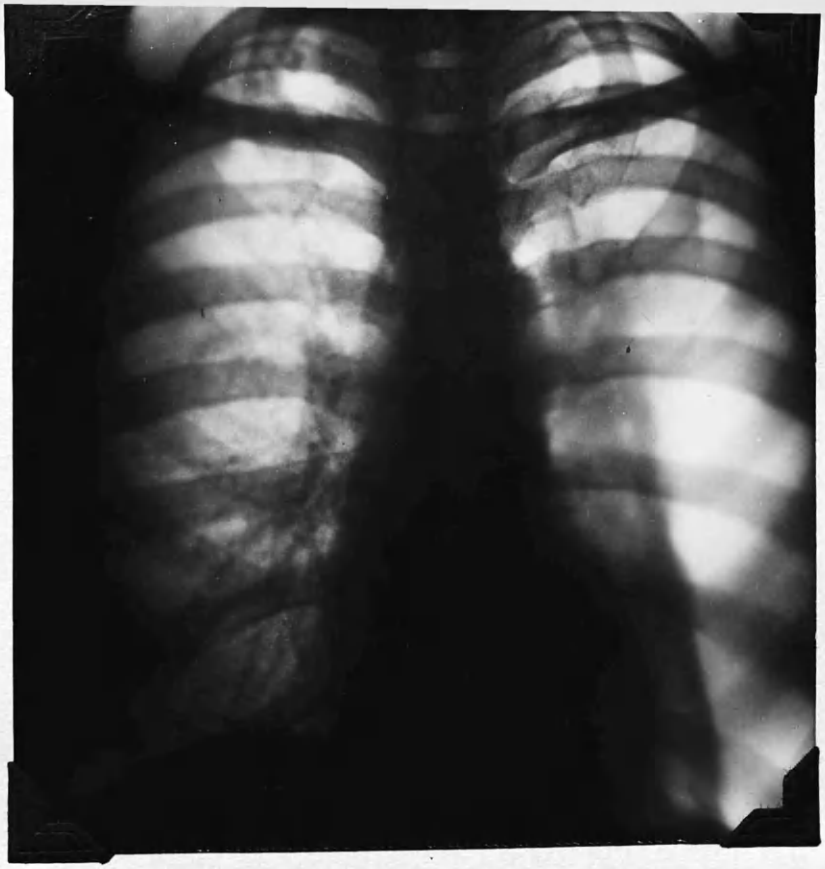
PRINTS OF X-RAY FILMS.



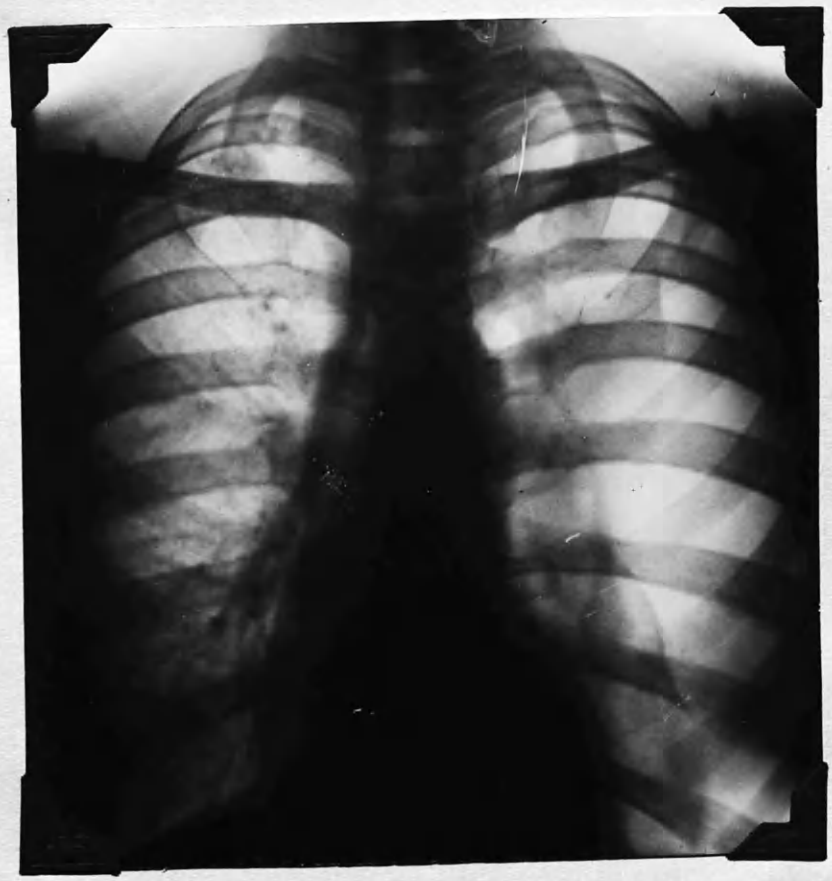
PRINT 1



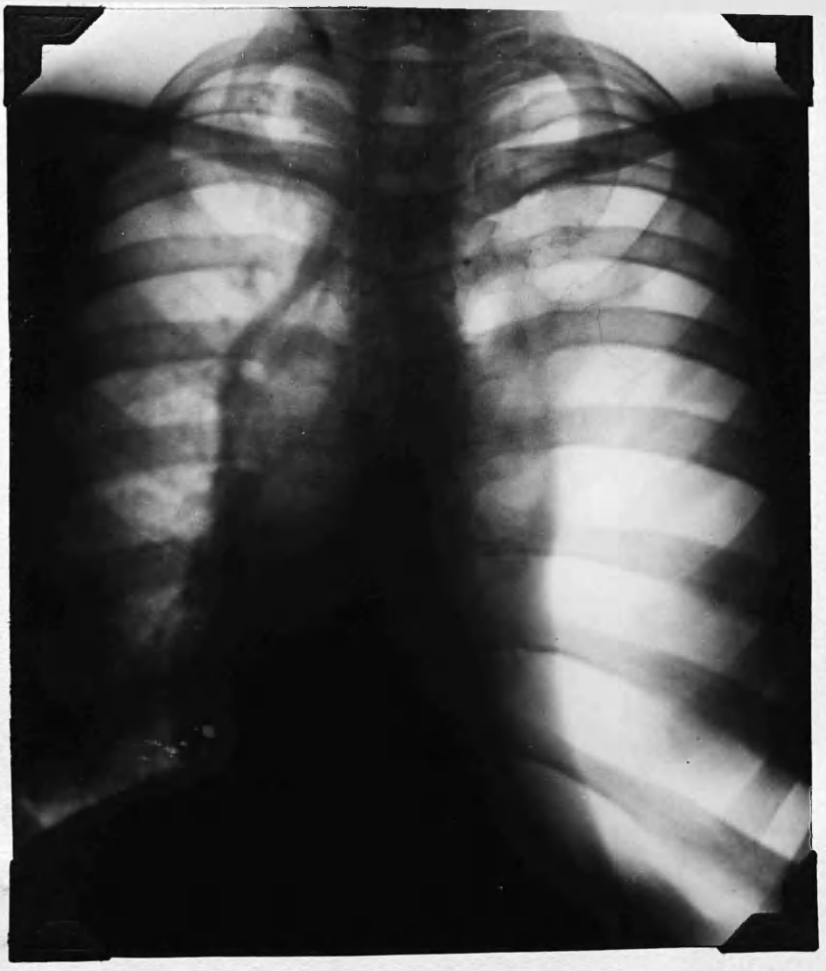
PRINT 2.



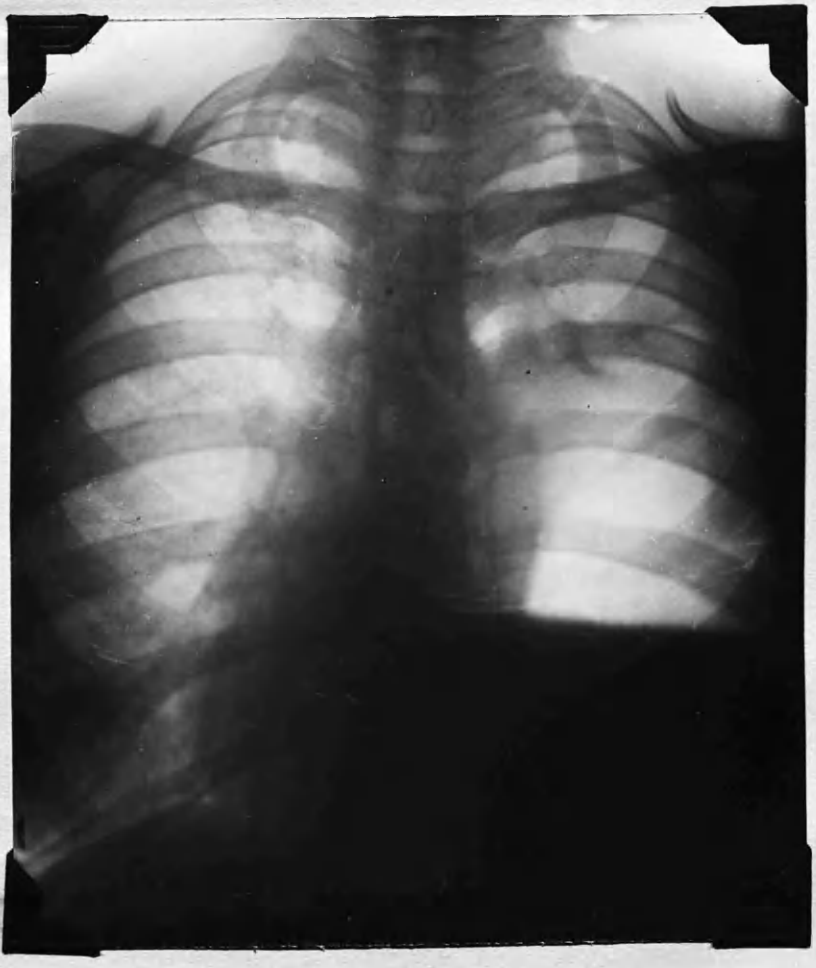
PRINT 3.



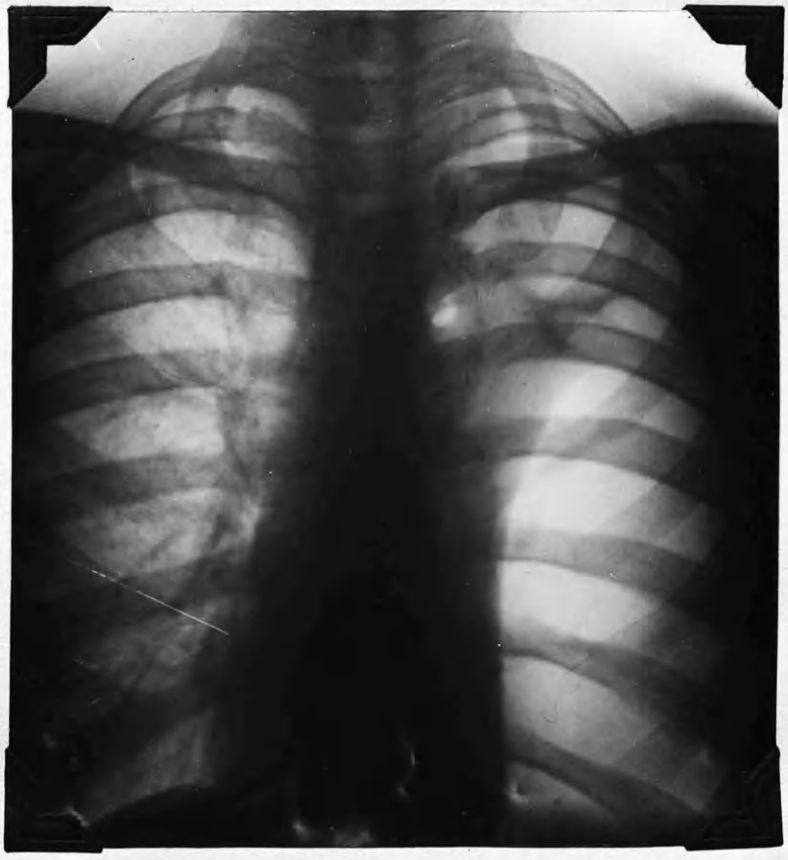
PRINT 4.



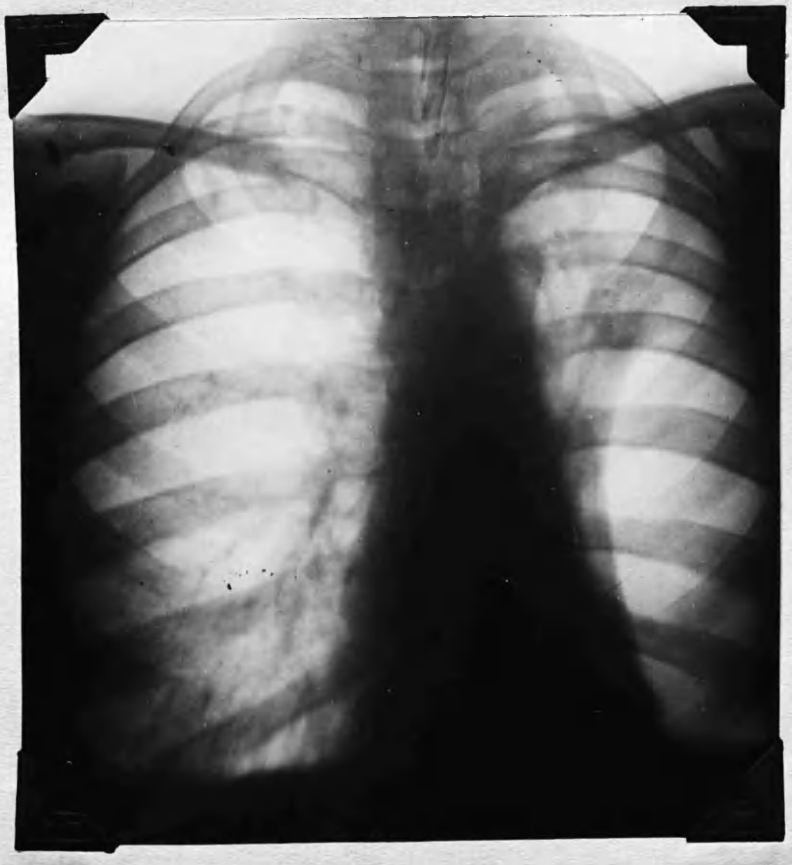
PRINT 5.



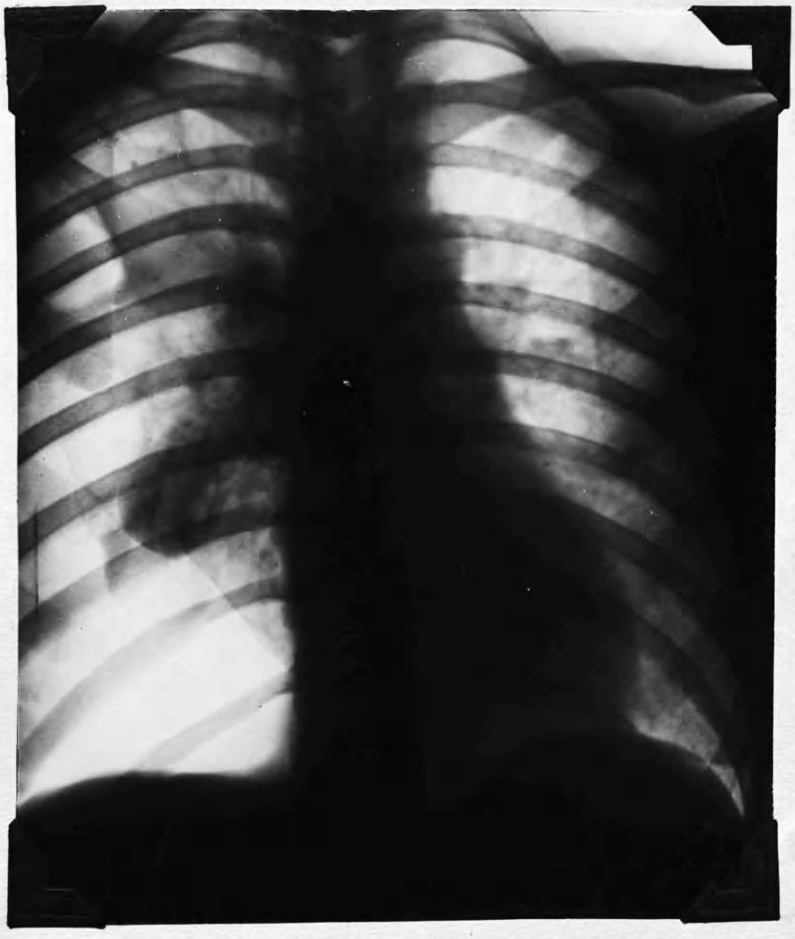
PRINT 6.



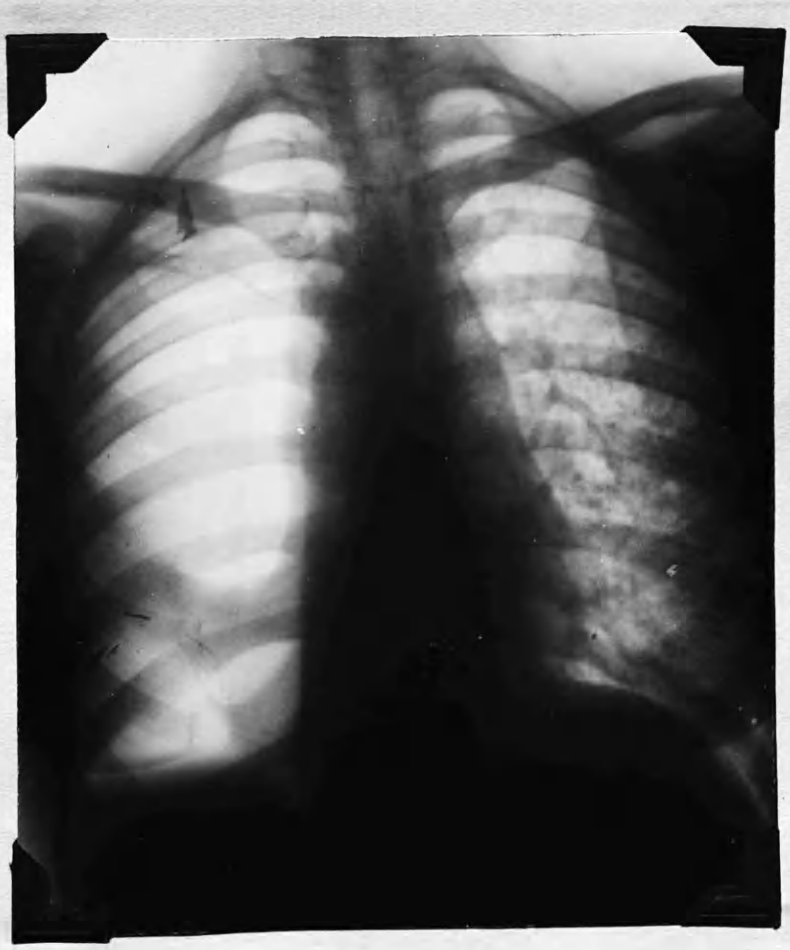
PRINT 7.



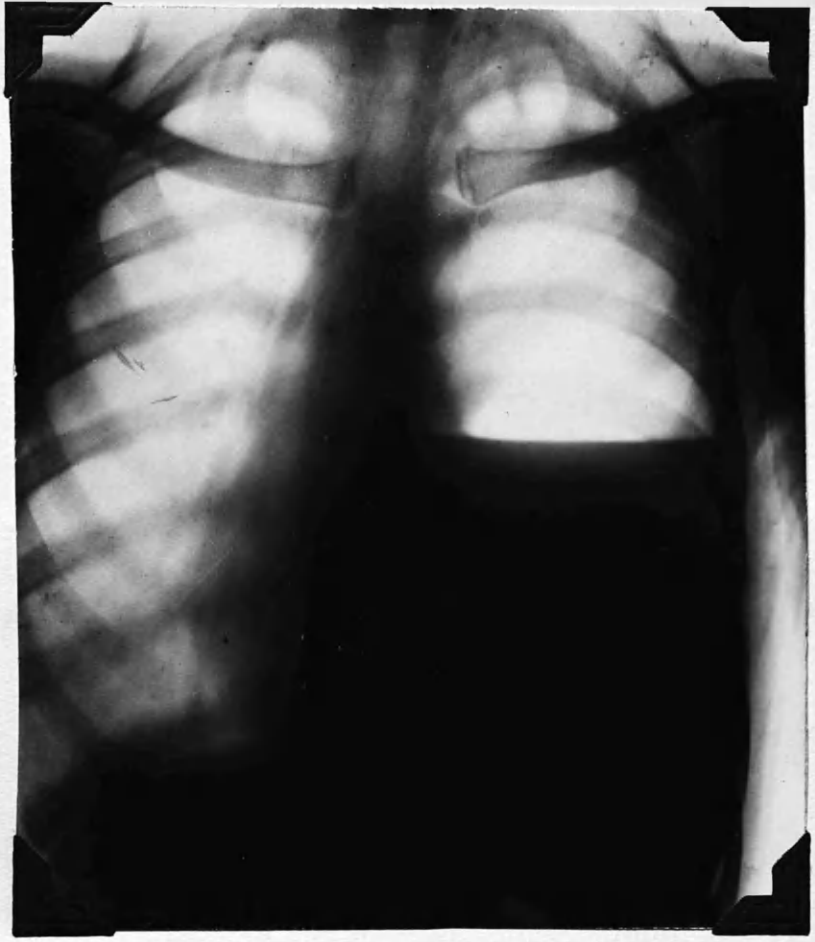
PRINT 8.



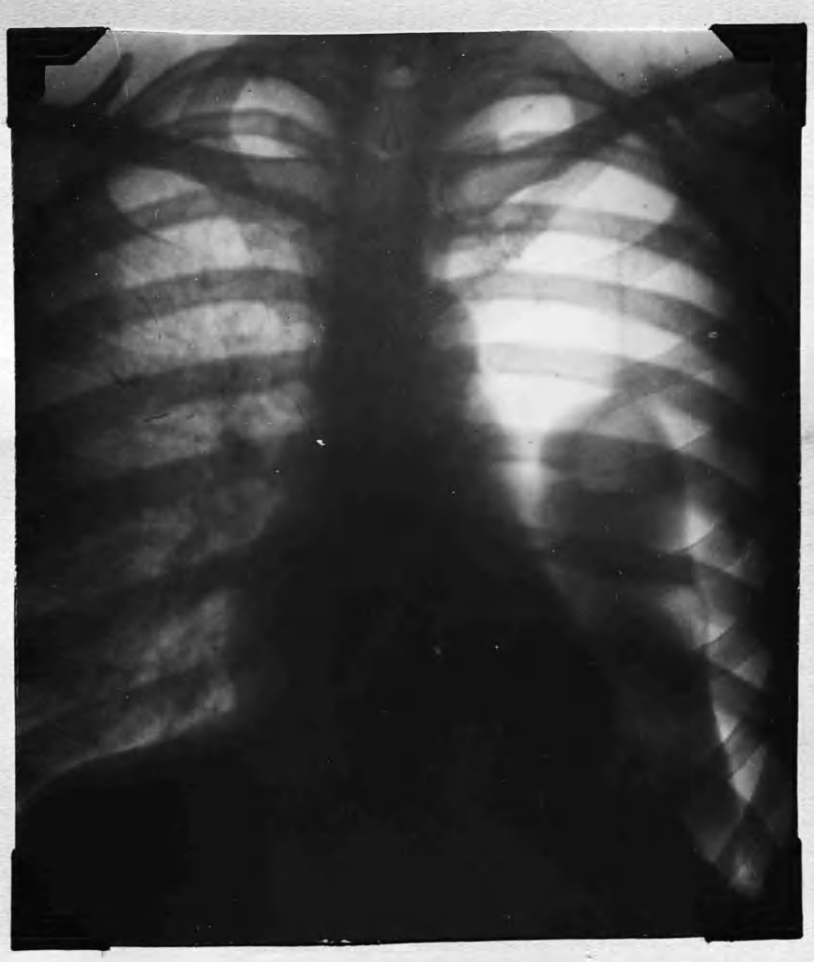
PRINT 9.



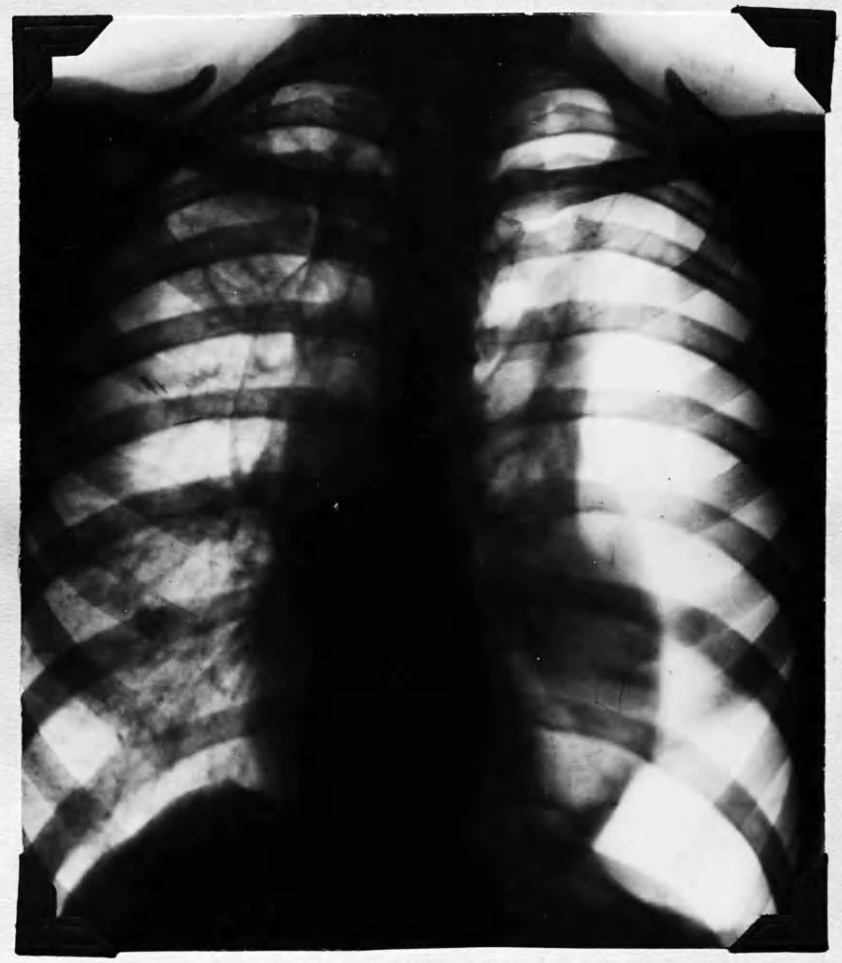
PRINT 10.



PRINT 11.



PRINT 12.



PRINT 13.