ARTIFICIAL PNEUMOTHORAX IN PULMONARY TUBERCULOSIS
ITS THEORY AND PRACTICE

BY

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Owing to the elasticity and continual movement of the Lungs it is one of the most difficult parts in the human body to keep at absolute rest in order to successfully treat any acute process which may be going on there.

Our nearest non-operative approach to immobility of the Lungs is by keeping the patient at rest in bed as in the treatment of Typhoid Fever, when the patient is not allowed to move to perform any function - even the function of taking his food himself being denied him.

As one of the most decided advances in treatment of acute diseases of the Lungs, pride of place must be given to the rest of the affected part which is afforded by the Induction of Artificial Pneumothorax.

HISTORICAL

In his "Essays, Practical and Physiological" James Carson of Liverpool in 1822 first brought forward the Theory of Treatment by this method and showed its practicability by animal experiment. He pleaded that "The diseased part would be placed in a quiescent state and would receive little or no disturbance from the movements of respiration". But his
theory at that time was not put into practice.

Several writers (Houghton in 1832, and Stokes in 1837) wrote reporting remarkable improvement in advanced Pulmonary Tuberculosis in cases in which Spontaneous Pneumothorax had occurred. For interest I am citing a case of this nature (Case of Miss H. No. ).

However, it was not until 1884 that Pneumothorax was first mechanically induced and that was by Potain, who replaced the fluid of a hydro-thorax by sterilised air. Along with two other cases successfully treated, this case was reported in the Bulletin de l'Academie de Medicin in 1888.

With the exception of one or two sporadic attempts - it was not until 1901 that Lemke reported the treatment of 53 cases by this method.

Bremer of Germany did much profitable research into this method of treatment and it is to him that we are indebted for the introduction of the Water Manometer, which is our main criterion as to the amount of gas to be introduced in collapsing a lung, and which is still in use at the present day. These cases were reported in 1905 and 1906.

During the years 1908 to 1912 this method of Treatment has found its way into all enlightened countries, and amongst the pioneers of the movement in England must be mentioned the names of Lillingston (he himself was treated in Norway by Dr Holmboe
in 1909 with such good results that he was able to return to England in 1910 and take up his work again). Vere Pearson, who first used it in his Sanatorium in 1910, and Rhodes who introduced the method in 1911.

One could not close the history of this great advance in Treatment without specially mentioning the name of the late Professor Saugmann of Copenhagen, who did so much to publish the benefits of this operation and to improve its technique.

INDICATIONS and CONTRA-INDICATIONS.

(1). The classical and ideal indication for putting out of action one lung is advancing one-sided disease, with symptoms of toxaemia as indicated by malaise, pyrexia, quickened pulse-rate, night sweats, loss of weight. But how seldom is the much to be desired picture presented, and how often is a "good" lung clinically destitute of physical signs found to be diseased when a radiogram is taken? In a mixed phthisis material, various authorities have put down the number of cases clinically suitable for Pneumothorax treatment at anything between 3% and 10%, but the very large number of unsuitable cases must have passed through a stage in which this treatment might have been available and suitable. As the classical indication is so rare it is usual for us to modify our statement as to unilateral disease and to define
a suitable case as one in which the disease is confined to one side, or if any active disease be present in the "better" side, that it be confined to the immediate neighbourhood of the Hilus, or that it be arrested disease, not involving more than a third of the surface of the "better" lung. In estimating the suitability or otherwise of a case it is not merely sufficient to subject the lung to a careful clinical examination - it is necessary that a careful X-ray examination be made as well: both by the fluorescent screen to estimate the movement of the chest wall and the diaphragm, and by radiogram. It is a very common experience (see case Mrs Pk. No. 15) to have a patient with the symptoms of acute Tuberculosis but with no clinical evidence of that disease either in the form of dullness or crepitations. Subsequently at a later examination signs of crepitation will appear on one side or the other - if on the right generally in the axillary region, and if on the left side, higher up over the praecordia. These will spread till they involve practically the whole surface of one lung, and the symptoms will probably become aggravated. Clinically one would say that this was an ideal case for treatment by Artificial Pneumothorax (one sided advancing disease with no abatement of symptoms and the other lung clinically sound) but on examination of the chest by the X-rays the same process may be found to be proceeding in
the opposite lung only the disease has not reached sufficiently near the surface to be within reach of the stethoscope. What a catastrophe would have occurred if one had induced artificial pneumothorax on account of the clinical signs only?

(2). In contra-distinction to this Hilus type of Pulmonary Tuberculosis is the Apical Type, starting at one or other Apex. Here the signs are apparent practically as soon as the symptoms. In this type the risks of infection of the other lung are very much more marked and the infection both in the original site and in the infected site tend to be very much more acute than in Hilus Tuberculosis. Pneumothorax should be a very much prompter method of Treatment in this type for the reasons herewith appended and I consider if no improvement either in symptoms or signs takes place in three months under the ordinary methods of treatment, that Pneumothorax should be undertaken provided other factors prove no contra-indication. The reasons are:—

(a) More liability to a quicker spread to the opposite lung and as this is generally an acute spread it proves a serious menace to the adoption of Artificial Pneumothorax.

(b) More liability to the formation of adhesions which would thus prevent complete collapse (see later), hence only partially successful results.

(c). The smaller and more circumscribed the area involved, the better are the ultimate results and the more perfect the
functioning power of the re-expanded lung when treatment is stopped.

(3). In cases of any type of Tuberculosis of both lungs, which are progressively getting worse and for which there seems no hope of any improvement, it is sometimes expedient to induce Artificial Pneumothorax on the worse side in the hope that it will lead to a certain amount of abatement of symptoms and so render the patient's life more comfortable. Should the disease in the other lung be moderate in extent and intensity, a betterment of its physical condition has often been observed when this has been done, probably due to one of two factors (a) lessening of toxic absorption and thereby improving the patient's general condition with a rise in his powers of resistance (b) increased blood supply to that "better" lung. Case of Miss H. No. 25, is of great interest in this respect. It was a case of extensive double sided disease with grave toxaemia and very acute spread. One dare not think of inducing an Artificial Pneumothorax but this patient's condition was much improved by a Spontaneous Pneumothorax occurring - in fact she was so much improved that she left our charge thus preventing the collapse being continued.

(4). The acute Broncho-pneumonic types of one-sided disease are suitable for Pneumothorax (see Case H.S. No. ). Caseous pneumonic cases are not satisfactory, but certain operators have reported successful results, notably Riviere. I have no cases of this
type so cannot speak from my own experience, but various writers speak of its unsuitability from two factors (a) the extreme liability of the caseous material being sucked into the other lung and thus causing an infection there, and (b) mechanic reasons: the solid pneumonic patch ill adapts the lung to compression.

(5). An important indication for Pneumothorax is the presence of haemoptysis, whether this be a single large haemoptysis or of a recurrent type, no matter the smallness of the quantity. But haemoptysis is purely a symptom of the disease and may be due to various causes and may occur at any stage. It is a symptom which would decide one to do an Artificial Pneumothorax when there was perhaps some doubt before, whether the doubt arose on the extent of the lesion, the comparative absence of toxic manifestations or on any other account. Haemoptysis when it occurs in the early stages would have simply to be regarded as a symptom and would have to be put in the balance along with the other symptoms when one was thinking of the advisability of compression. But given two cases of equal extent and severity, if recurrent haemoptysis were a symptom of one and not of the other it would be a deciding factor in my mind in favour of Pneumothorax in the former case, although I might decide against it in the latter. In the later stages of the disease when changes have taken
place in the vessel wall and when severe haemoptysis occurs either from rupture of an aneurism of the pulmonary arteries or from ulceration through the wall of an artery, Pneumothorax becomes an operation of emergency and immediate collapse must be obtained to prevent a haemorrhage which might otherwise be uncontrollable. But in such a case it is so often that there has been old-standing changes in the pleura and that a space is impossible to find. The only case in which artificial Pneumothorax was carried out for large late haemorrhage in my practice proved fatal though entrance was obtained. (Case of S. Group I) (6). Artificial Pneumothorax should be carried out in all cases of tubercular pleurisy with effusion when it becomes necessary through symptoms of discomfort to withdraw the fluid. Withdrawal of fluid and introduction of gas should be carried out at the same time for the following reasons. (A). It permits the withdrawal of fluid without pain or discomfort and without the attendant dangers of an ordinary aspiration. (B). After withdrawal of fluid it permits of a complete radiological examination being made and allows one to decide whether the Tuberculosis is purely pleural or whether there is (as in the vast majority of cases) intra-pulmonary disease. (C). It prevents the pleurae becoming adherent so that if intra-pulmonary disease is found and treatment by compression is decided upon, one does not meet with the disappoint-
ment of finding no pleural space which would probably have occurred if absorption had been allowed to take place. (D). Oxygen is the gas of replacement of fluid as, by virtue of its chemical properties it tends to prevent the formation of more fluid. To my mind this is one of the most useful indications for Artificial Pneumothorax because one so often finds in the history of cases of active Pulmonary Tuberculosis which come for treatment that the primary manifestation has been pleurisy with effusion which has been aspirated or allowed to absorb and after which an ordinary convalescence has been carried out, only to find that in after years, it may be few or many, that the patient has developed active Tuberculosis in the same lung and any hope of treatment by compression of the lung has gone owing to the adhesions which have formed between the parietal and visceral layers of pleurae. Of course the primary fault lies in the fact that the point is not sufficiently emphasised in our Medical Schools that these cases of Hydro-thorax are in the big percentage of cases of tubercular origin, and until that point is sufficiently emphasised we shall continue to have the same catastrophes. Better far would it be to teach that the fluid, which would act as a compressor of the lung, should be left or only drawn off in small quantities if signs of oppression appeared, rather than teaching immediate aspiration as is done. Besides I am convinced that
the serous fluid of an ordinary tubercular Hydro-Thorax has a distinct therapeutic value as well as its mechanical value, but this does not fall within the scope of this paper.

(7). Persistent pleuritic pain is an indication for a partial Pneumothorax (Case Miss E.D. No.21 and Mrs A. No. 23). In these cases complete collapse is not aimed at, only sufficient gas being used to separate the two layers of pleura, nor is it necessary to carry on the treatment for the same length of time as in intra-pulmonary disease.

(8). I shall not here give the indications for Pneumothorax in other conditions which are not tubercular.

CONTRA-INDICATIONS.

(a). Acute disease of any extent in the "better" lung, or the involvement of one third of the "better" lung by arrested disease except in special cases (vide supra).

(b). Extensive tubercular disease of other organs, kidney, bowels, etc.. I do not consider tubercular laryngitis to be a contra-indication, in fact it is usually improved by Pneumothorax.

(c). The presence of grave constitutional disease such as diabetes, chronic nephritis etc.

(d). Apprehensive nervous patients generally do badly.

(e). Organic heart disease.
APPARATUS REQUIRED FOR ARTIFICIAL PNEUMOTHORAX.

The essentials of a Pneumothorax apparatus are exceedingly simple. They consist of a needle for introducing into the chest wall, connected by rubber tubing to a bottle graduated into hundred c.c. spaces containing the gas which it is desired to introduce. Between the needle and the gas container is a manometer which registers the intra-pleural pressure; connected to the gas bottle by rubber tubing is a pressure bottle which can be heightened or lowered at will to alter the pressure and thus drive out the gas contained in the gas bottle into the lung. These are the first essentials and the apparatus which most nearly conforms to this principle in the simplest way is the one devised by Lillingstone and Vere Pearson. It is the only one I have used personally and I have found it entirely satisfactory in every way. The whole apparatus is contained in a cabinet on the inside of the door of which is fixed the water manometer. The ends of the manometer limbs are bulbous, so that if the patient coughs, thus raising the intra-pleural pressure, water will not be so readily expelled from the tubes nor if any abnormal negative intra-pleural pressure be encountered will the water be so readily sucked into the patient's chest. The manometer consists of two straight pieces of glass tubing about 40 cm. long connected at the foot by a short length of rubber
tubing of the same diameter. Between these tubes a scale ruled to \( \frac{1}{2} \) cm is fixed to the door, each division of \( \frac{1}{2} \) cm. is numbered 1.2.3.4. up to 15

DIAGRAM of PEARSON-LILLINGSTON APPARATUS.

The Lillingston-Pearson apparatus consists of two bottles, A. and B. A, the gas bottle is graduated from 100 to 1100 c.c.s. Both bottles are fitted with rubber stoppers to admit glass tubing. A glass tube C passes to the foot of bottle A, and a similar tube D to the bottom of bottle B. These two tubes are connected by a rubber tube E, fitted with a stopper M. Short glass tubes F and G opening to outside air in Bottle B, and connected to the rubber tubing leading to the manometer and to the chest through bottle A. If bottle B and the tubes D,E,C. be now filled with water, the water will flow into bottle A by syphonage until the water is at the same level in both bottles. Air to replace the water will enter by tube F and be
forced out of tube G. Tube G is connected by rubber tubing H to a T-shaped piece of glass tubing 1. One limb of 1 is connected to the manometer by a rubber tube, while the other limb is connected by rubber tubing K to the needle. L and M are metal clamps, one situated between bottles A and B, and one between bottle A and the T-piece 1. Bottle A is now filled with hot water slightly coloured with lysol or weak carbolic solution. The rubber tubing is removed from G. If Oxygen is the gas chosen to introduce into the chest, it is stored in cylinders and the rubber tubing of the cylinder is connected to G, clip M removed and the gas cylinder opened and the pressure there forces the solution in A into the tube C.E.D. and thence into bottle B, so that A becomes full of Oxygen. When the gas goes to mark O in A, the cylinder is shut off and clip M replaced. The bottles are now ready for use. The same method applies to Nitrogen.

If Air is going to be the gas used, the fluid in A is withdrawn into B by syphonage, and Air replaces it. Instead of sucking the Air directly into A, it is our custom to draw it through a bottle containing a weak solution of lysol thus purifying it. An ordinary bottle with a rubber stopper with two holes is used. Into these holes a short length of glass tubing is introduced and this is connected to A by a rubber tube. A long length of glass tubing reaching to the bottom of the antiseptic fluid is also introduced,
with the other limb open to the outside air. The air is thus sucked into the fluid and it bubbles through this and out of the other tube in G and then fills bottle A.

Bottle A is now raised on blocks so that the level of the fluids in A and B are equal or even so that the fluid in B is lower than in A in order that the gas will not be driven into the pleura with its negative pressure and thus cause embolism. The needle is now introduced into the pleura with its clamp L closed. A negative pressure will be noted by the fluid in Limb M being sucked up, a positive pressure by its being pushed down. P is a glass filter filled with cotton wool between the needle and J; and Q is a glass rod in the rubber tubing near the needle so that blood or fluid can be easily seen, supposing there should be fluid in the pleural cavity or a vein should have been pierced. As soon as a negative pressure is registered the rubber tube H is connected to G and clamps L and M removed so that the Air is allowed to flow slowly into the chest. As the pressure rises in the chest, bottle B can be raised on the wooden blocks R to increase the pressure there and so drive the Air on into the chest. The fluid in the manometer may be coloured so as to record the press-
ure more easily. Methylene blue, red ink etc. may be used for this purpose.

OTHER APPARATUS IN COMMON USE.

Dr Parry Morgan (Lancet ii, 90, 1914) has described an apparatus with two manometers so that the intra-pleural pressure and the pressure in the gas bottle can be taken at the same time. There is a lot to be said for the double manometer as when the gas is flowing it is only the pressure in the gas bottle which is recorded with slight respiratory fluctuations owing to discrepancy in size between the rubber tubing and the bore of needle, but it is only a question of a second to pinch the tube with the fingers to record the intra-pleural pressure. To see that the gas is flowing is only a question of watching the level of the fluid in the gas bottle. The double manometer overcomes these slight objections.

Morriston Davies (Surgery of Lungs and Pleura, 1919, page 16b) has a more elaborate apparatus which consists of two graduated cylinders, one for Oxygen and one for Nitrogen. The gas from either of these cylinders is forced into the chest by water pressure, the rate of flow being graduated by a tap above the water cylinder.

Woodcock (B.M.J. ii, 1055, 1913) uses an apparatus in which he generates his own Nitrogen and the gas and pressure bottles move up and down on rods. Lister uses a similar apparatus with the difference
that Oxygen can also be generated. All these above are examples of the movable bottle apparatus, but on the Continent various apparatus with fixed bottles are used, but they are very cumbersome and not to be recommended.

**MANOMETER.**

It is necessary here to describe the manometer. The calibre of the manometer tubing is of some importance because if it be too large the more would be the discrepancy between the bore of the needle used and the manometer limbs so that there would be less response in the column of fluid and the finer fluctuations would not be shown. It is highly important that the fluctuations of the manometer should be absolutely definite because it is the clearest evidence that the needle is in the pleural cavity and not in the lungs. Saugmann recommends a bore of 3 millemeters and that is quite satisfactory - anything from 3 to 5 Mm. can be used. Above that size the readings become sluggish and indefinite and difficult of interpretation. The water manometer is the one in general use but some writers describe their pressure in terms of MM of Mercury, and they do this by marking on the scale alongside the water scale the equivalents in MM of Mercury. Again, some operators have affixed a Mercurial manometer as well so that especially low or especially high pressures may be recorded, but I have never seen a lower pressure than -30 recorded nor a
higher than +30 MM of water brought about so that the water manometer graduated from 30 cm to 35 cm. fulfils all requirements. Of course it is quite possible that a lower pressure than -30 might be recorded, in which case the fluid might be sucked into the chest with disastrous consequences, but there are two factors in the formation of the Lillingston apparatus which would tend to prevent that accident, or at least cause it to be effected more slowly so that one could either pull out the needle or grasp the tube. The presence of the bulbous ends before described and secondly the filter which is placed between the limb of the manometer and the needle are the two factors which tend to prevent this accident because the water would firstly be caught in the bulbous ends and would then only pass through the filter slowly. The tubing between the manometer and the chest must be kept unclosed all the time the operation is in progress so that the pressure may be recorded at any given moment. Readings should be taken frequently so that an undue pressure would not be given.

CHOICE of GAS TO BE USED.

(1). At the Primary Induction. The three gases which are accepted now as suitable for Artificial Pneumothorax are Oxygen, Nitrogen and Air, and it is our practice to give Oxygen for the induction, and for five or six refills afterwards. I do not think
that it possesses any advantage over the other two
gasses, although some writers say that it is safer in
case the needle should pierce a vessel wall and there-
by produce a gas embolism, in which case the Oxygen
would be more readily absorbed by the blood and thus
diminish the serious sequelae of that accident. That
would probably be so if the wound were in a systemic
vein (as in the chest wall) where the veins contain
venous blood and would readily absorb Oxygen; but this
accident must be extremely rare. The most probable
source of embolism is from the wounding of a pulmonary
vein, and as this vein contains already blood saturat-
ed with Oxygen further absorption could not take place,
and therefore embolism would take place equally with
Oxygen as with Air or Nitrogen. I personally have
never seen a case of embolism and I think that if the
other precautions be rigorously observed it is a very
rare occurrence.

REFILLS.

Oxygen may be ruled out of court for the main
reason that it is much more quickly absorbed than Air
or Nitrogen, therefore shortening the intervals for
refilling and thus causing more inconvenience to the
patient, as one could imagine the operation to be any-
thing but a pleasant one - it is also expensive. The
most generally used gas for refills is Nitrogen as it
is inert and its absorption is slow. But I do not
think that that minor advantage justifies the expense
and trouble associated with its use as, in my experience, the intervals for refills is not appreciably lessened if Air be used and I have found that Air fulfills every requirement and it does not necessitate the use of the cumbersome cylinders in which Nitrogen is supplied. It is free to all, and as I said before it only causes a negligible diminution in the interval of refills.

In this connection one must say a few words on some experiments which have been carried out to show the effects of the introduction of these various gases and the chemical changes which have taken place in the composition of them after their introduction into the pleural cavity by interchange with the gases in the blood.

In 1823 John Davy conducted some experiments on the absorption of gases after they had been introduced into the pleural cavity of dogs. (Philosop.Trans 1823) and he made it clear that their composition was very quickly altered. In a case of Pyo-Pneumothorax by Morriston Davies, when pus was withdrawn from the pleural cavity and replaced by Oxygen, the analysis of the pleural content was $O_2$ 21.56%, $N_2$ 74.09%, $CO_2$ 4.35%, after eight days before Oxygen displacement took place the Oxygen was practically absorbed - the analysis showing $N_2$ 98.13%, $O_2$ 0.69%, $CO_2$ 1.18% (Morriston-Davies, Surgery of the Lungs and Pleura, 1918, p.54) Gilbert and James, Havens Webb, (Archives of Int.Medicine 1914) found that within 24 to 48 hours in cases
filled with $N_2$, $O_2$, and $CO_2$ were then present and that with Air a similar result occurred. The authors argued that owing to the rapid diffusion of gases, that Nitrogen possessed no advantages over Air.

For all practical purposes one finds that the interval for refills is not appreciably diminished when Air is used instead of $N_2$ and Air, as is shown by the following two cases when both Air and $N_2$ were used. (Case of MacS. No. 5 and Case of R.L.T. No. 6).

**NEEDLES.**

Again a difference arises as to whether the operation is an initial one or whether it is a refill after Pneumothorax has been induced. In the former case there is only a potential space and damage to the lung much more likely than in the latter case where there is already a space of greater or lesser size and therefore not so much likelihood of puncturing the lung.

Riviere recommends a needle made like a trocar and cannula - the cannula being 1.8 MM guage, and it has a side-opening about 1/16th on an inch from the tip and graduated in centimeters with a sliding guide to regulate the distance it has to be forced into the chest wall. The trocar and cannula are thrust into the side and through the underlying tissues till one imagines one is near the parietal pleura (about 1 Cm. is sufficient). The trocar is then withdrawn and the cock closed, and the cannula which has a sharp edge is pushed in through the parietal pleura. I have used
this form of needle and have found it quite satisfactory, but it is a little cumbersome and causes more inconvenience to the patient.

Another needle in frequent use is a simple hollow one with a terminal oblique aperture, but I have found that these needles become blocked by the small piece of epidermis which was cut out when penetrating the skin, and this was often difficult to remove by the stilette. Besides, the danger of infecting the pleural cavity by pushing this epidermis into it is quite considerable so I have given up using this form of needle, and I now use Riviere's trocar and cannula for the initial operation and for the refills a hollow needle with a lateral aperture and the end cut obliquely and filled up, and I have never found that any appreciable damage to the lung was done and it certainly causes very much less inconvenience to the patient. I find also that the accidental blocking of the needle with blood or epidermis is of very much less frequent occurrence. The latter needle is one introduced by Lillingston and I always use one which screws on to an obturator with a stop-cock so that if by chance the stilette requires to be passed to clear it, the pleural cavity is protected from the outer air. Saugmann used the same needle but with a terminal opening as well as a lateral.
Diagrams of the needles referred to.

1. Riviere's needle.  2. Lillingston's needle.

PREPARATION of PATIENT.

It is our custom to administer a cathartic the night before and to give a light meal in the morning before operating – especially in the initial operation. When collapse has been established a patient can be done at any time and is able to proceed home within half-an-hour.

One hour before the initial operation ½ c.c. of omnopon scopolomine is administered. This diminishes the anxiety and sensibility of the patient and quietens the breathing. If a patient be especially nervous I often give a bromide mixture for a few days before.

The patient is laid on the sound side with one or two pillows underneath the chest so as to arch it and widen the inter-costal spaces. The head is kept low. The site of puncture is washed with spirit and coated over a good area with Tincture of Iodine. A sterile hypo-dermic syringe containing a 2% solution of Novo-caine is next used and the track of the puncture is anaesthetised and a few seconds are allowed to elapse before introducing the Pneumothorax needle.
waiting, the Pneumothorax needle which has been stand-
ing in alcohol, should be thoroughly dried by passing
it through a spirit flame and then by blowing air
air through it into the flame. Pituitrin and brandy
should always be kept in readiness in case of collapse.

SITE of PUNCTURE.

The most suitable site for puncture should be
declared upon sometime before the operation so as to
avoid delay when the time arrives. Other things
being equal, the axillary region is the most suitable
for the following reasons. (A). At this point the
respiratory excursion is greatest therefore it is the
least likely part in which to find adhesions. (B). The
basal movements are much freer than the apical so that
the lung surface is more likely to be free from adhes-
ions. (C) The intercostal spaces are at their widest
here, and the chest-wall has its thinnest muscular
covering in this region.

A great deal of course depends on the site of the
original disease, and if the disease be largely basal
it might be expedient to puncture in the 2nd or 3rd
intercostal space outside the mammary line. The point
furthest away from the original site of disease and
where the signs are least intense is the best site of
puncture, but in 75% of cases the 6th interspace in
the mid-axillary line will be found to be the best
and if one does not get in there the probabilities
are that the Pneumothorax will be unsuccessful. A
rigid rule should be observed and that is never to puncture over the site of cavities, firstly because there will probably be too strong adhesions, and secondly because, if the cavity wall be injured a Pyo-Pneumothorax might be produced.

**THE OPERATION.**

After anaesthetising the patient and the Pneumothorax needle being thoroughly dry and connected to the manometer tubing, it is passed through the intercostal space swiftly and firmly. If the trocar and cannula is used, we pass it as far as the guide, then having withdrawn the trocar, the cannula is pushed slowly forward by steady careful pressure and strict watch is kept on the manometer to note any fluctuations of it. One often feels a distinct 'snap' at the moment the parietal pleura is reached and this can be used as a guide to indicate that one is in the pleural cavity. However, this is not always noted as a 'snap' may be felt by piercing the innermost fascia of the intercostal muscle, so that it is only by watching the manometer that one can be sure of being in the pleura. If the ordinary sharp needle is used the same procedure is adopted - except for the manipulation of the trocar.

When the pleura is reached a sharp response is noted in the fluid level, the pressure going down to -8 or -10 as an average, with a respiratory fluctuation
of from 4 to 6 cm. Such a response is clear evidence that one is in the pleural cavity. One can now introduce the gas, and at the initial operation this must be done very slowly at a negative pressure. This is done by keeping the level of the fluid in the pressure bottle below that of the fluid in the gas bottle, so that the gas is more or less sucked in by the higher negative pressure of the chest, and this reduces the risk of gas embolism. As soon as gas enters the pleural cavity, indicated by the rise of the level of the fluid in the gas bottle, the risk of embolism is completely past, and the gas may be allowed to enter at ordinary atmospheric pressure. One does not always meet with the same easy task and it may be only after several attempts that a free pleural space is found. One may feel the 'snap' as the needle passes through the parietal pleura and find no manometric response. This may be due to several causes. (1) The most frequent cause will be the blocking of the needle with blood or solid tissue from the chest wall. The first thing to do then is to pass the trocar through the needle when trocar and cannula are being used, taking care not to pass it to the hilt. When the ordinary sharp-pointed needle is being used a stilette is passed and if failure of manometric response has been due to that cause fluctuations should be indicated immediately on withdrawing the trocar or stilette as the case may be. (2). Two pleural layers being adherent may be
another cause, and, if after the stilette's removal fails to elicit response, then there is no other course open but to remove the needle and and make a fresh attempt. Before removing the needle, however, a little gentle manipulation may be allowed.

Suppose one did pass on through the visceral pleura, what would one find? (a). One would get manometric response and fluctuations of from 2 to 4 cms, but these would be about atmospheric pressure and would not show the decided negative pressure of the pleural fluctuations, and one would never proceed further with a response of that kind. (b). Instead of entering the lung parenchyma proper one might push the needle into a bloodvessel when one would also get slight fluctuations but they would soon stop by the needle becoming blocked with blood clot. In the case of a vein gas might be sucked into it and thus produce embolism.

INCOMPLETE RESPONSE.
(2) The second difficulty may be that one gets a negative pressure but with a less response than from 3 to 4 cms. This may be due to not very dense adhesions, but one can only withdraw the needle and try elsewhere because the evidence that one is in the pleural cavity is not sufficient.
(3). One may get a larger negative response with no fluctuations. The fact of the negative response points to one being in the pleural cavity and the cause of the
fluctuations being absent is practically certain to be due to blocking of the needle, which will be cleared by the stilette. However, I have seen cases when actual blocking has not taken place and this may have been due to the blocking of the needle during expiration by the visceral pleura coming against it. and This is an argument in favour of the double manometer.

AMOUNT OF GAS TO BE INTRODUCED.

At the first operation I never exceed 300 ccs of gas and I never bring the pressure beyond a distinctly negative one. I say this advisedly because 300 ccs would never bring the pressure up to more than 2 to 4 but in a pleural pocket cms in a completely free pleural cavity/in which one might get a distinctly negative pressure on entering 300 ccs might bring the pressure to a very high positive one, depending of course on the size of that pocket. (Case No. 24 ). I consider it far better to give up that site of entering and to try elsewhere, rather than to try and break down the adhesions by effecting a high pressure. My general procedure is to give 200 to 300 ccs of gas and to repeat this next day or the day after - depending on the amount of reaction. There is no doubt in my mind that since smaller quantities have been used and have been slowly introduced, that far fewer of the distressing results of Artificial Pneumothorax have been seen. In the days when it was customary to introduce 1000 ccs of gas at the primary operation, one often saw cases of great distress, much
displacement of the heart, dyspnoea, fever and sweatings, and these reactions often took a long time to settle down, thus preventing the continuance of the treatment for some time: and there was also a very real danger of affecting the other lung by literally squeezing out the secretions of the diseased lung into the bronchi in such large quantities that they could not immediately be got rid of by coughing. The only time in which a large amount of gas could be justified in the light of recent experience is in the case of Pneumothorax induced for hemoptysis. In our practice the first filling, and a few subsequent ones are given in the patient's bedroom so as to ensure as little movement as possible.

PLEURAL ADHESIONS.

From the foregoing it will be seen what an important part is played by pleural adhesions. (1) They may be so great as to absolutely frustrate any attempt to induce pneumothorax. (2) In lesser degree they may prevent the complete collapse of the lung, and I shall later cite some cases of this nature which we call Partial Pneumothorax. (3) In still lesser degree they may be present in bands inside the chest wall and thus produce irregularities in the collapse of the lung. In these cases the therapeutic value of Pneumothorax is nearly as good as in a complete Pneumothorax, because in time these bands become stretched by the continued pull on them by the gradually increasing intra-pulmon-
Skiagram showing partial Pneumothorax.
ary pressure. (4). It is only in the complete absence of adhesions that one is able to obtain what is known as Complete Pneumothorax.

Failure to obtain a free space at all occurs in a varied percentage of cases according to the various writers, but this discrepancy of numbers is easily understandable when one considers the different class of cases selected for treatment. When old-standing cases or only acutely ill cases are chosen the percentage of failure must be higher than in early cases or in cases of peribronchial spread (already quoted in my Indications for Pneumothorax) and I think that early cases will become more and more often chosen as the efficacy of this treatment becomes more widely known. Of course in a private Sanatorium one's percentage of failures should be much less than in a public institution where so often Sanatorium treatment is only given to very advanced cases. In my series of cases extending over five years there have been only four in which entrance was unobtainable, giving a percentage of . Saugmann gives his percentage at 11%, Zine 24%, Hamman & Sloan 15%, Keller 12.5%, Riviere 20%.

PARTIAL PNEUMOTHORAX.

Although adhesions may prevent complete collapse of the lung, yet sufficient collapse may be obtained of part of it to have a distinct therapeutic effect making it worth while to continue the treatment. This is known as Effectual Partial Pneumothorax, and it is
Skiagrams showing two different types of complete collapse.
only by practical results in the abatement of toxic effects that one can judge whether a Partial Pneumothorax is going to be effectual or not, as the result depends so much on the strength or weakness of the adhesions. A very inconsiderable pocket may become in course of time sufficiently large by the stretching of the adhesions to be of distinct therapeutic value. Higher pressures, provided one keeps an eye on the mediastinum for fear of too great displacement, are justified in these cases, in the hope that these adhesions may break or stretch sufficiently. Operative measures are now in vogue to cut adhesions through a Thoracoscope by galvano-cautery, but a consideration of this is outside the scope of this paper.

When stretching of adhesions fails to be produced and a small Pneumothorax giving no abatement of the signs of symptoms results, then that Pneumothorax is known as an Ineffectual Partial Pneumothorax. It is only by persevering for some time that will show us which Partial Pneumothorax is going to prove Effectual or Ineffectual.

COMPLETE PNEUMOTHORAX.

Complete Pneumothorax requires nothing further to be said.

DIAGNOSIS OF ADHESIONS.

Since adhesions prove to be such a stumbling-block to this treatment, it is only fair to the patient that before holding out the hope of being able
to treat the case by Pneumothorax, one should make an effort to come to some conclusion with regard to the presence or absence of adherent pleura. This is a matter of extreme difficulty, as in cases where one might have expected to find completely adherent pleura one finds a free space and vice versa. The points which might be helpful are. (1) The extent and duration of the disease. The more localised the disease the more likelihood there will be of getting a free space at least sufficient to produce a therapeutically valuable Pneumothorax. This is a strong argument in favour of early Pneumothorax in cases where other methods have failed to arrest the progress of the disease. (2). A history of pleurisy, especially if the patient's history is corroborated by dullness in percussion, weakened breath sounds and a basal shadow as revealed by X-rays. (3). The presence of large superficial cavities generally means thickening and adhesion of the pleura. But the thickening may be confined to the visceral pleura and there may be no adhesions between it and the parietal pleura. (4) The presence of a free or slightly restricted Tidal expansion contra-indicates the presence of adhesions, but on the other hand diminished or absent Tidal expansion may mean intra-pulmonary changes leading to diminished expansion of the lung itself.

REFILLS.

Interval after the initial operation. The next refill should be given the day after or at least not
not later than two days after, and the same precautions i.e. entering the gas at a negative pressure and stopping before the pressure becomes positive, must be observed. Our practice is to allow 100 c.cs more gas to be introduced than at the primary operation. The third refill is given on the fifth day, and a day may be added to the interval of each refill. At each refill the amount of gas introduced is increased and the pressure gradually raised till collapse is more or less obtained. The intervals then must be judged separately in each individual case and the judge the optimum time and pressure of refills is one of the most difficult problems in treating a case by this method, and it is here that the X-rays prove such a valuable ally. A factor which should tend to cause one to go slower both in regard to pressure and time of refills is the extent of mobility of the mediastinum causing displacement of the heart. When collapse has become established the patient's symptoms and signs are often a very good guide to the necessity of a further refill and it no uncommon thing for a patient to ask one for a refill when he feels he requires it. The indication may be either increased cough with sputum or pyrexia, or both. Indeed any of the toxic manifestations of Tuberculosis may present themselves.

The method of refilling is exactly the same as at the initial operation except that anaesthetising the track of the needle is enough and no omnopon need be
given and one can use any needle one wishes, as there is a pocket of air which prevents injury to the lung.

When collapse is well established a change of puncture spot is good practice, as the pleura round the spot which has been frequently punctured becomes thickened and may present difficulties of penetration.

When introducing the needle at refills a close watch must be kept on the manometer to recognise immediately when one is in the pleural cavity. Having satisfied oneself that the needle is there, the rubber tubing is connected to the gas bottle (it should not be connected before as I have seen the manometer water sucked into the rubber tubing immediately the stop-cock was released. Probably this was due to a partial vacuum being produced in the tubing owing to the heat of the antiseptic fluid in the gas and pressure bottles. I always use heated fluid so as to introduce the gas at as nearly body temperature as possible, because air expands on heating and if one introduces cold air into the chest wall it will expand owing to the body heat and one might get a greater pressure than one desired and cause a feeling of tightness and thus distress the patient.)

**MANOMETER READINGS AT REFILLS.**

These present a very wide variety and they depend on various factors.

(1) The pressure at which the last refill was completed. (2) The length of time which has elapsed since last refill - the longer, the time the lower we would
expect the pressure to be. (3) The extent of the disease and the state of the pleura. The smaller the extent of the disease the healthier the pleura will be and in that case the rate of absorption is very much quicker than in a thickened diseased pleura. (4) The state of compression of the lung. In an ideal case the lung should be collapsed to the size of a cricket ball, and in that case absorption is very slow. In other cases the lung is flattened against the mediastinum and absorption is relatively much quicker. Imperfectly collapsed lung may be brought about by the moveability of the mediastinum, thus preventing too high a pressure being exerted to thoroughly collapse the lung. (5) The typical reading at the refill at its optimum time is one slightly around atmospheric pressure, but more on the negative side. A pressure of -4 -0 is very typical. The manometric swing must be present all the time during a refill and should be and should be constantly sought for by digital compression of the tube between the gas bottle and the manometer. Failure to get this should immediately put one on one's guard and no more air should be introduced until it has been re-established. The probability is that the needle has become blocked, but if the needle cannot be cleared by the stilette it is necessary to withdraw it and re-introduce it elsewhere.

QUANTITY AND PRESSURE OF GAS AT REFILLS.

As before stated 250 to 300 c.c.s of gas should
only be introduced at the initial filling. The amount of gas should be gradually increased at each refill, but only by a small amount and the pressure should only be brought to atmospheric at the 7th or 8th refill. Some authors state that complete collapse should be attained at the fourth or fifth refill, but I consider that better results are obtained by a more gradual collapse. Systemic disturbances are much lessened and are more able to be dealt with by the resisting powers of the body, also the chances of more secretion than can be dealt with per via naturalis being poured into the bronchi, and bronchitis of the "healthy" lung are diminished. The disturbance to the heart and circulation are not so marked and gives the heart time to adapt itself to its altered conditions. The opposite lung is more gradually exercised in its double function. There is therefore no great advantage in such quick collapse as some writers assert.

Another point in the quantity is that in my practice I rarely give more than 1000 c.c.s at a refill and I rarely bring the pressure of a completely collapsed lung to a greater degree than +2 +4 or close to that. I have found that will maintain an adequate degree of collapse - more frequent refills may require to be done - but I consider that to be a minor point compared to the dangers of too high pressures. In cases of Partial Pneumothorax where the pressure runs up very quickly, I have given, as shall be shown later, positive pressures
of +26 +30 at each refill for months with no ill res-
ults and it was the only pressure which caused the
Pneumothorax to be effectual. One was able probably
to give such high pressures on account of the mediastin-
um being fixed. Many operators finish at each refill
with a pressure of +10 +14 especially on the Continent,
but after experience of high and low pressures and
large and small amounts of gas, I have no hesitation in
condemning the high pressure and large amounts of gas.
Results are better in the alternative case - at least
I have found them so - at anyrate a patient begins to
feel distressed at from 800 to 1200 c.cs of air. The
heart's action becomes quickened and the patient feels
uncomfortable and this discomfort may continue for 24
hours or more, and if on examination of the chest by
X-rays one finds that the lung is collapsed by these
lower pressures, and if it is sufficient to keep it
collapsed for three to four weeks a higher pressure is
uncalled for.

The ideal object in refills should be to get the
lung collapsed so as to prevent any movement and the
best method of attaining this should be refills of
small quantities, round about 500 c.cs at fairly short
intervals, and the surest indication that a refill is
required can be decided by X-ray examination.

The test of effective collapse is the failure of
the lung to expand with inspiration and the loss of
movement of the diaphragmatic cupola on the side of
the collapse. Movements of the lung due to the respiratory fluctuations of the mediastinum should not be mistaken for increase of the volume of the lung. The reappearance of symptoms should never be awaited in judging the optimum time for refill as there will at most certainly be an auto-inoculation after with its effects of further weakening the patient.

The absorption of gas from the pleura diminishes as time goes on and refills can be given at longer intervals. It also varies with the amount of disease as already noted.

The activity of the patient also hastens the absorption of gas and a patient on full work or exercise would require shorter intervals than a patient who is taking things quietly and it is often necessary to restrict a patient's activities when the rate of absorption becomes excessive. But the greatest influence of all on absorption rate is the presence of pleurisy and if an attack of pleurisy supervenes in a pleura already full of gas, the pressure may rise, apart from the presence of fluid, and the fall in the rate of absorption becomes permanent, as we shall see later, and I shall cite a case in which collapse remained absolute even after three months without refill. (Case J.B.P. No. 14).

To GENERALISE: the intervals between refills in the first three or four weeks are from one day to seven days, seven days for the next month, from seven to four-
teen days after the next two months, and thereafter from three to four weeks, and this is the general interval in well advanced cases if pleurisy does not supervene, in which case refills may be left for two or three months.

In cases of Partial Pneumothorax, on account of the mobility of the uncollapsed portion of the lung, making absorption quicker and on account of the probably higher pressures to be maintained, intervals between refills will require to be shorter than in cases of complete Pneumothorax. But again in these cases an attack of pleurisy may lengthen the intervals to almost those of the complete Pneumothorax.

GENERAL TREATMENT OF PNEUMOTHORAX CASES.

Even in an a-febrile case it is incumbent that the patient should be kept in bed during the early stages of the Induction, but as the treatment is most generally carried out in febrile, toxic cases and that toxaemia only disappears gradually, taking perhaps several weeks or even months, we would in any case be guided by the degree of toxaemia present. But even in an a-febrile case bed should be the rule until complete collapse is attained on account of the displacement of the other organs of the chest and also to prevent quick absorption. But once the toxaemia abates and the organs have become used to their displacement one of the most gratifying results of the treatment is the fact that the patient will be able to extend his exercise quickly
without fear of auto-inoculation; whereas if one had got the patient to the same degree of fitness by conservative methods his energies would have to be very much modified in comparison or toxaemia would be immediately induced. Therefore in weeks or at most months a patient is brought to that degree of fitness which might have taken years to accomplish by the ordinary methods.

**SIGNS OF PNEUMOTHORAX.**

After the first few fillings the breath sounds become gradually weaker, the percussion note becomes more resonant, but rales and crepitations, if present before, still remain. As the fillings go on these rales and crepitations gradually disappear and the resonance increases until it may become tympanitic. However that depends upon the contained gas. In the opposite lung the percussion note may alter a little, becoming hyper-resonant if there is much displacement of the mediastinum, and the breath sounds become harsh. The breath sounds in the collapsed lung seldom disappear totally as they are conducted through the gas from the opposite lung. It is not till late on in the collapse that the typical signs of Pneumothorax appear—i.e. amphoric breathing, and the bell-note, and this latter may remain absent altogether. Metallic vocal resonance, metallic whisper and tinkling rales may also be present; but I have generally found these later signs to be associated with a small amount of fluid. The
healthy lung must be carefully watched for the appearance of crepitations. These may be due to collapse from mediastinal pressure, but they may also be due to oedema, and in any case should be regarded with suspicion and the Pneumothorax more gradually proceeded with.

**SYMPTOMS OF PNEUMOTHORAX.**

The immediate symptoms on starting a Pneumothorax are often an acute exacerbation of temperature, increased cough and sputum, dyspnoea and a feeling of tightness in the chest. In acute cases the auto-inoculation may be severe and is due to the squeezing out of the secretions into the blood stream and into the bronchial tubes and one may have to wait several days before proceeding in order that these symptoms may abate. It is at this stage that a hyper-sensitiveness to the toxins may develop, especially if the other lung be diseased at all and may lead to a break-down in that, the "better" lung. Soon after, exacerbations become less and less and the well-being of the patient is markedly enhanced. Cough and sputum after being increased, become diminished, and will gradually cease if the collapse is complete. The presence of sputum may denote some lesion in the opposite lung, or the fact that the collapse is not quite complete. The character of the sputum also varies, changing from the thick nummular sputum to the thin whitish mucous.
WEIGHT. An extraordinary thing in connection with Collapse Therapy is the gradual loss of weight which occurs in practically all patients and it is a very disappointing fact that patients who under conservative treatment have gained a considerable amount of weight gradually lose it under the altered treatment. I have seen or heard no satisfactory explanation of this phenomenon.

COMPLICATIONS OF ARTIFICIAL PNEUMOTHORAX.

The most important and commonest of all complications is undoubtedly Pleurisy, and it occurs in at least 50% of cases. It may occur at any stage of the treatment, either early or later and I append Saugmann's statistics taken in 96 cases.

Within 3 months.......... 21%
  " 6 " ............ 33%
  " 9 " ............ 41%
  " 12 " ............ 44%
  " 3½ years.......... 5%

The occurrence of Pleurisy is greatly influenced by the character of the case under treatment - the more acute and advanced the case the quicker the Pleurisy appears. Weinstein of Davos experienced effusions in 70% advanced cases, 33% of moderately advanced and 10% of early cases. There is, on account of the different selection of cases, a great discrepancy amongst writers as to the percentage of cases of Pleurisy which occur in the course of treatment. These effusions are generally sterile to culture and they generally contain tubercle bacilli in large
Skiagram showing half collapsed lung with small quantity of fluid. Picture taken from behind.

X-ray of same case showing complete collapse and increased amount of fluid. Skiagram taken from front.
numbers. One explanation of the frequency of serous effusions in Pneumothorax may be that in an ordinarily treated case of Phthisis, Pleurisy is an extremely common complication, but it is quickly shut off by adhesions forming, whereas in a collapsed lung the two layers of pleura being separated the Pleurisy is not shut off but spreads all over and gives rise to an effusion. Also we must bear in mind that the pressure of the gas in the pleural cavity leads to changes in the mucosa of that cavity as evidenced by the changing time of absorption and the constant irritation of this foreign body may lead to the formation of Pleural effusions. Over-exertion and exposure to cold are often a predisposing cause of these Pleurisies, and I have seen in my cases some relationship between an ordinary catarrhal cold and the production of Pleural effusion.

PLEURAL EFFUSIONS.

(1) It may be a mild slow-forming serous exudate which gives rise to no symptoms and which will tend to disappear in the course of a few weeks or months. (2) Effusions which start as serous effusions and slowly increase until a large quantity is present. It may remain serous for many months but gradually tends to become purulent and it may fill up the whole chest. In one Case (L.W.) which I shall describe later, the effusion must have been present for quite eighteen months without giving rise to any symptoms and suddenly - after a severe wetting - the patient's temperature
went up to 104° and a purulent effusion supervened. These purulent effusions might conveniently be divided into three classes.

(a). Pyo-pneumothorax, which starts as a serous Pleurisy and which after an interval of time, anywhere from 2, 6, or 7 months, becomes turbid and then becomes purulent. This fluid may be of a benign character and give rise to no symptoms except from pressure due to great increase in its amount. This fluid does not absorb and its treatment consists in aspiration and replacement by Oxygen.

(b). In this class the onset is the same as in Class A but the benign fluid may become malignant, especially if the patient is below par from any intermittent disease. The temperature rises, becomes hectic in type with the concomitant symptoms of Hectic Fever and the outlook in this class of case is bad. Operative treatment by excision of part of a rib and drainage holds out little hope. This type may arise from infection introduced from without.

(c). Here the onset is sudden from caseation or rupture of an acutely diseased lung into the pleural cavity. This is always a fatal complication. To recapitulate, Effusions may be:-

1. serous

2. Purulent (a. Benign
   (b. Malignant
THE EFFECTS OF PLEURISY IN PNEUMOTHORAX.

Just as one has seen wonderfully curative effects from Pleurisy with effusion occurring in the course of an ordinary case of Phthisis, so in cases treated by Pneumothorax the effect may be very good; in fact it is often only after the appearance of fluid that the beneficial effects of the Pneumothorax are noted. Therefore it cannot entirely be due to the pressure that these effects take place, although no doubt the mechanical action plays a great part in a case not treated by Pneumothorax. Not only does the fluid seem to benefit the patient generally, but, as I have said before, the Pleurisy causes a diminution of the rate of absorption of gas and therefore by lengthening the intervals between refills adds greatly to the mental well-being of the patient.

There must be some serological effect of an effusion as well as mechanical else it would not react so beneficially on cases which are already collapsed. This view is upheld by the discovery of Specific Antibodies by Mayer and Von Muralt and reported by them in the "Butrage 3 Klin der Tuberk 1914". But every case of Pleurisy does not have the same beneficial results, as the effusion instead of being ushered in by no exacerbation of the general symptoms may be heralded by an acute pyrexia with its concomitant symptoms and the patient's health may receive a serious set-back. This set-back may be sufficient to allow the resistance to
be so lowered that the other lung which was not entirely above suspicion at the start, may flare up and the second state be worse than the first, i.e. before the Pneumothorax was commenced. Another serious effect of Pleurisy is the ultimate effect when one wishes re-expansion of the lung to take place. This may be prevented or greatly limited by thickening of the Pleura.

SYMPTOMS OF PLEURISY DURING PNEUMOTHORAX.

(1). The effusion may develop insidiously and it is only at the time of refill or during the routine X-ray examination of the case that it may be discovered.

(2). A succussion splash may be noted by the Physician, or even by the patient.

(3). Suppose one were to refill a case at its usual interval and to find that the pressure had not markedly diminished from the finish of the last refill, then one would most certainly suspect fluid.

(4). In the greatest number of cases some pyrexia and pain in the side is complained of. This pain is usually situated at the level of the diaphragm, and it occurs in the pleural reflection there, but one should always suspect Pleurisy when pain in the shoulder is complained of. The pyrexia may be very slight and fleeting.

(5). Vomiting may occur especially in the cases ushered in by high fever (102 - 103) and shivering. The acuteness of the onset is no criterion to the amount of exudate, as only an inch or two of fluid may be present in these cases.
PHYSICAL SIGNS.

(1). FRICTION can only occur at the pleural reflection, so there may be little evidence of that.

(2). RESONANCE is markedly diminished even before the presence of fluid can be otherwise demonstrated. When the effusion occurs of course dullness is absolute.

(3). SUCCURSAL SPLASH is demonstrable even with infinitesimal amounts of fluid.

(4). AUSCULTATION. In an ordinary Pleurisy with effusion, breath sounds are absent, but in a case treated by Pneumothorax breath sounds are naturally absent or diminished by the presence of the gas. The breath sounds may be more audible over the fluid than over the gas. Vocal resonance is not much altered.

(5). X-RAYS examination is of course the quickest, surest and easiest method to diagnose Pleurisy with effusion and in this connection it is found that the upper level of the fluid shows as a straight horizontal line, whereas in an ordinary effusion one finds the parabolic curve so characteristically presented.

(6). DISPLACEMENT of other organs depends on the pressure entirely and may not be markedly different from the displacement brought about in the course of treatment.

TREATMENT.

It is extremely difficult to lay down hard and fast lines of treatment but my belief is that it should be as conservative as possible. The symptoms must be
treated by rest in bed, especially if pyrexia is present.

The question arises as to aspiration, but I think that a simple fluid should be left. Pressure should be taken occasionally and more gas added if this tends to become low enough to prevent efficient collapse. If the fluid is causing dyspnoea owing to its pressure on the heart or if it is filling the whole pleural cavity some of it should be drawn off and replaced by Oxygen. In my practice I never withdraw the whole of the fluid. Very often in the acute Pleurisy ushered in by fever, shivering and malaise the accustomed pleural pressure seems to cause discomfort and this discomfort will only be relieved by withdrawal of the fluid. The pressure in the chest after being relieved by this means should be kept at atmospheric level. This may require to be done two or three times before the symptoms are relieved.

A more difficult question presents itself in the case of purulent effusions and one cannot be so dogmatic about it but I certainly think that these effusions of purely tubercular infection or of mixed infection should be treated by aspiration and gas replacement if possible rather than by open operation. In the latter case a large suppurating cavity is left between the lung and the chest wall which it is impossible to close. My practice is to aspirate all effusions which become turbid as these will go on to pus format-
-ion in the course of time and the pus may become too thick to aspirate therefore necessitating a Pleurotomy being done. Aspiration and replacement will often suffice to cure the patient. In frankly purulent effusions the same procedure should be adopted, but in addition to this lavage of the chest wall may be carried out through the aspiration needle and this is especially useful in secondarily infected effusions.

Perhaps at this stage the operation for aspiration and replacement of gas might be advantageously described.

INSTRUMENTS.
(1). Pneumothorax Apparatus as before described.
(2). An Aspirator.

I always use an ordinary Petain's Aspirator, but omit the trocars, only using a wide-bored aspirating needle. I have tried trocars and find them cumbersome compared to the ordinary needle. I have found the aspirating bottle exhausted by a hand pump much more easily and quickly worked than the method of withdrawal by syphonage as practiced by many. With the patient propped up as high as possible, the aspirating needle is introduced preferably at the posterior axillary line in spaces 9 or 10. A little fluid is withdrawn to the bottle to make sure that the needle is in situ. The stop-cocks on the bottle are now closed. The Pneumothorax needle is then introduced and care must be taken to do this well above the level of the fluid. The
usual manometric fluctuations will be observed when the needle has entered the pleural cavity. If the initial pressure is high some fluid may be drawn off until the patient's usual pressure is arrived at, and then it is best to maintain that pressure by a simultaneous steady withdrawal of fluid and admission of gas. This is only possible by the moveable bottle pneumothorax apparatus and is a further argument in favour of its use. In the fixed bottle apparatus one requires to alternately withdraw and replace with gas and great variations in the manometer readings may be produced. It is also a tedious method.

None of the usual distress occasioned by aspirations is noted in the cases which are replaced by gas. When the fluid is withdrawn, as is evidenced by the escape of bubbles into the aspirating bottle, the aspiration needle should be removed and the opening closed with collodion and gauze. Before withdrawing the Pneumothorax needle the pressure should be adjusted so that the intra-pleural pressure is a little lower than customary to the patient, to allow for the further development of fluid which will, in all probability, take place. The peculiar point is that a smaller quantity of gas to bring the pressure to the same level is always introduced than the amount of fluid withdrawn. An accident which may occur after withdrawal of the aspiration needle is infection of the track and the development of a cold abscess at that site. I have
only seen this in one case when an abscess the size of a hazel nut appeared but this caused no distress and required no further treatment than protective dressings and in the course of a few weeks it disappeared.

**NATURAL PNEUMOTHORAX AS A COMPLICATION OF ARTIFICIAL PNEUMOTHORAX.**

In my series of cases I have only seen this complication occur once, nor have I seen it mentioned in any literature. I do not classify this complication along with those cases of perforation of the lung which is another complication and which resembles in its symptomology very closely this accident which I shall describe amongst my Cases (E.S. Case No. 4). In this case the hole evidently closed immediately causing symptoms by a sudden and great rise of pressure - in the case of perforation the opening remains patent or valvular causing in the first example fall of pressure and in the second a diversity of pressure according to whether the hole is open or shut.

**REACTIONS**

As a general rule after the initial operation and after the first three or four refills there is a febrile reaction. The severity and duration of this depends greatly on the acuteness of the case operated upon. It also depends on the amount of gas introduced, hence the necessity of only obtaining a very slight rise of pressure at the initial operation.

**TREATMENT.** No special treatment is required but delay in giving another refill must be observed until the
temperature has returned to the point it reached before 
the operation.

**SURGICAL EMPHYSEMA.**

This is a fairly common complication but it need give rise to no anxiety as it rapidly clears up without any untoward symptoms occurring. Indeed its most distressing symptom is the anxiety to which it gives rise in the patient's mind. It may be due to gross carelessness on the operator's part by introducing the gas when the needle is not in the pleura at all but only in the tissues of the chest. I can recall in the early days of pneumothorax one operator doing this at an initial operation and in an hour the patient was emphysematous all over one side of his body. A small escape may take place into the tissues if the track does not close up immediately, especially if there is much coughing, but this can generally be obviated if the skin round the puncture be pinched up before withdrawing the needle. No treatment is called for.

Deep or mediastinal emphysema is rare but it may occur if the gas gets under the visceral pleura into the lung, from thence it tracks into the mediastinum and appears in the neck above the sternum. It causes tightness of the chest and distress in breathing. Cyanosis may occur but this soon passes off, and Aronson reports one case of surgical emphysema of great severity and which caused the death of the patient. (Tubercle June, 1922).
PERFORATION OF THE LUNG.

This is a rare occurrence luckily and I have only seen it once and I have grouped my case under a different heading as it was evidently not a true perforation of the lung or at least it closed quickly. Fortanin experienced eight cases of perforation in one hundred and thirty nine cases. The cause is generally ulceration of a cavity or caseous area into the pleural space but it may occur in an incomplete Pneumothorax especially where cavities are present, by violent efforts of the patient tearing down a piece of thin-walled lung. Symptoms are sudden, violent pain is felt in the side and is reflected in various directions. In the case I cite the pain was over the left half of the abdomen and looked extremely like one of the abdominal crises with rigidity and tenderness there. Vomiting occurred. The temperature rapidly rises, often to 104 or 105. Before long fluid develops and dyspnoea and cyanosis will be present if the aperture closes but not if it remains patent. The only sure diagnostic point is the introduction of a needle attached to the manometer when one of the following two conditions will be found. (1) A raised pressure (in which case the other organs will be displaced to a greater degree than formerly and that indicates that the aperture is closed. (2) A lower pressure than formerly and this is not affected by the introduction of gas. If the opening be valvular one would expect zig zag movements of the manometer.
PROGNOSIS is generally very bad, acute infection of the pleural cavity may carry off the patient in a few days or weeks. If not, a lung fistula persists and the pleural fluid is coughed out through that or is drained by that channel. Hectic fever persists and the patient gradually dies or weakness any may be of amyloid disease. Treatment is entirely symptomatic.

PLEURAL SHOCK.

This complication I have not met with except in a very minor degree on two occasiona when it only caused a slight feeling of faintness, pallor and sweating with no appreciable lowering of the quality of the pulse. It proved transitory and the patient felt all right after a draught of water.

The SYMPTOMS of PLEURAL SHOCK may be immediate at the time of puncture or remote, occurring 15 to 20 minutes after, or even an hour. They consist of pallor, giddiness, pulse becomes irregular and weak. Breathing becomes shallow, toxic and clonic spasms occur affecting either all the muscles of the limbs or isolated muscles. These symptoms may last from a few minutes up to a few hours and they may even go on to coma and death. Forlanini says that the peculiarity of pleural shock is that it tends to reappear at subsequent fillings and this may lead to the abandonment of this method of treatment. It occurs 12 times in 134 of his cases with over 10,000 fillings. Saugmann denies that patients suffering from the above symptoms are examples of
pleural shock but that these cases are due to embolism. He says that in 210 cases he had 16 accidents, two of which proved fatal, and it is noteworthy that 14 out of these 16 including the 2 deaths occurred in his early experience of Pneumothorax when he adopted the principle that it didn't matter whether he wounded the lung or not. Saugman advised that no more than two attempts at finding a pleural space be made at the initial operation. Sachs records 26 cases of pleural shock, none fatal, in 1058 cases.

**PROPHYLAXIS.** As mentioned before if omnopon be given a half to one hour previous to the initial operation and local anaesthetatation of the track be carefully carried out the danger of pleural shock should be reduced to a minimum. In fact I have not seen any cases of recorded in any recent literature.

**TREATMENT** consists of withdrawal of the needle and the application of hot cloths to the heart and the exhibition of pituitrin, which should always be kept ready on the instrument table.

**GAS EMBOLISM**

The symptomology of this complication is exactly similar to that of pleural shock and it is a complication which is so very rare that I am not going to describe it further except to reiterate the statement that the stop-cocks allowing the entrance of gas from the gas bottle should never be opened until one is satisfied that the needle is in the pleural cavity by the respir-
atory fluctuations occurring in the manometer.

VALUE of X-RAYS in PNEUMOTHORAX TREATMENT.

I consider that the use of the X-rays as a guide in Pneumothorax treatment is absolutely essential. It is necessary to have a radiogram of the chest before commencing treatment in order firstly to determine the state of the 'better' lung as to the presence of disease and its extent if present, and secondly to see the condition of the diseased lung as to cavities or thickened pleura so that it may be a help as to selecting the site of puncture. After induction it is only the X-rays which can tell us the degree of collapse, the regularity of collapse and the presence or absence of adhesions or bands. X-rays are also necessary in determining the optimum time of refills, because the optimum time is before clinical symptoms or signs appear. This is best shown in screening the chest by the presence or absence of movements of the diaphragm on the collapsed side and also of the lung. Its greatest value is the detection of small quantities of fluid.

I shall deal with the X-ray plates shown in this Thesis under the individual cases.
CASE No. 1.

G.G.K. admitted to Sanatorium originally on 5/7/17 with a history of sudden haemoptysis (2 oz) in May of that year while training in the Army. The physical signs then were dullness, bronchial breathing and mucoid crepitations over the upper lobe of the left lung and dullness and a very few crepitations over the apex of the right lung. Sputum positive. Pyrexia present. After about 14 days in bed he was allowed up and put on exercise and by the time he left at the end of October 1917 he was feeling well and was able to do about 5 miles walking exercise daily.

He kept well all that Winter but in the early Spring of 1918 he had two quite sharp haemoptyses and he returned to the Sanatorium on 30/4/1918 with the signs much the same as before. Haemoptysis occurred about a week after arrival, twice on two succeeding days and pyrexia was present. He continued up and down all that Summer with occasional haemoptysis, exacerbations of pyrexia, increase of cough and sputum and the physical signs became worse, some softening appearing in the left upper lobe. During the Winter 1918 - 1919 he was continuously in bed rapidly going downhill until May 1919 when Pneumothorax was induced. I have not the details of his early progress but collapse was attained in about 10 days. His toxaemia then rapidly subsided and he was able to get up on exercise, the first time for almost a year. Pleurisy developed
about September 1919 with an effusion which gradually increased till it practically filled the left pleural cavity. In January 1920 it required to be aspirated on account of the distress it was causing and 1800 c.c. of fluid were withdrawn and replaced by 1500 c.c. of Nitrogen. No further refill was required after that, illustrating the point of the slow absorption of gas after pleurisy. A small amount of fluid still remained in the pleural cavity. He left the Sanatorium in June 1920 and returned home and we then lost sight of him for some time but I have been able to obtain his history from June 1920 till he returned to us in October 1922. I shall quote his own words:

"Left Nordrach on 30th June 1920. Lived very quietly for 2 months. Gradually resumed, more or less, normal life. Spent six months on a hen farm and kept fairly fit. Left for Glasgow district about April 1921 Lived a quiet normal life in various parts of the country till about end of December 1921, when I left for Switzerland. On arrival at Grindelwald went to bed with 'flue, which developed into pneumonia in my 'good' lung. Seriously ill. Was blown up about Feb. Wasn't necessary, but some gas put in. About middle of March 1922 was taken in a stretcher from Grindlewald to Montana. The 3000 ft difference in altitude caused pressure to rise to over +21. Gas was taken off till pressure was about +12 +14. Great relief from pain. Pneumonia gradually cleared off. Great increase of
sputum about this time. Pleural cavity well filled with fluid. Got fit enough to travel back to Glasgow about end of May 1922. Exploring syringe proved sputum to be fluid from pleural cavity. Entered Nursing Home and had fluid drawn off and replaced by air every few days for about a month. Again took pneumonia in my 'good' lung on 13th July 1922. Seriously ill. Gradually recovered sufficiently to travel to Nordrach in October 1922. (G.G.K)"

He is an extremely intelligent patient and takes a great interest in his condition so that we may take it that his statement is fairly accurate, but we must accept the fact of his coughing up fluid from his pleural cavity with a little doubt. Of course it is quite possible that it was so and I am inclined to believe it as his statement is so clear on the point that, if the fluid was withdrawn to a certain level and gas introduced his sputum stopped, or nearly so, and immediately the fluid rose again above a certain level he recommenced having sputum in large quantities. Evidently by the time he returned to us in October 1922 the rupture in his lung had healed as he had no sign of that sputum whilst here and we even injected methylene blue into the pleural cavity to see if it would reappear in the sputum.

Another point of interest in his statement is the effect of altitude on the intra-pleural pressure. He suffered extreme discomfort and on examination of the
intra-pleural pressure it was found to be about +21. Gas required to be drawn off. According to Bernard Hudson (Brit. Jour. of Tuberculosis) 2000 c.cs of air at Sea-level increase by 80 c.cs for every 1000 ft, and Riviere in his book on Pneumothorax treatment states that in a full Pneumothorax of 3 - 4 litres a rise in altitude of from 3250 - 5000 ft. is equivalent to introducing 400 - 800 c.cs of gas. By that one can see that dangerous pressures may be caused and a patient with Pneumothorax should not be sent to high altitudes, without the pressure being suitable adjusted and the patient warned to break his journey should signs of dyspnoea appear. Patient returned here on 3/10/22, when on examination there was marked retraction of the left chest wall. Crepitation audible over left intra-scapular area, fluid to lower angle of scapula, breathing sounds practically inaudible all over left chest. Lassitude was marked, cough very troublesome, pyrexia to 100. Right chest was clear. On 28/10/22 intra-pleural pressure +5 +15. 31/10/22, pressure +10 +16. Fluid increased, 325 c.cs of purulent fluid withdrawn, 400 c.cs of air introduced. Final pressure +4 +8. Fluid was found to contain many tubercle bacilli but only a few septic organisms. Kept in bed owing to pyrexia.

21/11/22. Initial pressure +12 +18, 600 c.cs fluid withdrawn, final pressure +6 +14, 600 c.cs air introduced. Fluid still very purulent. Cough still very troublesome, and sputum copious. After this temperature
CASE No. 1.

Skiagram showing practically flat lung in left side and small amount of fluid. Note deposit in right lung.
tended to come down and patient was able to be up. He gradually improved very much and was able to do about 4 miles a day.

24/2/23. Initial pressure +8 +12. Fluid withdrawn 500 c.cs, end pressure +4 +6, air introduced 500 c.cs. Patient still up and about, except after refills when there was always some reaction. All symptoms better except cough and sputum which are still rather troublesome.


20/6/23. -8 -4 200 c.cs Air
+6 +10 No fluid withdrawn.

Patient felt well, able to do about 5 miles daily without toxaemia. Sputum still present but contained no T.B., and in all probability was due to a bronchiectatic condition of the right lung. He left us for the Summer when he led an ordinary life, motoring about all over the country. Since leaving he has been refilled at intervals of about a month, pressure being round -6 -2 at the commencement and being brought up to -6 -8 after about 150 - 200 c.cs gas.

4/11/23. Re-admitted. Refilled 20/11/23 -3 -3 100 c.cs
+10+14 Air

27/12/23 -8 -2 100 c.cs Air. After this last refill he had malaise and irregular pyrexia and fluid accumulated until 15/1/24 until aspiration and replacement by gas performed, -0 +4 260 c.cs turbid purulent fluid withdrawn, gas introduced quantity unknown.

I am afraid that lately he has had rather
a set back, but after a few aspirations and replacements I have no doubt that his condition will become as good as it was before, so he will again be able to lead a moderately active life and get some pleasure out of it.

This case presents several features of interest.

(1). Early development of fluid thereby causing great increase in the intervals between refills.

(2). It shows the value of Pneumothorax as this patient had been ill for two years with a gradual increase in the severity of his symptoms causing him to lead a life of acute invalidism which would only have ended in death at a sooner or later period.

(3). Even though pneumonia on two occasions developed in the sound lung, recovery took place.

(4). The effect of altitude on pressure.

(5). The probability of rupture of the lung with infection of the pleural fluid, and recovery.

(6). The correct treatment of Pyo-pneumothorax is by aspiration and replacement with gas.

Even with all the complications and set-backs which this patient has suffered, if this patient required to work for his living he is quite able to do so in my opinion.
CASE No. 2. (L.H.W.)

L.H.W., age 34, admitted 8/1/19.

History. June 1918, influenza, not sufficiently bad to 'lie' up, but never felt well after it, easily tired, commencing cough and sputum in the morning. Began to lose weight. August, 1918, consulted Specialist in London who diagnosed Pulmonary Tuberculosis, sputum positive. End of August, haemoptysis 2 ozs repeated in small amounts on the four following days. 29/9/18 admitted to Finewood Sanatorium, Wokingham. 3 weeks in bed there with pyrexia. After that was allowed up on exercise and was doing 5 - 6 miles daily till admission here. Previous history, nil to note. Family history: One brother with whom he lived died of tubercle. Symptoms on admission were; cough morning and evening, fair amount of sputum; weight pretty well up to normal, and otherwise feeling pretty well. No digestive symptoms. Temperature ranged from 98 a.m to 100.2 p.m. Signs. Loss of expansion over right upper lobe. Dullness to R.4 in front and to junction of middle and lower third of scapula behind. Bronchial breathing over that area and moist sounds both on expiration and inspiration over that same area. V.R. increased back and front but no signs of cavitation. Tubercle bacilli were very numerous indeed. Nothing to note in left chest but a few crepitations after coughing, in interscapular region.

Progress. 28/1/19, haemoptysis ¼ to 1 oz, repeated on
three succeeding days, and then off and on till 9/2/19.

20/2/19 recurrence of haemoptysis until 26/2/19 since when sputum cleared. Temperature now going to 100.6 p.m. The signs in the left chest were rather less intense but there was definite evidence of excavation in right upper lobe. Patient was kept on typhoid rest throughout March and April with little change in temperature, but cough and sputum became less (see charts). He was kept on typhoid rest until 18/6/19, although temperature was tending to be a little higher there was no more bleeding. On 2/7/19 pleurisy developed over right lower lobe and there were sonorous rhonchi both back and front. Crepitations remained much the same but cavity very evident. Cough and sputum increased - digestion becoming upset. Pyrexia increased and our only deduction was increased activity. 29/7/19, Haemoptysis which recurred about sixteen times up till 9th September. Generally these haemoptyses were small but one or two were fairly large. 15/8/19 staining stopped pyrexia now ranging as shown in charts. After this, improvement set in; pyrexia became less, general condition improved, cough and sputum less, but digestion continued to be troublesome - the bowel muscles becoming very atonic and much fermentation taking place in the caecum and colon. 1/12/19 symptoms again became more marked and on examination at this time there was distinct evidence of increased activity going on to softening in the right lower lobe. Nascent iodine, sodium
morrhuate, along with general sanatorium treatment had all been tried with no success so in January 1920 it was decided to attempt an artificial pneumothorax.

9/1/20 Pneumothorax successfully induced.  

<table>
<thead>
<tr>
<th>Date</th>
<th>Pressure</th>
<th>C.C.S.</th>
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<tbody>
<tr>
<td>11/1/20</td>
<td>-12 -9</td>
<td>400</td>
</tr>
<tr>
<td>15/1/20</td>
<td>-12 -9</td>
<td>400</td>
</tr>
<tr>
<td>20/1/20</td>
<td>-17 -9</td>
<td>800</td>
</tr>
<tr>
<td>27/1/20</td>
<td>-8 -4</td>
<td>850</td>
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</tbody>
</table>

Lung completely collapsed. There were very slight reactions but we had a good deal of trouble with the flatulence of which he had complained so long. There was some diminution in cough but sputum was hardly changed in amount. Pyrexia to 100.2 p.m. still present. Able now to be up for 3 hours daily.

Refills continued.

<table>
<thead>
<tr>
<th>Date</th>
<th>Pressure</th>
<th>C.C.S.</th>
</tr>
</thead>
<tbody>
<tr>
<td>5/2/20</td>
<td>-6 -3</td>
<td>800</td>
</tr>
<tr>
<td>12/2/20</td>
<td>-8 -2</td>
<td>1100</td>
</tr>
<tr>
<td>19/2/20</td>
<td>+2 +4</td>
<td>800</td>
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</tbody>
</table>

Cough improving and sputum diminishing, digestion very much improved. Complete collapse except over cavity in right apex.

<table>
<thead>
<tr>
<th>Date</th>
<th>Pressure</th>
<th>C.C.S.</th>
</tr>
</thead>
<tbody>
<tr>
<td>3/3/20</td>
<td>-6 -2</td>
<td>900</td>
</tr>
<tr>
<td>18/3/20</td>
<td>+0 +4</td>
<td>900</td>
</tr>
</tbody>
</table>

Improvement. Cough and sputum still present but less probably coming from upper lobe of right lung where collapse was incomplete. No signs of any moisture in
left lung.
Refilled.  
6/4/20  
27/4/20  
Now able to be outside on rest, still slight degree of
toxaemia but improving generally.

18/5/20  
4/6/20  
28/6/20  
Sputum and cough much diminished. X-rays showed coll-
apsed area in upper lobe to be much smaller, bacilli
fewer than before.

15/7/20  
2/8/20  
20/8/20  
For the first time since admission tubercle bacilli
are absent from the sputum. Cough and sputum practical-
ly nil.

4/9/20  
6/9/20 left Sanatorium to live in Upper Deeside. There
he kept very well, no pyrexia, cough and sputum often
absent, until some re-expansion took place, when it
increased.

15/10/20  
9/11/20  
1/12/20  
1300 c.cs air
925 c.cs air
750 c.cs N₂
900 c.cs air (doing small
amount of exercise now)
1300 c.cs N₂
1100 c.cs air.
1125 c.cs air
After this refill a very small amount of fluid developed.

<table>
<thead>
<tr>
<th>Date</th>
<th>Change</th>
<th>Fluid Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td>22/12/20</td>
<td>-5  +1</td>
<td>300 c.cs</td>
</tr>
<tr>
<td></td>
<td>+0  +4</td>
<td>O₂ given</td>
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</table>

1921. Fluid now reached 5th rib in nipple line.

<table>
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<tr>
<th>Date</th>
<th>Change</th>
<th>Fluid Amount</th>
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<tbody>
<tr>
<td>2/2/21</td>
<td>-10  +4</td>
<td>500 c.cs O₂</td>
</tr>
<tr>
<td></td>
<td>+1  +5</td>
<td></td>
</tr>
<tr>
<td>24/2/21</td>
<td>-10  +6</td>
<td>500 c.cs air</td>
</tr>
<tr>
<td></td>
<td>+8  +10</td>
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</table>

Since that time patient passed out of our hands but I have seen him just recently and he is being refilled at intervals of about 2 months with gradually decreasing amounts of gas so as to gradually re-expand the lung. One one occasion fluid required to be withdrawn and to be replaced with gas, otherwise it has not required to be interfered with. He has kept well during all these years and has been living a fairly active life, having done a good deal of travelling all over the Continent. He has been back to work as a Stockbroker, and plays golf and hunts.

This was a very successful case of Pneumothorax as this patient was fast going downhill and no treatment had any effect. Our only regret is that the Pneumothorax treatment was not started earlier. In the light of our present experience there is no doubt but that we would have commenced it at least 6 months earlier than we did. That is practically the only lesson to be learned from it.
CASE No. 3. (R.G.B.)

R.G.B., Practitioner of Medicine, age 28, admitted 26/11/19.

HISTORY. August 1915 invalided from France, diagnosed debility, off duty till December 1915. May 1916, haemoptysis (2 ozs), tubercle bacilli found in sputum. Kept on home service feeling fairly well until cough and sputum developed in November 1918 when he did not feel quite so well. Boarded and invalidated from the Service but was able to carry on his work. May 1919 haemoptysis (3 ozs), off duty for a week, did not feel ill. July 1919 recurrence of haemoptysis (3 ozs) again in bed for a week, occasional pain was noticed over right base. 15th September 1919, further haemoptysis, pyrexia now developed up to 102 p.m. orally, only two weeks in bed and again resumed duty. 2nd October 1919 pyrexia and haemoptysis. Pyrexia has never abated so he was kept in bed till admission here.

PREVIOUS HISTORY nil to note.

FAMILY HISTORY nil to note.

SYMPTOMS. Cough with considerable sputum, pain in chest, dyspnoea on exertion, no night sweats (these had been present in September). Loss of about one stone in weight. Pyrexia to 100 p.m.

SIGNS, right lung, loss of expansion at right apex with slight flattening front and back. Dullness to R 3 in front and to mid-scapula behind, also dullness at base. Breathing, bronchial with crepitation on ordinary
breathing over same area; breathing was very faint over right base. Left lung: with the exception of dullness at extreme apex there were no other signs.

PROGRESS. 1/12/19. Pleurisy over right base, settled down after a day or two, soon began to get better and not so tired, cough and sputum diminishing. There was distinct increase in the signs in the right lower lobe however. 24/1/20 still in bed although temperature lower; signs much as before, feeling better. March 14th allowed up for an hour or two but had pyrexial attack next day, temperature going to 102, so put back to bed. Developed friction at right base, allowed up on March 29th, temperature having settled down again. Signs were still intense. Only 12 days up when he had to return to bed and was kept there till 17th April. The signs in the right lung were much more extensive, crepitation being heard practically all over front and back. Left lung was clinically sound.

ARTIFICIAL PNEUMOTHORAX induced 28th April, 1920.

<table>
<thead>
<tr>
<th>Date</th>
<th>O2</th>
<th>N2</th>
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<tbody>
<tr>
<td>28/4/20</td>
<td>200 c.cs</td>
<td>700 c.cs</td>
</tr>
<tr>
<td>30/4/20</td>
<td>300 c.cs</td>
<td>600 c.cs</td>
</tr>
<tr>
<td>3/5/20</td>
<td>500 c.cs</td>
<td>700 c.cs</td>
</tr>
<tr>
<td>6/5/20</td>
<td>600 c.cs</td>
<td>700 c.cs</td>
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<tr>
<td>9/5/20</td>
<td>700 c.cs</td>
<td>900 c.cs</td>
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<tr>
<td>12/5/20</td>
<td>900 c.cs</td>
<td>925 c.cs</td>
</tr>
<tr>
<td>19/5/20</td>
<td>650 c.cs</td>
<td>1050 c.cs</td>
</tr>
<tr>
<td>26/5/20</td>
<td>650 c.cs</td>
<td>1050 c.cs</td>
</tr>
<tr>
<td>10/6/20</td>
<td>900 c.cs</td>
<td>1050 c.cs</td>
</tr>
<tr>
<td>21/6/20</td>
<td>925 c.cs</td>
<td>1050 c.cs</td>
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</table>
By now there was good collapse of the lower lobe, but none over the upper lobe. Digestion was very troublesome and patient had no appetite. Able to go for about 2 miles daily with no toxaemia. The indigestion was probably caused by pressure on the liver and other abdominal viscera. Sputum was still present and contained tubercle bacilli.

<table>
<thead>
<tr>
<th>Date</th>
<th>Temperature</th>
<th>Pressure</th>
<th>Treatment</th>
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<tbody>
<tr>
<td>2/7/20</td>
<td>-6 -2</td>
<td>1300 N₂</td>
<td></td>
</tr>
<tr>
<td>12/7/20</td>
<td>+1 +5</td>
<td>625 c.cs N₂</td>
<td></td>
</tr>
<tr>
<td>24/7/20</td>
<td>-6 -2</td>
<td>800 c.cs N₂</td>
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Crepitations still heard over uncollapsed portion of lung. T.B. still present in sputum. Toxaemia nil. Two to three miles exercise daily, digestion still troublesome.

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<th>Date</th>
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<th>Pressure</th>
<th>Treatment</th>
</tr>
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<tbody>
<tr>
<td>4/8/20</td>
<td>-6 -2</td>
<td>800 c.cs Air</td>
<td></td>
</tr>
<tr>
<td>14/8/20</td>
<td>+1 +5</td>
<td>800 c.cs Air</td>
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Digestion much better. T.Bs not so numerous. Still crepitations over upper lobe.

<table>
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<th>Date</th>
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<th>Pressure</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>26/8/20</td>
<td>-6 -2</td>
<td>900 c.cs N₂</td>
<td></td>
</tr>
<tr>
<td>28/8/20</td>
<td>+1 +5</td>
<td>800 c.cs N₂</td>
<td></td>
</tr>
<tr>
<td>22/9/20</td>
<td>-1 +3</td>
<td>500 c.cs N₂</td>
<td></td>
</tr>
<tr>
<td>13/10/20</td>
<td>+5 +8</td>
<td>1000 c.cs Air</td>
<td></td>
</tr>
<tr>
<td>3/11/20</td>
<td>-6 +2</td>
<td>900 c.cs Air</td>
<td></td>
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</tbody>
</table>

No tubercle bacilli found after concentration.

<table>
<thead>
<tr>
<th>Date</th>
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<th>Pressure</th>
<th>Treatment</th>
</tr>
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<tbody>
<tr>
<td>24/11/20</td>
<td>-7 -9</td>
<td>1200 c.cs Air</td>
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After this refill he left Sanatorium as sputum had been negative on three occasions during the previous month. He went to Speyside where he carried on treat-
ment, returning for refills. He was refilled, getting an average of 600 c.cs Air, the end pressure being about 4+10. Complete collapse did not take place. He kept very well and there was no return of bacilli in the sputum. He kept well all 1921 except towards the end when digestion became very troublesome and he was advised to undergo observation at Duff House, there the condition was diagnosed as attributable to Tuberculosis and to the treatment by Pneumothorax pressing on and displacing the abdominal viscera. As time went on the air space in the pleura gradually became smaller, due to the growth of adhesions, so that a very much higher pressure was recorded with smaller quantities of gas.

1/12/21  4+0 17  500 c.cs N₂ and this refill was typical of those immediately preceding and succeeding it.

31/12/21  -3 +1
          +20+23  450 c.cs N₂ even in that short interval space had been lessened but he was advised to carry on Pneumothorax as long as possible.

After being at Duff House, patient carried on treatment at home, returning every three weeks or so for refills, and kept well. In April 1922 he went to Switzerland and became Assistant Medical Officer to a Sanatorium there and he has remained there since.

October 1922 he writes: "I think I may say I am distinctly better although I have not put on much or any weight and there is still a little sputum. I have been getting my refills at intervals or 3 - 4 weeks and
Skiagram taken immediately before refill:
pressure -4

Skiagram taken immediately after refill:
pressure +20. 400 c.cs gas.
always with the pressure relations much as you left me" April 23rd he writes: - "I feel very fit. I was recently X-rayed before and after a 'blow-up' and send you the pictures (see photo. No. 3). This refill was after an interval unattended of about seven weeks and I took only 400 c.c.s, pressure 44±20, the cavity present being very small and it is doubtful whether a refill can be having much effect on me now".

21st January 1924, he writes: - "I am still having refills The cavity is apparently contracting slowly. I take about 450 c.c.s at about +10 every three or four weeks. Sputum is not all gone but is 'bug-free' and I can do quite a good day's work. I can only run up one flight of stairs in comfort, but one can't expect to do more at this altitude"

We have in this case an example of a long-standing slowly progressive case of Tuberculosis. It went on for nearly 4 years with hardly any symptoms but it passed in 1919 into a sub-acute phase which did not react well to Sanatorium treatment - in fact toxaemia was present after 4 months rest in bed and the signs became more extensive and intense. Progress was much delayed by the mal-nutrition of the patient and by his exceedingly poor digestion. In fact this complication nearly proved his undoing. After seven months' treatment by Pneumothorax, although a partial one was only obtained, this patient was sufficiently recovered to leave the Sanatorium and to carry on treatment at his home, leading a moder-
ately active life. Two years after commencing treatment patient was able to resume the practice of his profession and has been able to carry on since, a result we could hardly have hoped for under ordinary methods. He had done some special pathological work and one or two locum tenancies in 1921 and early 1922. A point of interest in this case was the gradual lessening of the Pneumothorax cavity. I don't remember seeing or hearing of a similar case. I can't see that much benefit can now be gained by continuing his Pneumothorax so long except for comfort as it is often noticed that on withdrawing the Pneumothorax that the patient complains of pain and discomfort, and often expresses the desire for its continuance.
CASE No. 4.

M.E.S. aet 23, admitted November 6th, 1920.

PREVIOUS HISTORY. Haemoptysis in January 1920 - half pint coughed up. Seen by Doctor who found nothing. She started training as a Nurse in February 1920 and had a slight haemoptysis during the first month but kept on working. July 1920 cough started after another haemoptysis and has persisted ever since, emaciation gradually increasing. In October marked pyrexia developed, night sweats and greatly increased cough and sputum. Gave up nursing about middle of October. Advised to come here and was admitted on November 6th. She has family history of two brothers dying of Tuberculosis.

ON ADMISSION she was about 17 lbs under weight, cough very troublesome and a large amount of sputum. Dyspnoea was present on the slightest exertion, and night sweats were also present. For temperature and pulse-rate see charts. Patient was put on typhoid rest but had continued fever. She gained a lot of weight.

CONDITION OF CHEST ON ADMISSION. Left lung. Flattening and deficient movement, dullness to percussion to R 4. in front and to lower angle of scapula behind. Fine and medium crepitations all over this area of dullness with consolidation of apex in front and behind. Right lung. Dullness at apex in front and behind with a few fine post-tussive crepitations, especially above the clavicle and at the root behind. This side settled down in a few weeks and at the time of the induction of Artificial
Pneumothorax was quiescent.

January, 1921. Artificial Pneumothorax decided on as after three months treatment there was very little improvement in the toxicity, but unfortunately she developed acute pleurisy and it was decided to postpone it till that settled down. Pneumothorax was induced on 4th March 1921 and a space easily found. First filling was of 200 c.cs Oxygen. Initial pressure -16 -8, -12-4 no reaction.

6/3/21. - 14 -8, - 12 -3  250 c.cs O₂ Slight delayed reaction.

15/3/21 - 13 -2 - 8 -3  350 c.cs O₂

16/3/21 - 10 -4 - 6 -0  500 c.cs O₂

Temperature remained up after these two refills at 102 showing no signs of coming down.

22/3/21 - 10 -2 - 6 -1  450 c.cs O₂

Fall of about a degree a day or two after this.

29/3/21 - 12 -3 - 4 -0  550 c.cs Air.

Reaction lasting 5 days after this.

5/4/21 - 10 - 1 - 3 - 1/2  600 c.cs Air

Temperature did not rise after this.

12/4/21 - 4 - 10 - 3 - 1  1000 c.cs Air. No reaction.

18/4/21 - 10 - 1 - 3 - 4  800 c.cs Air

Sharp reaction but over next day.

22/4/21. In the early hours of the morning patient reached over the bed for something and felt a sharp sudden pain in the left side radiating to the left shoulder and she vomited. On examination she was markedly dyspnoeic, pulse was very weak and she looked distressed. There was pain and tenderness and rigidity over practically the whole left half of the abdomen but the heart was not displaced further to the right
than it had been formerly. It looked extremely like an abdominal crisis and one had to consider that possibility as the signs were mostly abdominal. Chest signs were about the same as before, nothing definite to be made out. However, a provisional diagnosis of spontaneous pneumothorax and a needle introduced and the diagnosis was confirmed by a marked rise in pressure +14 ±16 and 800 c.cs air were drawn off, leaving the pressure at 4 ±4, with marked relief of the symptoms practically immediately. Temperature rose to 102 on the following morning and was 105 that night, but beyond the discomfort due to the high temperature, the patient was really very comfortable and the pain much less.

27/4/21. Temperature still remained high and patient had a haemorrhage (about 200 c.cs), fluid developed to about lower angle of scapula, and after this temperature gradually fell and she was able to get up on 3rd June.

9/6/21. Refill -2 +2 +8 +7 200 c.cs O_2 showing that after that time had elapsed there had been very little absorption of gas and the intra-pleural pressure was well maintained on account of the pleurisy. Temperature now remained normal, or nearly so, cough and sputum were practically nil, but fluid increased to almost spine of scapula. Patient nevertheless felt well and left the Sanatorium on November 1st. She continued to be very well and was able to do 6 - 8 miles exercise daily, but in February 1922 developed an ordinary catarrhal cold
with pyrexia which kept her in bed.

20/2/23. Developed pleurisy of right base with high fever; fluid gradually appeared and 70 ozs were immediately drawn off on account of the respiratory embarrassment. Cough was very troublesome.

2/3/23. Readmitted to Sanatorium and was kept in bed till about 16/4/23 when the temperature had gradually come down to normal. She left the Sanatorium on 4th June 1923. At the time of leaving there was marked flattening of her left chest, a few crepitations at the apex, breath-sounds absent all over the lower lobe. A few crepitations were also present in the right inter-vertebral region, but expansion was pretty fair there.

24/11/23. She reported to me her further progress as follows: "Since leaving the Sanatorium in June 1923 I have never looked back. I have made steady progress. Weight on arriving home 8 stone 4 lbs. Last Spring and Summer I lost a little and at present I weigh 9 stone 19½ lbs, have had no cough, sputum nor temperature, and I have had no more refills, the last being given by you in June 1921. About two weeks ago I was examined by Dr. Turkington of Belfast and he told me that my left lung was quite collapsed and quite dry. He was very well pleased with my progress. The adhesions caused me a good deal of pain but I haven't any pain now - it seems to have gone. I am feeling splendid, leading a simple life and am busy every day housekeeping. I am able to do as much almost, if not altogether, as I did prior to
my illness. I haven't been able to keep up my regular rest-hours, but I take a rest in the afternoon whenever possible".

This case proved a very interesting and exceptional one and an extremely difficult one to interpret. I have never in all the literature on Artificial Pneumothorax seen a similar case quoted in which a rupture occurred and evidently closed up again immediately as had evidently happened in this case. But there it was and must be regarded as a possibility and in future one must exclude that condition of spontaneous Pneumothorax before arriving at a conclusion. Its diagnosis from an acute abdominal condition was extremely difficult. It is an extraordinary fact that the benefit from the Pneumothorax was only evidenced after that acute flare up and when the fluid developed.

Another interesting complication was the acute Pleurisy with effusion developing on the other side, which is also exceptional, at least I have never seen it nor have I seen it recorded. Luckily it did not recur after Paracentesis and the 'good' lung had not been sufficiently nor long enough compressed to prevent its complete or nearly complete re-expansion, so that it was able to carry on its functions. Immediate aspiration was the only chance of life in this case and on the slightest return of fluid it would have had to be immediately repeated.

Another interesting fact in this case is the weight
On admission she was 56.600 kilogrammes (8 stone 2 lbs) and after 12 weeks she was 67.800 - gain 11 Kilos or about 24 lbs. She kept round about that weight, but after the Pneumothorax started, in 7 weeks she lost about 16 lbs, being only 60.000 kilos then, and after her spontaneous Pneumothorax she lost a further 3.3 kilos (another 7 lbs) so that she was within a few ounces of her original weight when all signs of toxaemia had disappeared. That is a very usual thing in Pneumothorax and must be expected, but I do not think I have seen a case which has shown this so well.
CASE No. 5

G. McI. aet 18. Admitted 16/9/19, with a history of an attack of influenza at Xmas 1918 and a cough which had persisted since then. Haemoptysis May 1919, recurred three times, the largest being about a teacupful. On admission she has pyrexia, cough, and dyspnoea on the slightest exertion, and she was emaciated. Night sweats were present.

On examination, the right lung was found to be extensively involved, gurgling medium crepitations being made out on coughing at the extreme apex of the left lung. The temperature on admission was running between 37 and 38 C. and this continued with no abatement. Pulse-rate was increased to sometimes over 100 per minute. Cough was troublesome, sputum copious, and the patient was gradually losing weight. Even although she was kept in bed none of these symptoms abated so Artificial Pneumothorax was induced on 10/12/19. By the end of December right lung was collapsed, symptoms gradually abating, till in January temperature was normal, pulse rate decreased, cough and sputum nearly nil. She was allowed up and gradually given exercise. Unfortunately in March 1920 some activity recommenced in the left lung and we had to go very slowly with her exercise and also with the pressures in the right lung. She did not show any sign of toxaemia from the left lung until November 1920, when she had a definite attack of pleurisy at the left base with slight pyrexia to 37.6 C., and she
was kept in bed. Nevertheless her collapse was kept up and these signs abated and she was again put on exercise. She left the Sanatorium in April 1921 with a complete collapse of the right lung, a few post tussive crepitations at the root of the left lung and able to do from 6 to 7 miles daily without inducing any signs of toxaemia. She continued Pneumothorax at home being filled every month or five weeks, the pressure being brought to about +1 +4 each time and requiring anything from 800 to 1000 c.c.s of Air. Her Pneumothorax was discontinued in September 1922, but from January 1922 her refills had been getting gradually smaller and smaller and the intervals gradually longer and longer so that the collapse could be steadily withdrawn and recommended if any untoward happenings occurred. In her own words: - "After finishing with it I began to feel ever so much better and stronger in every way and put on a lot of weight and have now (November 1923) returned to a normal life, though careful. I take walking exercise and ride and now and again a little dancing which does me no end of good (?) as we do occasional sprees in the Capital". (Needless to say, she is Irish). "During the expansion process I had a sort of 'stretchy' pain a few times at the top of my lung, but it never lasted long and I didn't bother about it". In September 1922, her last refill, she had what I imagine to have been an embolism, probably due to the wounding of the almost distended lung. She describes her symptoms as follows: - "As soon as the needle was well in my arm went numb and I went
into a dead faint from which it took them half an hour's work to bring me round. It was the first of a series of faints lasting all that afternoon and night. In between times I was delirious and had rigors. They expected me to peg out at any moment. From the following morning I began to improve but had a pretty thin time for the next forty eight hours, especially at night, raging headache, eyes queer (seeing double and treble) deadly sick delirious now and again". A pretty picture of gas embolism.

The points to be gained from that case are.

(1). It was a clinically practically ideal case for Artificial Pneumothorax - the X-ray photograph showing practically no disease in the left lung, however, acute disease developed there and retarded the progress of the collapse very much. At one time in late 1920 it was sufficiently grave to make us consider the question as to whether it would not be advisable to stop the Pneumothorax.

(2). Even with these misadventures I consider it a very successful case and it showed an almost dramatic cessation of symptoms of toxaemia within a month. I feel convinced that if the process had been allowed to go on, the prognosis, both on account of her age and the extent of her disease would have been very grave and that collapse gave her her only chance.

(3). The wounding of the lung in the process of re-expansion and the causation of gas embolism teaches us to be
very careful of our punctures at that stage. I have no doubt that it was a true case of gas embolism from her description.
CASE No. 6.

Picture showing completely collapsed right lung.
CASE OF R.I.T. No. 6

R.I.T. age 23, admitted 19/6/20

March 1919 cough started but was not treated and it carried on till he had in December 1919 a haemorrhage of about one pint. T.B. diagnosed. In bed six weeks at home and then transferred to Foster Green Sanatorium, where he was 4 months in bed: he was then transferred here.

On admission. There was flattening of the right lung over the upper lobe with diminished expansion. Dullness extended to nipple line in front and to inferior angle of scapula behind. There were numerous fine crepitations all over that same area back and front with bronchial breathing and increased vocal resonance. Cough and sputum were present, the latter containing numerous tubercle bacilli. After a few days in bed he was allowed up to rest outside. September 6th he had a rise of temperature to 104, with about 80 c.cs haemorrhage, which was repeated daily in small amounts for about a week. Temperature gradually came down till in about 10 days it was running from 99 - 100, and remained at that level for about six weeks during which time he was kept in bed. He was allowed up and gradually put on exercise, but temperature never became satisfactory. He had frequent slight exacerbations necessitating his return to bed for a few days at a time and it was decided to treat him by Artificial Pneumothorax. At this stage the signs in the chest were much the same, only there
was distinct evidence of cavitation in the right upper lobe behind. March 5th 1921 Pneumothorax was attempted unsuccessfully. Acute pleurisy developed so had to wait its subsidence. March 9th space successfully found.

March 9th  
-12 -7  300 c.cs O₂
March 11th  
-10 -5  400 c.cs O₂
March 14th  
-8 -6  400 c.cs O₂
March 17th  
-5 -4  500 c.cs N₂
March 21st  
-5 -0  350 c.cs N₂
March 25th  
-5 -0  800 c.cs N₂

There was no reaction after these refills but temperature was still running between 98 and 99.6 so he was kept in bed although he was feeling much better. There was good collapse of the lung except at extreme apex where crepitation could still be made out.

April 2nd  
+2 -4  700 c.cs N₂
April 9th  
+10 -4  800 c.cs N₂
April 16th  
+8 -4  1050 c.cs N₂
April 25th  
+6 -4  600 c.cs N₂
May 3rd  
+4 +3  350 c.cs N₂
May 17th  
+2 +2  650 c.cs N₂

After the refill on 9th April he was allowed up and gradually given exercise till at the end of this period he was doing about 5 miles daily. Cough and sputum were both gone and collapse was complete except at extreme apex.

May 30th  
+8 +2  900 c.cs N₂
June 4th  
+8 +2  1000 c.cs N₂

Now doing about 6 miles and feeling well.
June 30th  -6 +4  900 c.cs N₂
July 15th  -6 +3  900 c.cs N₂
July 30th  -4 +2  800 c.cs N₂

Feeling well, doing about 7 miles, no symptoms
Apex still not collapsed, but no signs of crepitation.
Away for six weeks for a change or surroundings.

September 2nd  -2 +10  1000 c.cs Air
September 26th  -2 +3  1050 c.cs Air.

Now doing 7 to 8 miles plus putting golf and
feeling well.

October 10th  -4 -2  800 c.cs Air
          -6 +4
October 25th  -2 +3  1050 c.cs Air
November 11th  -2 +3  1000 c.cs Air

Patient was refilled at intervals of from 16 to
20 days and kept continuously well all Winter, doing a
very fair amount of exercise. He left on 28th April
1922 to proceed to another Sanatorium, there to contin-
uue his Artificial Pneumothorax and to commence some
occupational training.

This also is a somewhat plain-sailing case, but it
is one which teaches us this lesson, that where there
is a very elastic mediastinum one cannot push the pres-
sure of the refills as much as one would like.

January 1924. Extract from letter.

"I finished with treatment 14 months ago........
keeping quite well, and am leading an ordinary
life, having returned to work".....
CASE OF MISS B. No. 7


HISTORY. February 1920, pharyngitis and recurrent colds. June 23rd, 1920, haemoptysis, about teacupful Tuberculosis diagnosed and received here.

FAMILY HISTORY. Paternal Grandfather and Aunt died of Phthisis. 1 Sister died of Phthisis.

ON ADMISSION she had a slight cough with sputum in which tubercle bacilli were found. Pyrexia to 100 p.m.

SIGNS. Dullness and loss of expansion all over left side with crepitations practically all over.

After a few days in bed temperature settled and she was allowed up and gradually put on exercise. Her signs and symptoms improved and she gained 10 lbs in weight within 3 months. She had a small haemoptysis at the end of October and temperature became irregular so she was off and on in bed for about six weeks and cough and sputum increased. Again she improved and was put on to exercise. February 1921 signs increased, with cough, sputum and lassitude. Temperature was very variable running between 97.4 to 99.4 - occasionally 100. A few crepitations were now audible at the right apex behind. Fever increased to nearly 101 so she was again put to 'strict bed', and not allowed to go to bath etc. but was sponged in bed. There were distinct signs of cavitation in left upper lobe. May 1921. Haemoptysis, Pyrexia, signs in right chest few and stationary. July 1921. Cavitation, back and front of left apex, signs
CASE No. 7.

Skiagram taken on admission

Skiagram taken before treatment. Note increasing disease in both lungs - chiefly in left.
intense on left side, crepitations being audible practically all over back and front both on expiration and inspiration.

July 18th 1921. Artificial Pneumothorax attempted without success.

<table>
<thead>
<tr>
<th>Date</th>
<th>14</th>
<th>8</th>
<th>300 c.cs O2</th>
<th>16</th>
<th>6</th>
<th>400 c.cs and sputum.</th>
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<td>19. 7. 21</td>
<td>4</td>
<td>2</td>
<td>-14 -10</td>
<td>10</td>
<td>4</td>
<td>-14 -10</td>
</tr>
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<td>-10</td>
<td>-10 -10</td>
<td>-10</td>
<td>-7</td>
<td>500 c.cs O2</td>
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<td>-10 -10</td>
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<td>-10 -10</td>
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<td>700 c.cs O2</td>
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<td>-2</td>
<td>-11 -8</td>
<td>-8</td>
<td>2</td>
<td>-11 -8</td>
</tr>
<tr>
<td>9. 8. 21</td>
<td>2</td>
<td>-2</td>
<td>-12 -10</td>
<td>6</td>
<td>2</td>
<td>-12 -10</td>
</tr>
<tr>
<td>18. 8. 21</td>
<td>2</td>
<td>-2</td>
<td>-12 -10</td>
<td>4</td>
<td>2</td>
<td>-12 -10</td>
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</table>

Cough and sputum much less, able to be up a few hours. Temperature varied from 98 to 99. Mediastinum displaced. Left lung well collapsed.

<table>
<thead>
<tr>
<th>Date</th>
<th>14</th>
<th>4</th>
<th>500 c.cs Air</th>
<th>13</th>
<th>13</th>
<th>600 c.cs Air</th>
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<tr>
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<td>0</td>
<td>-12 -4</td>
<td>8</td>
<td>13</td>
<td>-5 -10</td>
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<tr>
<td>7. 9. 21</td>
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<td>-4 -4</td>
<td>6</td>
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<td>-4 -4</td>
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<tr>
<td>18. 9. 21</td>
<td>4</td>
<td>1</td>
<td>-13 -10</td>
<td>4</td>
<td>1</td>
<td>-13 -10</td>
</tr>
<tr>
<td>29. 9. 21</td>
<td>2</td>
<td>2</td>
<td>-10 -2</td>
<td>4</td>
<td>2</td>
<td>-10 -2</td>
</tr>
</tbody>
</table>

On September 19th she had a reaction of temperature but no symptoms. Evidently some pleurisy must have been present as fluid developed during this time as evidenced by the pressure at the next refill. Notice how quickly a positive pressure was attained at the refill on 29th with a small quantity of Air. The presence of fluid was confirmed by X-rays. On the 29th a higher reaction occurred and lasted till October 11th, since when temperature became normal. Bed during this time.
Lung collapsed. Increasing shadow in right lung - probably congestive.

Left lung collapsed. Cavity of upper lobe of right lung is absolute now: probable cavitation there.
Unfortunately now signs became more evident in right chest. No refilling was given till

<table>
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<th>Date</th>
<th>RR</th>
<th>PH</th>
<th>Amount</th>
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<tbody>
<tr>
<td>23/11/21</td>
<td>-10</td>
<td>+10</td>
<td>900 c.cs Air</td>
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Fluid was now to angle of scapula. Activity recommenced on left side due to adhesions having formed and preventing collapse. However, she became fairly well and was able to go away for a month as she had become rather nervous. On return on 25/1/22 fluid was found in a thin layer to R 3 in front. I say advisedly 'a thin layer' as there was only moderate displacement of the heart and crepitations were able to be made out faintly all over left chest. Right chest / S. Q

Pyrexia again present.

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<tbody>
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<td>+14</td>
<td>600 c.cs Air</td>
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<td>15/2/22</td>
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<td>+8</td>
<td>200 c.cs Air</td>
</tr>
<tr>
<td>24/2/22</td>
<td>-20</td>
<td>+14</td>
<td>300 c.cs Air</td>
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Air collapse of lower lobe obtained but not of apex.

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</thead>
<tbody>
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<td>+8</td>
<td>300 c.cs Air</td>
</tr>
<tr>
<td>27/3/22</td>
<td>-10</td>
<td>+8</td>
<td>250 c.cs Air</td>
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</table>

Pyrexia still present to 100 p.m. Activity progressive in right chest; larynx also becoming affected.

<table>
<thead>
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<th>RR</th>
<th>PH</th>
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<tbody>
<tr>
<td>27/4/22</td>
<td>-15</td>
<td>+4</td>
<td>170 c.cs Air</td>
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</table>

Pyrexia lower. Patient allowed up a little to encourage her as she was very nervous. Disease in right chest advancing.

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<th>Date</th>
<th>RR</th>
<th>PH</th>
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<tbody>
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<td>15/5/22</td>
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<td>+8</td>
<td>200 c.cs Air</td>
</tr>
<tr>
<td>25/5/22</td>
<td>-8</td>
<td>+8</td>
<td>500 c.cs Air</td>
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</table>
Left lung collapsed but excavation had commenced in the right lung. She had severe reactions after each of these refills so we had to give up hope and desist from refilling. She left us soon after to return home where she died early in July.

I have gone into the progress of this case in detail as it is very interesting and teaches a good deal.

(1). We missed our best chance by not doing Artificial Pneumothorax sooner as there were no signs in the right lung when admitted. If I had this case over again I should have given patient a month's rest in bed and induced Artificial Pneumothorax.

(2). Although I do not advocate withdrawal of fluid in every case, I think that in this case it should have been done and replaced with gas. By this means one could have had a more uniform and known pressure so as not to seriously incommode the good lung which was showing signs of commencing activity. In future I shall always advocate withdrawal of fluid when it gets to the level of the inferior angle of the scapula in cases where there is definite commencing disease of the other lung.

(3). By not withdrawing the fluid we allowed adhesions to form at the apex of the left lung (the site of the most active disease). We were not able to induce sufficiently high pressures to break them down as it would have caused more strain on the good lung and hastened the end. Perhaps if we had taken our courage in both
hands and done so at first it might have been better as then the main focus of infection would have been shut off and thus lessened the toxaemia and the acuteness of the spread in the other lung - at least she would have been able to cope with it better.

(4). This case shows typically the low pressures registered with fluid and the quickness it can be brought up by a small quantity of gas.

(5). We did try to collapse the left lung completely in March 1922 but I am afraid our policy was adopted too late.

(6). I have a feeling that more could have been done with this case, but it is an extremely instructive one. There were a lot of adverse conditions to be fought against. The prognosis wasn't good from the start on account of her age and family history, the acuteness of the disease and her evident low resistance. Her temperament was also much against her.
CASE OF R.C. No. 8.
R.C. age 24, admitted 7th April 1920

HISTORY. March 1919, Influenza with broncho-pneumonia. April 1919 demobilised and returned to his home in Glasgow, after two months there he still felt rather 'seedy' and cough and sputum returned.

July 1919. Tuberculosis diagnosed, sputum positive.

October 1919. Admitted to Sanatorium in West of Scotland, suffering from pyrexia, night sweats, loss of strength and weight. Cough and sputum much aggravated. In this month had haemoptysis twice. Great improvement by residence there and was able in December to do 4 miles daily.


April 1920 admitted here.

Nothing to note in personal or family history.

On admission. Cough and sputum present, also pyrexia which lasted about a week, but otherwise few symptoms.

Signs. There was flattening and deficient expansion of right chest with dullness to nipple in front and practically to base behind. Crepitations after coughing were present over the same area and there was definite excavation in the right upper lobe back and front. There were a few post-tussive crepitations in left lung behind in the inter-vertebral region.
Progress. After ten days in bed was allowed up but after two days he had a rigor at mid-day and exacerbation of temperature. Kept in bed for a week.


July 1920. Intensity and extent of signs increased Toxaemia on slightest exertion; bed again. As the patient had had practically eight months' treatment on general Sanatorium lines with no improvement it was decided to treat him by Artificial Pneumothorax, which was induced on 16th August 1920.

<table>
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There were no temperature reactions. Lung only partially collapsed at this time - still adherent over upper lobe. Cough and sputum were less and he said he felt better.

No signs of activity to be detected. No pyrexia: collapse more complete.
October 9th - 0 -5 600 c.cs + 2 +6
" 20th - 0 -5 600 " + 3 +7
Nov. 3rd - 0 -5 900 " + 3 +6

Great improvement in signs and symptoms.

Nov. 26th - 0 -4 800 " N2
Dec. 15th - 4 44 500 " N2

No physical signs beyond that of Pneumothorax

Jany. 7th - 9 -4 600 c.cs N2
" 27th -10 -6 800 "

Some re-expansion took place between these two refills with exacerbation of symptoms, showing that disease was not quiescent.

Feby 10th - 2 -8 520 c.cs N2
" 24th - 2 -9 700 c.cs Air

Keeping very well and on about 2 miles exercise. Heart easily displaced with refills.

Pneumothorax was continued at about 16 day intervals about the same pressures being observed. Signs became noticeable over right upper lobe and also increased slightly in left lung, but there was no systemic disturbance.

Progress continued satisfactory till August 1921 when he developed fluid in right chest with pyrexia, indigestion and general malaise.

October 1921. Fluid increased, indigestion much more troublesome and losing weight rapidly.

November 29th, 1400 c.cs fluid swarming with tubercle bacilli drawn off and replaced by gas 1625, c.cs
bringing the pressure to +4 +10, sharp reaction to 102 followed. Distinct improvement after this but there was definite increase in amount of adhesion so that on refilling pressure was quickly brought up by small amount of gas.

Jany. 4th, 1922. - 2 -7
   +10 +17 100 c.cs N₂.

Fluid increased but it was allowed to remain as it effectively collapsed lung. There were no systemic symptoms and patient felt very much better. Tubercle bacilli absent.


May 11th  950 c.cs fluid withdrawn  -22 -16
       950 "  Air introduced  + 2 +8


July 1st 1920. Patient left the Sanatorium.

Further progress. Continuing pneumothorax, 100 - 200 c.cs, high final pressures. Fluid gone. Fairly well but not fit for work. No systemic disturbance so long as on low exercise. Digestion great bugbear.

The after history of this case has not been a great success. He still requires refills every 3 - 4 weeks taking 150 - 200 c.cs air which brings his pressure to a very high level. His digestion proves the great bugbear to his progress. He writes to say that he feels well and keeps well so long as he does not do very much
exercise, but he has never been fit for work since leaving the Sanatorium. He has not required to be in bed at all. His chest contains no fluid and tubercle bacilli are still absent from the sputum.
CASE OF MISS M. No. 9.

E.A.M. age 31, admitted 25/10/21 with a history dating back to 1912.

1912. Slight haemoptysis. Tubercle bacilli found in sputum. Underwent six months treatment in a Hospital and convalesced in Isle of Wight for ten months. Returned to Hospital where she was training as a Nurse and finished her training, keeping very well till, 1914 when she came on to the Staff here. After ten months work she broke down and was six months under treatment here. 1915-1916-1917 nursing special cases. 1918 - 1920 Sister here. Resigned then owing to ill-health. Treated at Midhurst 1920 to March 1921. Was treated there by Pneumothorax but this was stopped after four injections as activity was found in the better lung. Then went to a Sanatorium in Scotland from March 1921 till admission here in October of the same year.

No history of any previous illnesses. Family history of no importance.

On admission left side was found to be extensively diseased with a large cavity in the upper lobe and crepitations all over on expiration and inspiration back and front. There were a few crepitations in the inter-scapular region of the right lung behind.

Symptoms were, troublesome cough, copious sputum, marked dyspnœa, slight degree of pyrexia with rapid pulse. She was kept in bed till February 1922 with no
improvement in the signs and only slight improvement in the symptoms, so we again decided to attempt Pneumothorax although we feared that the previous attempt might have jeopardised her chances. However, it proved successful.

Feb. 25th, 1922  
-4 -4  
+2 +2  
325 c.cs O₂

" 27th "  
-10 -10  
+4 +10  
400 c.cs O₂

Mar. 3rd  
-12 -10  
+6 +10  
400 c.cs O₂

These pressures taken in conjunction with the small quantities of gas able to be introduced showed us that only a small cavity was present and that we would only be able to get a partial Pneumothorax, however, we persevered and the lower lobe was collapsed.

Mar. 5th, 1922  
-10 -1  
+8 +12  
350 c.cs O₂

" 8th "  
-6 -8  
+14 +16  
450 c.cs O₂

" 11th "  
-12 -2  
+6 +10  
500 c.cs O₂

" 16th "  
-2 -8  
+6 +12  
400 c.cs O₂

" 20th "  
-8 -2  
+6 +6  
400 c.cs O₂

" 25th "  
-7 -6  
+8 +12  
400 c.cs Air

" 29th "  
-8 -6  
+8 +12  
300 c.cs Air

Apl. 4th  
-3 -8  
+12 +16  
400 c.cs Air

Patient was now getting up and out and felt better, cough and sputum were lessened. On examination breath sounds were absent over the lower lobe of the left lung but the moisture was still intense over the upper lobe both in front and behind.
During this period the area of collapse was becoming larger and the signs in the upper lobe thereby much diminished. Cough and sputum less, but still lassitude. Now doing 3 to 4 miles exercise daily. Refills continued to be given at about 8 or 9 day intervals to try to keep up a good pressure so as to undermine the adhesions.

Unfortunately in July 1922 signs began to appear in her right lung again and the symptoms becoming aggravated the refills were discontinued from 12th July till 4th August 1922 when she had a very small one:

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<tr>
<td>Aug 4th 1922</td>
<td>-4 -2</td>
<td>100 c.cs Air</td>
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She was kept at rest during this time

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<th>Pressure</th>
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<tbody>
<tr>
<td>Aug 23rd 1922</td>
<td>-9 -2</td>
<td>250 c.cs Air</td>
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given and refills continued at about 7 - 9 days' intervals but the patient was kept largely at rest until November 1922 when she was given exercise again. In January 1923 symptoms again became marked even with refilling, so she was put back to bed and was kept there until 26th April. During this time she was refilled every ten days or so with about 200 - 300 c.cs of Air, bringing the pressure up to +20 +22. So her story goes on till the present day but she has not required to be kept in bed from April 1923 and has been able during this time to do up to 5 miles a day. At the present
moment she feels well if she is kept on about that amount of exercise, but if given more becomes very tired.

This case is a very good example of what we find in Partial Pneumothorax. The patient is only able to take small amounts of gas but can stand the pressure being brought up to a high positive one. I have often brought her pressure to 426 +28 with no signs of discomfort and no signs of displacement of the mediastinum. When we got her as a patient the optimum time for successful Pneumothorax treatment had been missed. However, there is no doubt that, even at that late stage, she was benefited greatly by having a Partial Pneumothorax done, as her left lung was very extensively involved and very acutely progressive.

The mental factor played a great part in hindering her more successful progress as she is not well endowed with this world's goods, and I am afraid that dread of her future (if she were not quite fit to work for her living) has made her tend to exaggerate her symptoms. I am quite sure if there had not been that factor present, she would have done much better and that that improvement would have been admitted quite 12 to 18 months ago. Even with that factor present she has had to admit she is much better now but will not admit it sufficiently to allow us to recommend her leaving the Sanatorium.
CASE OF H.S.L.W. No. 10

H.S.L.W. was admitted to the Sanatorium on 18th June 1920 with a history dating back to the previous Summer when he complained of lassitude, but he attributed that to overwork. In November 1919 he had pyrexial attack and when temperature came down he went to Switzerland for a holiday. There he had exacerbation of temperature and had to stay in bed. Hoarseness developed. He had various exacerbations in the Spring of 1920 and was finally recommended to come here.

On admission the physical signs showed definite acute disease over the right upper lobe and at the base of the same lung behind. The larynx showed definite infiltration but no ulceration. After about 10 days in bed, the temperature having settled, he was allowed up and put on gradual exercise till he was doing about 7 miles a day. After about 2 months exacerbation occurred and he was 14 days in bed, then two weeks up on mild exercise and then again bed for nine weeks with irregular pyrexia. After being up for about three weeks he developed an ischio-rectal abscess, requiring operation. He was on rest for 13 weeks after that, temperature still remaining at 100 or over at night. His weight kept up but he was feeling ill. After this long spell of treatment with no amelioration of signs nor symptoms - in fact they had become aggravated - it was decided to try Artificial Pneumothorax although it was doubtful if a space would be found, as there had been several attacks.
of pleurisy during his residence. However, it was successful at the first attempt on 7th March 1921.

March 7th  -12 -8  200 c.c.s O₂
        "  9th  - 8 -4  300 "
        " 12th  -10 -6  300 "

After these three refills temperature dropped to over 99 before which it had been running about 101 for some time.

March 16th  -10 -4  350 c.c.s O₂
        " 20th  -12 -3  600 " N₂

After this refill exacerbation of temperature probably due to reaction as a larger amount of gas had to be introduced.

March 26th  -12 -5  800 c.c.s N₂ Temperature again settling
        April  4th  -12 -1 (ature again settling
        "  8th  -10 -2  450 c.c.s
        " 18th  -10 -4  550 c.c.s N₂
        " 27th  -14 -0  900 "

Temperature had remained satisfactory till after the refill on 27th April when it rose to 101 and he had symptoms of pleurisy. On May 4th the temperature rose to 103 - 104 and remained between 102 and 103 for over a week and gradually fell by lysis in about three weeks. Fluid meanwhile had developed and was keeping up the collapse. It became troublesome and the heart and liver were displaced and dyspnoea pretty marked so pressure was taken and fluid were withdrawn and 300 c.c.s O₂ given, leaving a pressure of
-0 +6. Reaction followed to 102.6 but subsided again in a day or two.

June 19th 1921. Again tightness developed so 700 c.c.s fluid withdrawn and no gas given as pressure was satisfactory. No reaction followed and temperature remained steady and patient expressed the opinion that he had not felt better for months. Fluid was now up to the inferior angle of scapula with gas above and compression of the lung was satisfactory, so as no untoward symptoms arose it was left at that and patient made steady progress and was able to do a certain amount of exercise. He left the Sanatorium on 8th September 1921. He then returned on 13th October 1921 and was found to be in the same satisfactory condition - lung still well collapsed by fluid and the throat healed. No tubercle bacilli in sputum. He left the Sanatorium on April 1922 able to do 6 - 7 miles walking exercise daily and feeling very well, except for some shortness of breath. Progress was uninterrupted and he was leading practically a normal life until April 28th 1923 while playing golf he got wet through and temperature shot up to 104 and it remained up for several weeks, the patient showing few symptoms except malaise. There were no symptoms which would point to a pulmonary condition, except a slow and gradual displacement of the heart to about ½" beyond its previous site and also a slow but distinct pushing down of the liver dullness and liver border. When one remembers that these organs were already not in their usual
place, one can realise the difficulties presented in making a definite diagnosis. An exploratory syringe was introduced and some white turbid fluid withdrawn, thus clinching the diagnosis of pyo-pneumothorax. To X-rays when he was screened before leaving the Sanatorium the right chest was entirely black and one thought that it was due to thickened pleura and not to fluid, but I am convinced now that the right chest had been full of fluid from the time of his pleurisy in 1921 and that the nature of the fluid was gradually changing and becoming more and more purulent until as an after effect of the severe chill the patient's resistance absolutely broke down and an acute pyo-thorax was set up. Withdrawal of fluid was performed on three occasions with gradual lowering of the temperature.

June 1923. Temperature again became high and he looked hectic. Fluid withdrawn on two occasions with not much relief of symptoms - on the third occasion it became too thick to be withdrawn by aspiration so he was operated upon and a rib resected. At the operation the lung was seen to be collapsed and hardly moving, but the pleura was studded with tubercles all over.

The after history of this case is the usual one of continued hectic fever, gradually increasing myocarditis but since Christmas things have taken a turn for the better and his temperature is much lower, only going to between 99 and 100 each evening. Discharge is much less and general condition much better. He had several times
been at death's door since his operation in July. The large cavity in his chest will probably go on discharging for the rest of his life unless his general condition improves sufficiently to allow an Eslander operation being done, i.e. resection of all the ribs and subsequent falling in of the chest wall on collapsed lung.

Pneumothorax in this case certainly saved this patient's life in 1921 and gave him nearly two years of a happy practically normal existence. Unfortunately he developed the pyo-pneumothorax in 1923 and the extraordinary thing about it was the suddenness of its onset as he had no symptoms of any gradual malaise or lassitude before. Perhaps the sudden onset of the pyo-pneumothorax would never have developed if we had recognised that the opacity to the X-rays had been diagnosed as fluid instead of thickened pleura and the fluid had been drawn off. But his excellent health rather tempted us to let well alone.

Another unfortunate thing was the fact that aspiration was no longer practicable after July - the fluid seemed to become coagulated.
M.M. admitted 1/5/22, age 23, Nurse.

HISTORY. May 1920, haemoptysis. Lived in country for four months afterwards with little or no improvement, was then sent to a Sanatorium for six months and improved.

In November 1921 patient came here as Nurse and in the following month had severe haemorrhage. Treated here for six weeks and then sent to lodgings in the Village. As she was obviously not making progress she was readmitted here on 1st May 1922 for Pneumothorax.

Nil to note in previous history or family history. On admission there was flattening with loss of expansion on the left side, and dullness to R 5 in front and to inferior angle of scapula behind with fine crepitations over practically the same area. On the right side there were a few post tussive crepitations at the apex behind. Cough and sputum and pyrexia to 100 p.m. were present. Pneumothorax induced.

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June 21st 1922 -8 -3 550 c.c.s Air
" 30th " -7 -4 500 "
July 13th " -6 -4 550 "
" 25th " -8 -6 500 "
Aug. 8th " -7 -1 550 "
" 25th " -10 -2 500 "
Sept. 15th " -7 -2 700 "
Oct. 10th " -10 -2 500 "

By the sixth refill collapse was complete. She had no reactions after refills, but for a day or two cough and sputum was increased. She was allowed up by the end of May and her exercise was increased gradually till when she left on November 6th she was doing about 6 miles a day. On leaving here she entered another Sanatorium as dispenser nurse and she has been on duty since, being refilled about every 3 weeks and she has had no further trouble and feels very well.

We gave very small refills and did not bring the pressure to a high level in this case as she had a very easily displaced mediastinum and it caused some collapse of the 'good' lung, and also a good deal of dyspnoea if the pressure was brought too high.

This is a perfectly plain straight-forward case, but it shows the benefit of Pneumothorax in hastening the cure in one who required to earn her living. She had a trial for six months of ordinary Sanatorium methods with little or no improvement in the physical signs or symptoms, yet with Pneumothorax treatment she
was able to return to full work within a further six months. Of course she was fortunate in securing a very suitable post as she could there live under favourable conditions and continue her Pneumothorax treatment.
CASE OF R.S. No. 12

R.S. age 16½, admitted 12/3/21 with the following history.

January 1921 consulted Doctor about cough, gastric disturbance and hoarseness of voice which had been present for nearly two months and had gradually got worse. He also suffered from increasing lassitude.

February 1921 sputum became worse and on examination was found positive to tubercle bacillus. During this month pyrexia developed, normal in the morning but up to 101 at night (oral). No history of any previous chest trouble. One sister died of Tuberculosis in 1918.

On admission. Dullness was noted to R 4 in front of the left chest and to the inferior angle of the scapula behind with crepitations after coughing practically over the same area. On the right lung there were a few post tussive crepitations over ribs 2.3.4 in the mammary line, but no dullness. There was dullness in that chest to the junction of middle and lower third of the scapula but no crepitations. Throat showed general pallor, epiglottis was infantile in type. Both true and false cords were congested and the true cords showed an indefinite ragged inner margin.

This case was diagnosed as one of bilateral Pulmonary Tuberculosis of a catarrhal type, the more acute being in the left lung, cords definitely affected, toxaemia of moderate degree. Prognosis was guarded on account of the extent of the disease, age of the patient
and the fact of his larynx being involved, also on account of the short duration of the disease. He was treated by typhoid rest and absolute silence till 8th June 1921 when he was allowed to sit up. Pyrexia had come down to 99.6 p.m. but the physical signs were much as before, in fact at this time there was evidence of caseation under the left clavicle. On 18th July 1921 he was allowed up and by August was allowed on exercise which was gradually extended. By this time he had gained 12 lbs in weight. By December he was doing about 8 miles a day without any signs of toxaemia. Xmas 1921 he was allowed to go home for a holiday and he developed a 'cold'. He returned to the Sanatorium on January 4th and had a sharp attack of pyrexia 102 with increased cough and sputum and slight dysphagia. Temperature came down to 99.4 in two days but continued at that level. There was distinct intensity in the signs in the left chest: he lost about 7½ lbs in weight in 3 to 4 weeks. Pulse-rate was accentuated so that there was no doubt we were dealing with an acute or sub-acute exacerbation. He was kept in bed till early in March - after being up a week on short walks he developed left sided pleurisy with pyrexia to 101. It settled down in about a week to 100 but then he had two or three rather sharp stainings of sputum. There was no abatement of toxaemia by May 9th and the signs in the left chest were again becoming more intense. The few crepitations in the right lung which were present on admission showed very little change all
### Case RS, No. 12

**Date**

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| ORAL | 97 | 98.5 | 98 | 97.5 | 97 | 97.5 | 97.5 | 97 | 97 | 97 | 97 | 97 |

**Pulse**

Chart showing highest and lowest temperatures recorded each hour before and after.

**Respiration**

Presently normal. Reflexes shown in lower.
through his treatment but they had never become worse
so Artificial Pneumothorax was decided on. The larynx
was still involved but rather more quiescent. Artifi-
cial Pneumothorax induced on 25th May 1922.

May 25th  -10-6
" 28th  -10-6
" 31st  -11-5
June 3rd  -10-2
" 6th  -11-5
" 10th  -10-6

By this time the lung was half collapsed, cough and
sputum much less, pyrexia gone. He was now allowed up.

June 15th  -17-7
" 20th  -10-6
" 28th  -5-4

Now doing about 2 miles daily, lung completely collapsed.

July 6th  -6-0
" 17th  -8-4
Aug. 1st  -8-2
" 19th  -0-3

Now doing about 5 miles daily with no signs of toxaemia.

Sept 8th  -6-3
" 30th  -6-3

He left the Sanatorium 7/10/22. 8/1/23 he returned for
examination, still kept well and refills had been done
about every 3 weeks to the same pressures as indicated.

Jany 1st 1923 -12-3
3/5/23 Returned for examination. Everything was found
satisfactory, and patient was living practically a normal life. 18/7/23. Still satisfactory, by this time a small quantity of fluid had developed. Since then he has been working on a farm and by me I have a letter dated December 10th 1923 in which he says:— "I have refilled every 3 weeks with quantities varying from 500 to 700 c.c.s and kept very fit till Christmas 1922 when I came up to you to be refilled. At that time you found my lung considerably expanded so that when I returned the amount of my refills went up to 1000 c.c.s with a maximum pressure of about plus 4: the previous interval remaining the same. During January and February 1923 I did not feel very fit. I kept getting higher temperatures of about 100 to 101 and had a cough, but by the end of February I started to pick up. Since May 1923 the lengthening of the interval between the refills gradually increased and since then it has gone from 3 weeks up to 7 weeks. During the whole time I have kept in tip top health. During the Summer holidays I rode and fished and played cricket, and I have ridden my motor bicycle many thousands of miles, doing three trips each of over 1000 miles. In September I came here to learn farming and stock breeding. I am keeping awfully fit and I think this life will suit me down to the ground. I only hope I shall be able to keep it up."

I have quoted this case rather in extenso, as I think it is rather an interesting example of what so often happens to our Sanatorium cases. The patient comes
in with acute disease and after several weeks or months in bed the disease settles down and he is able to be on exercise. Some slight intercurrent affection occurs with its slight lowering of bodily resistance, and the result is that the disease lights up and continues as a very acute exacerbation with probably fatal results. Or, as in this case, may continue as a sub-acute exacerbation which necessitates rest in bed for months, and with the outlook of invalidism for the rest of the patient's life, and it is only the induction of Pneumothorax which prevented this same sequel in this patient's case. Also I quoted in detail, as it is a typical of what one might expect from Pneumothorax treatment in a plain sailing case as this proved to be. Gradual increase of pressures and intervals of refills, with a gradual collapse of the lung and lessening of toxic symptoms. It shows also how the small amount of disease in the better lung is no deterrent and that this tends to clear up. Tubercular laryngitis, as in this case also is no contra-indication. I quote from a letter from his Doctor dated 20/9/22:

"I hesitated sending this patient to you as I was afraid he was too far advanced for Sanatorium treatment"
CASE OF E.G.W. No. 13

E.G.W. age 28th admitted 22nd March 1921.

HISTORY, dated back to June 1920, when he had increasing lassitude, expectoration but no cough.

Early December 1920, felt vague pains in both chests, but carried on till Christmas, when pains became much worse and he felt very ill. Consulted Doctor who diagnosed pleurisy. After a week in bed and a fortnight's convalescence in Bournemouth he was advised to go to Switzerland where he went in for all the gaieties to be found there. February 1921. Returned to England and 2 days later developed very severe pain in left shoulder, worse than ever before. Sputum examined for first time and found positive. Advised Sanatorium treatment.

PREVIOUS HISTORY. Nothing of note.

FAMILY HISTORY. Bad. Mother, chronic invalid from Tuberculosis. One brother died in 1913 from Tuberculosis whilst another brother has spent the last 14 years in Sanatoria.

PERSONAL HISTORY, also was against him, as he had lived a very gay life in London.

On admission cough was present with varying amounts of expectoration. Pyrexia was present, lassitude was easily induced. There was loss of weight and his digestion was easily upset. Crepitations were present in both upper lobes back and front. Consolidation was not very marked. The signs showed that it was probably a spread outwards from the Hilus. After admission he was
only ten days in bed, as he was temperamentally very restless and it was difficult to make him understand the advisability of rest. May 6th 1921. Attack of pleurisy on left side, put back to bed, digestion troublesome, general malaise present, no marked pyrexia. June 13th 1921. Attack of pleurisy widespread on right side temperature rose to 103.6 which gradually fell by lysis. The physical signs at this stage were not much more marked than on admission, except that there was marked loss of expansion on the left side with very weak breath sounds. His temperature remained very variable ranging between 97.6 a.m. and 99.6 p.m. The digestion was very troublesome, cough and sputum increased and lassitude marked. August 1921, although temperature was not quite settled, he was allowed up again as he was getting very restless in bed. By October he was on exercise to about 2 miles a day. November 6th 1921 recurrence of left sided pleurisy with pyrexia necessitating bed. At this time he developed a subacute prostatitis, from an old gonorrheal infection and as this might be a source of secondary infection tending to keep up the temperature he was sent to Edinburgh for treatment of this complication. During his stay there he had a definite attack of left sided pleurisy with high temperatures, night sweats, increased cough with staining of sputum and general malaise. He returned here on 10th December looking very ill. He picked up again and was able to be on short exercise
1st February 1922 return of pyrexia and left sided pleurisy. 17th February signs in the left lung were now much more intense and there was definite evidence of breaking down of the left lower lobe with moist crepitations, bronchial breathing and dull note on percussion. Pyrexia present. He was kept in bed with the signs getting gradually worse, so Artificial Pneumothorax was decided upon and successfully induced on 18th March 1922.

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Three weeks after Pneumothorax was induced patient
CASE No. 13.
X-ray showing almost complete collapse.
showed few or no signs of Toxaemia, cough practically gone, sputum almost nil and he felt better than he had done for years. There were no untoward events and no reactions after the refills. He left the Sanatorium on 26th May to live outside and returned periodically for refills. After the refill on July 20th patient caught a chill, had vague chest pains and a slightly higher temperature than usual (up to 99 p.m.) and on examination a slight amount of fluid was detected in the left costo-diaphragmatic angle. This toxaemia cleared up after a little rest.

Aug. 5th  
-2 + 2  
+4  
625 c.cs Air

" 22nd  
-4 + 3  
-9 +8  
460 "

Fluid increased reaching to about 1½" from the inferior angle of the scapula.

Sept 15th  
-4 +4  
-1 +6  
400 c.cs Air

He left the district at this time almost able to do 6 miles a day and feeling very well. Fluid still present but causing no inconvenience. He returned to his work as an Accountant in London in October 1922 and has remained at it ever since, being refilled at intervals of about five weeks. I saw him again in August 1923 and he continued to feel well and there was no sign of any activity in either lung. There was still about 2" of fluid at the left base. The interesting points about this case are:

(1) Although bilateral at the start the right side settled down under ordinary treatment and the acute
advance localised itself to the left lower lobe.

(2). That after repeated definite attacks of pleurisy a free space was found and complete collapse was able to be attained - one felt rather doubtful about it but no difficulty was experienced.

(3) The dramatic cessation of symptoms in a very short space of time after collapse: an industrial patient who might have been the mainstay of his family could easily have been able to return to moderately heavy work within three months of the induction of Pneumothorax.

(4). Evidently this patient's resistance was of the lowest degree as the slightest attempt to give him exercise ended in an exacerbation of the disease.

(5). That although to my knowledge this patient is leading a most unphysiological existence from a consumptive's point of view, it seems to do him little or no harm so long as his lung is collapsed.
CASE OF J.B.P. No. 14

J.B.P. age 25. Admitted 31st October 1921.

HISTORY. 1919. Invalided from Army on account of wounds. Had nine operations to leg with general anaesthetic each time. October 1919, took up work in a Bank in London. June 1920 - April 1921 at home in Fife suffering from debility (?). August 1921, cough developed, consulted Doctor who diagnosed T.B. and advised Sanatorium treatment. October 1921 admitted here.

PREVIOUS HISTORY. Nil to note except severe gunshot wounds of left thigh damaging sciatic nerve.

FAMILY HISTORY. Nil.

On admission patient looked ill, nutrition poor, cyanosis marked, cough troublesome but sputum of small amount. Night sweats were present.

Examination of Chest. Right lung. Marked loss of expansion and dullness to nipple in front and practically all over behind. Medium crepitations to nipple in front and to inferior angle of scapula behind. Cavitation in upper lobe front and back. Left lung. Dullness over upper lobe front and back with crepitations on coughing over same area. There was very little pyrexia present but pulse rate was accelerated. On account of extent and intensity of signs and mal-nutrition patient was kept in bed.

January 16th 1922 allowed up for a few hours. By this time signs had cleared in left chest and were materially better on right. He had gained about 8 lbs in weight and felt much better. During February and
CASE No. 14.

Skiagram taken before induction of Pneumothorax.
Ring indicates left side.

X-ray showing incomplete collapse. Notice adhesion in centre of lung pulling tongue-shaped process out. Apex not collapsed.
March he was up practically continuously with occasional spells in bed due to exacerbations of temperature. On the whole temperature kept fairly normal but he again started to lose weight, cough and sputum became increased and he wasn't quite so well as he was on getting up. The signs in the right chest were much the same and there had been no recrudescence in the left.

May 1922. Condition much the same. General condition poor and any attempt to push exercise only caused increased toxaemia. Pneumothorax treatment was decided on as he had been over six months under general treatment without improvement.

May 30th 1922 -16 -6
-10 -6 300 c.c.s O₂

This was followed by a marked reaction of temperature, going up to 102.2 but falling again in three days.

June 3rd -8 -4
-4 -2 300 c.c.s O₂. No reaction.

" 6th -10 -4
-6 -0 500 "

" 9th -6 -2
-6 -2 250 "

Evidently we were dealing with a fairly moderately sized space as the pressures went up quickly with not much gas. Signs had disappeared from right lung which was about two thirds collapsed. No collapse over the cavity.

June 15th -7 -3 650 c.c.s Air +2 +4

" 20th -6 -0 400 " +1 +3

" 27th -8 -3 700 " +1 +2

July 5th -3 -0 500 " +2 -2

Non-toxic now.
July 14th  

-6 -2  

-1 +3  

500 c.cs Air

Patient now doing about 2 miles in the morning and the same in the afternoon and was feeling distinctly better. Tubercle bacilli absent from sputum for the first time.

July 20th. Developed pyrexia with vague pains over right lung - small amount of fluid developed. Temperature remained up for nearly a fortnight. August 12th nearly a month had elapsed since last refill due to pyrexia and pleurisy.

Aug. 12th  

-3 -0  

+3 +8  

300 c.cs Air. Note the higher pressures on entering, due to the presence of fluid, and the small amount of gas required to bring pressure to a positive one.

Aug. 27th  

-2 -4  

+6 +8  

100 c.cs Air

Sept 21st  

-7 -4  

+7 +9  

200  

Doing 5 to 6 miles daily. Fluid much increased; about inferior angle of scapula. Adhesions had formed at level of root of lung and extended to chest wall. See sketch.

Oct 10th  

-10 -1  

+8 +13  

150 c.cs Air

Nov. 14th  

-18 -12  

+8 +10  

500  

" 25th  

-14 -6  

-9 +8  

250  

Patient did not feel quite so fit, digestion troublesome, pyrexia easily induced, cough and sputum more. I think this was due to the collapse of the right lung not being so good owing to the above-mentioned adhesion.

Dec. 7th  

-16 -8  

+4 +6  

260 c.cs Air

" 16th  

-14 -10  

+6 +10  

220  

" 30th  

-10 -4  

+10 +12  

210  

Fluid absorbing.
Jany. 13th, 1923  -10  -4  +10  +12  250 c.cs Air
"  31st  "  -10  -4  +12  +14  250  "

Condition not at all satisfactory. Back to bed again. Temperature always unsettled and digestion troublesome, due probably to bad collapse of right lung; also there were a few signs appearing in the left lung behind and both testicles became infected.

Feb. 23rd  -10  -4  -0  +2  250 c.cs Air
Mar. 15th  -12  -4  +2  +6  200  "

Acute reaction to 10J - 6, which continued for three to four days. April 1923 both epididymes removed. Caeation had taken place, and pyrexia continued to a light degree after his return from operation, although wound healed by first intention. Digestion very troublesome. Pain and tenderness over right iliac fossa - probable infection of abdominal glands.

April 18th  +2  +6  No gas given
May 29th  -9  -2  +10  +14  125 c.cs Air

Temperature still up with the same abdominal symptoms.

June 26th  -12  -6  +10  +12  210 c.cs Air

Fluid greater in amount. August 1923 patient began to feel a little better - no further refills - abdominal pain and tenderness tending to settle down. Able to be up again. By September he was able to do 3 miles daily.

Sept. 14th  +10  +14  375 c.cs Air

His improvement continued all through October and November.

December 10th.  -18  -12  +7  +9  425 c.cs Air
He kept well till January 19th of this year when

temperature rose so refill given on :-

Jany, 21st 1924  -14 -8
    +12 +14  150 c.cs Air
Temperature settled rather better after this and he
was able to be up again.

Feby 19th  -12 -6
    +30 +32  200 c.cs.
Temperature since then has remained high, digestion
again has become troublesome and patient feels very ill.

SUMMARY. One cannot quote this as being an entirely
satisfactory case of Artificial Pneumothorax, but I
do not think the treatment is entirely to blame. In
the first place the soil was bad as is evidenced by the
very slight reactionary power after admission. During
the course of the treatment he easily developed pleur-
isy with effusion, adhesions formed and prevented com-
plete collapse of the lung. Nevertheless the signs
cleared up and symptoms improved even by the partial
Pneumothorax and all might have gone well if infection
of the testicles had not taken place: that gave him a
set-back and he was pretty ill after the operation. He
was recovering from that when another complication
arose in the digestive symptoms and I am quite sure
that there was some tubercular mischief of the appendix
or caecum, or the glands in that neighbourhood. Oper-
ation was not performed as the general condition was
bad and after that the local condition settled down
and he became rather better than he had been since ad-
mission. Around Christmas 1923 he became worse and
the question arises now what is the condition which is
giving rise to his symptoms. Although there are no
definite indications at the moment I am inclined to the
opinion that it is the abdomen which is at fault again,
as his lungs do not show any signs of being any worse.
The right lung is fairly well collapsed and shows no
crepitation. The left lung is clear. True, there is
a small amount of fluid in the right pleura and before
coming to a conclusion one must explore this to see if
this is becoming pustular. I think not, as in that
case it would probably have increased (at the time of
writing it only extends to within 2" of the inferior
angle of the scapula). Again, there is no definite
indication to interfere with the abdomen.

It is an extremely interesting case and gives one
much room for thought. The lessons we learn from the
point of view of Collapse Therapy are:—

(1) The signs of a small pleural cavity.
(2) The signs of the early development of fluid.
(3) The quickness with which adhesions may form
even with collapse.
(4) The quick delay in the absorption of gas after
pleurisy develops.
CASE OF MRS. P.K.  No. 15

Mrs. P., age 25, admitted 25th March 1921.

HISTORY. September 1920 cough started and persisted all winter, with small amount of sputum, nothing done. February 10th 1921, haemoptysis, about egg cup-full, not kept in bed at all but went about her usual duties. Lassitude commenced and has gradually increased. Advised to come here. Nothing of note in her family history. On arrival she was found to have a temperature ranging to 100 at night.

Physical signs. I shall go into these in some detail as they are very interesting and show a very typical case of Hilus Tuberculosis gradually spreading to the surface. On admission one would have thought that it was a practically normal chest. Inspection, nothing to note, no flattening nor loss of expansion. Percussion, no dullness. Auscultation. Breathing was vesicular all over and there were no adventitious sounds to be made out at all. There was evidently an open lesion somewhere as tubercle bacilli were present in the sputum. Kronig's area 1 1/2" each side. Tidal expansion equal on both sides and normal in extent. X-rays showed diffuse mottling all over both sides of the chest, particularly marked on the right side, some thickening of right pleura.

Her symptoms improved after three weeks in bed and she was allowed up on exercise and this was gradually increased. She gained weight at a great rate. At her first examination in May 1921 a few fine crepitations
were found after coughing over the right side and extending into the right axillary region, but no signs of toxaemia appeared. This was evidently a spread towards the surface because at an examination three weeks later these crepitations were audible practically all over the right lower lobe back and front. Her exercise was continued. With the exception of a slight attack of pleurisy in the right axilla at the end of June her progress was uninterrupted and she was able to leave the Sanatorium on 24th October 1921. The signs in her chest had considerably abated, no toxaemia, no cough nor sputum. She had gained over two stones in weight. At the time of leaving she was able to do 6 to 8 miles daily without inducing any fatigue or signs of toxaemia. She was re-examined in December and the signs were found to be practically nil.

April 17th 1922. Patient readmitted with marked intensification of signs in the right chest. She had felt a return of her lassitude and cough and sputum early in March after having nursed her mother through an attack of influenza for about five weeks. Pyrexia again present to about 100 p.m. but this gradually subsided after a few days rest. However, as her disease had recurred and was so completely localised to the right lower lobe we advised Artificial Pneumothorax and this was commenced on May 10th 1922.

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May 13th  -10 -6  250 c.c.s O₂.
" 15th   -10 -6  300 "
" 17th   -10 -6  450 "
" 20th   -5 -5  600 "
" 25th   -5 -3  950 "
" 29th   -3 -1  1200 " (up after this refill)
June  3rd  -6 -0  1000 " on 2 miles exercise daily)
" 10th   -2 +2  900 "
" 21st   -4 +2  950 "
July  3rd  -3 +3  1000 "
" 18th   -7 +2  900 " (5-6 miles exercise)
Aug.  3rd  -0 +2  700 "
" 13th   -6 -2  900 "
Sept. 9th  +1 +4  1200 "

Patient was now able to leave the Sanatorium and returned about once a month for refills, which were kept up at about the same level and about the same amounts of air. She had her last refill on 9th May 1923 and was again allowed to proceed home. She returned to us on July 2nd and we found no expansion of the lung. We kept her under observation giving her exercise to nearly 10 miles daily without any toxaemia - the lung was then allowed to re-expand. 13th October 1923, lung fully expanded - very well. 4th January 1924, very well, leading ordinary life.

The points of interest in this case are:-

(1) The method of its spread as described in the text of the case.
(2). The ease of induction of Artificial Pneumothorax in a case of this type and the completeness of the return of function after the Pneumothorax had been in existence for such a short time. Unhappily a case of this type is only too rare owing to the difficulty in diagnosis and it is generally at a much later stage of the disease that patients come to us, but many must have passed through such a stage when this treatment could have been adopted.

I consider this case is an excellent plea for adopting this procedure, before the disease becomes too far advanced. No doubt this patient may have done well enough under ordinary Sanatorium treatment, but I am quite sure it would have taken twice the time. Her disease was as completely arrested in October 1921 as one can ever say this disease is arrested, no cough, no sputum, no symptoms; yet she had relapsed by March 1922. By October 1922 she was able to proceed home and lead an ordinary life and thus she has continued for over a year.

(3). Under ordinary Sanatorium treatment this patient gained over 28 lbs. On re-admission in April 1922 her weight was 67 kilogrammes and on leaving in October it was 65 - a loss of 4 lbs.
CASE OF G.W.N. No. 16

G.W.N., age 29, admitted 8th April, 1921.

HISTORY. Cough and debility dating from October 1918 when on leave from France. Although his cough persisted all the time he felt well with occasional periods of ill-health, but T.B. not diagnosed until March 1921 when he was found to have pyrexia. On examination his sputum was found positive. He was treated by rest in bed and while there he developed pleurisy on the right side.

On examination. There was extensive disease of the right lung with marked consolidation in the upper lobe. Crepitations were present back and front probably all over this lung. There were no signs of any disease in the left lung although the root shadows were marked to the X-rays. Cough was very troublesome and sputum copious. Pyrexia ranging from 100 to 101 p.m. was present. He was treated by complete rest in bed with no improvement in the physical signs or in the symptoms - in fact excavation was commencing in the right upper lobe behind. He had two attacks of pleurisy in the right side.

June 1921. Signs the same: a few crepitations appeared in the left axilla.

July 1921. Temperature tended to go higher, up to 103 p.m. and symptoms and signs became more marked. Skiagram again taken with a view to Pneumothorax, and even in the short space of time which had elapsed since the first skiagram was taken, there was evidence
of much more extensive consolidation. The left side looked fairly clear and the physical signs there appeared sufficiently negligible to attempt pneumothorax although there was definite evidence of disease there.

July 20th 1921 Artificial Pneumothorax induced successfully.

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Fleurisy developed on left side when the right lung was partially collapsed. Refills were abandoned to allow this to settle

Aug. 23rd  -18 -6  500 c.cs O₂
Sept 2nd Needle introduced but failed to find pleural space.
" 9th      """"""""""
" 20th     -12 -6  180 c.cs O₂
" 25th     -16 -4  150 "
Oct. 3rd Needle introduced -1 +1 and no gas given.

During this time pyrexia was slightly less, reaching 101.6 p.m. The left sided pleurisy had not recurred and there was a slight degree of collapse of the right lung with some lessening of the intensity of the moisture. The patient was markedly dyspnoeic and cyanosed. He was much too ill to attempt to X-ray him so
as to attempt to elucidate the problems which had been raised in these preceding refills. On looking back at this case I think there must either have been fluid present (although there were no clinical signs of this) or that adhesion had taken place between the pleurae in one or several places. Probably the latter is the correct solution as fluid would not account for the two failures on 2nd August and 9th September to obtain a space. Then on the succeeding attempts a space was found, but the pressure was very quickly raised to positive by the introduction of a very small quantity of gas. This would occur with fluid but is not incompatible with the alternative theory. Besides if there had been fluid there would have been little difficulty in detecting it and we would have attained a greater degree of collapse.

At this stage Pneumothorax was abandoned. 10th November 1921 acute pleurisy on left side with another attack on 21st. Crepitations were now present practically all over the left side in front. Cavitation was present in right upper lobe and signs of softening at the apex of the lower lobe. Patient died on 27th, January, 1922.

I quote the case as there are many lessons to be learned from it. In the first place it was a case in which I am sure Pneumothorax would have been successful if the optimum time for its performance had been taken. As it was I am afraid it was well past when we
attempted it and the system had been so saturated by the toxic products that the patient's resistance was nil. If we could have foreseen that this case was to proceed so rapidly to a fatal termination we ought to have collapsed the lung on the day of his admission and then probably we should have been too late.

It also teaches us that the strain on the other lung is quickly shown by the appearance of activity there. The probabilities are that Pneumothorax hastened rather than delayed this patient's end by setting up activity in the better lung. I am sure that this patient had been going about with active Tuberculosis since 1918.

The refills are interesting, especially those in September - evidently there had been pleurisy on the right side as well as on the left, with no painful symptoms, owing to the two layers of the pleura being separated, but with a gradual exudation of plastic lymph causing adhesions and thereby causing the pleural space to be obliterated in parts and forming loculi which were quickly filled by a small quantity of fluid.
H.L.S. admitted 6/10/22, age 34.

HISTORY, 1918 from March till December prisoner of War in Germany. On repatriation examined by Medical Board and told he had moist patch on left lung. At that time he had pyrexia to about 100 daily, which lasted for a month. Kept fit from then till November 1920. Had operation on nasal septum under general anaesthesia which was followed by congestion of the lungs. Temperature remained up for fourteen days. Again became well and remained so till September 1922. Patient felt fevered and found temperature to be 102.4 (oral). Cough and sputum present. Sputum examined and found positive. Cough has been present and sputum in small quantities ever since operation in November 1920.

FAMILY HISTORY nil of interest, except one brother with whom he lives had been a patient here and he has still chronic Tuberculosis.

On admission, there was loss of expansion over left lower lobe, marked dullness to percussion, numerous moist sounds with bronchial breathing and marked increase of vocal resonance, both in front and behind. Some infiltration of right upper lobe but no moisture. X-rays showed marked opacity over whole of left lung. There was pyrexia from 100 a.m. to over 102.4 p.m., large amount of cough and sputum and patient looked very ill. The signs showed rapid advancement and softening began to take place. Artificial Pneumothorax induced on 18th
Case of S. No 17.

Showed reduction of about two degrees after 3 injections of gas.
October 1922 as a matter of urgency.

<table>
<thead>
<tr>
<th></th>
<th>Initial</th>
<th>End</th>
<th>200 c. c. Oxygen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oct. 18</td>
<td>-10 -16</td>
<td>-10 -6</td>
<td></td>
</tr>
<tr>
<td></td>
<td>20</td>
<td>-9</td>
<td>400</td>
</tr>
<tr>
<td></td>
<td>28</td>
<td>-6 -10</td>
<td>750</td>
</tr>
<tr>
<td></td>
<td>31</td>
<td>-6 -0</td>
<td>400</td>
</tr>
<tr>
<td>Nov 4</td>
<td>-10 -0</td>
<td>-6 -0</td>
<td>800</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>-2 +2</td>
<td>500</td>
</tr>
<tr>
<td></td>
<td>17</td>
<td>-4 +2</td>
<td>700</td>
</tr>
<tr>
<td></td>
<td>24</td>
<td>-6 +2</td>
<td>700</td>
</tr>
<tr>
<td>Dec 3</td>
<td>-12 -4</td>
<td>-6 +2</td>
<td>900</td>
</tr>
<tr>
<td></td>
<td>18</td>
<td>-4 +2</td>
<td>600</td>
</tr>
<tr>
<td>Jan 6</td>
<td>-10 -2</td>
<td>-4 +2</td>
<td>700</td>
</tr>
<tr>
<td></td>
<td>19</td>
<td>-3 +4</td>
<td>800</td>
</tr>
<tr>
<td>Feb 5</td>
<td>-14 -2</td>
<td>-6 +6</td>
<td>800</td>
</tr>
<tr>
<td></td>
<td>28</td>
<td>-6 +6</td>
<td>900</td>
</tr>
<tr>
<td>Mar 19</td>
<td>-11 -3</td>
<td>-2 +4</td>
<td>1300</td>
</tr>
<tr>
<td>Apr 1</td>
<td>-10 -3</td>
<td>-2 +8</td>
<td>800</td>
</tr>
<tr>
<td></td>
<td>30</td>
<td>-10 -2</td>
<td>800</td>
</tr>
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The above is a note of the refills, their pressures and amounts from the time of induction to the time of leaving the Sanatorium to proceed home. After each refill the temperature showed a marked reaction to 103 - 104 F. but it soon began to settle down to about 100 p.m. between refills. These reactions continued just as severely right to the end of December. The refills in January were followed by much slighter reactions and his
Symptoms were very much better - cough and sputum being practically absent. It was not till 28/2/23 that a refill was followed by no reaction. Patient was kept in bed from admission until about 12th March and even after that was only allowed to rest outside. He left the Sanatorium on 8th May 1923 to have Pneumothorax treatment carried on at home. His subsequent progress has been entirely satisfactory, and I quote the patient's own words:

"Re my progress since leaving the Sanatorium last May. I can say that the favourable course thereof has been continuous and uninterrupted. I have put on a stone and a half in weight during the last seven months. I am able to go for walks of three to four miles without being any the worse for it, and could possibly do more but I am chary of asking my 'good' lung to do too much. Of course I am more or less continuing the treatment of rest etc and am refilled every three to five weeks when I take in from 300 to 800 c.c.s Air, according to the longer or shorter period between refills..."
CASE OF K.S. No. 18

K.S. admitted 6/1/23, age 24, with a history of frequent attacks of malaria from January 1918 while on Service in Mesopotamia. In January 1922 had a very severe attack which left him pretty debilitated.

August 1922, cough and sputum developed and continued till, December 1922 when sputum was found positive and Tuberculosis diagnosed.

State on admission. The signs of disease were not very marked, but infiltration of both upper lobes was present with some extension to the lower lobe on the right side. Moisture was only elicited on each side after coughing. Cough was troublesome and sputum fairly copious, but the signs of toxæmia were slight, patient looked well-nourished and temperature was practically normal. The right side was the more intensively and extensively diseased. After three weeks' treatment in bed patient was allowed up but on 5th February the temperature rose to 102 suddenly and remained up. No Malaria parasites were found in the blood. On examination then the signs on the right side were less intense but there was a marked intensification of them on the left. He had gained by that time nearly a stone in weight. During this pyrexial attack there was definite pleurisy at the left base. He was kept in bed for five weeks till temperature became normal, and he was gradually allowed to take exercise up to 3 - 4 miles daily.

On 12th May 1923 temperature again rose to 102.6
with signs of pleurisy in left lung. Examination at this time showed disease in the right lung to be practically quiescent and it was considered that a Partial Pneumothorax by keeping the pressures very low might cause some fixation in the right lung and give the inflamed pleural surfaces time to heal. We feared to cause too great a strain on the right lung. On 27th, May Pneumothorax was induced.

<table>
<thead>
<tr>
<th>Date</th>
<th>Pressure</th>
<th>Volume</th>
<th>Gas</th>
</tr>
</thead>
<tbody>
<tr>
<td>May 27th</td>
<td>-2 -6</td>
<td>250 c.cs</td>
<td>O₂</td>
</tr>
<tr>
<td>&quot; 29th</td>
<td>-2 -6</td>
<td>400 c.cs</td>
<td>O₂</td>
</tr>
<tr>
<td>&quot; 31st</td>
<td>-2 -6</td>
<td>500 c.cs</td>
<td>O₂</td>
</tr>
<tr>
<td>June 4th</td>
<td>-2 -6</td>
<td>600 c.cs</td>
<td>O₂</td>
</tr>
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</table>

These refills show very well the gradual progress of the frequency and size of the refills in a normal case.

<table>
<thead>
<tr>
<th>Date</th>
<th>Pressure</th>
<th>Volume</th>
<th>Gas</th>
</tr>
</thead>
<tbody>
<tr>
<td>June 7th</td>
<td>-3 -6</td>
<td>600 c.cs</td>
<td>O₂</td>
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<tr>
<td>&quot; 10th</td>
<td>-10 -4</td>
<td>700 c.cs</td>
<td>O₂</td>
</tr>
<tr>
<td>&quot; 15th</td>
<td>-10 -4</td>
<td>500 c.cs</td>
<td>Air</td>
</tr>
</tbody>
</table>

By this time the left lung was two thirds collapsed with no sign of any undue strain on the right lung. Breath sounds were faint over the left upper lobe. Temperature was normal and other signs of toxic absorption nil, although the cough and sputum were increased. He was being allowed up for a few hours daily.

<table>
<thead>
<tr>
<th>Date</th>
<th>Pressure</th>
<th>Volume</th>
<th>Gas</th>
</tr>
</thead>
<tbody>
<tr>
<td>June 20th</td>
<td>-10 -4</td>
<td>700 c.cs</td>
<td>Air</td>
</tr>
<tr>
<td>&quot; 28th</td>
<td>-3 -4</td>
<td>500</td>
<td>&quot;</td>
</tr>
<tr>
<td>July 5th</td>
<td>-2 -4</td>
<td>400</td>
<td>&quot;</td>
</tr>
<tr>
<td>&quot; 16th</td>
<td>-10 -6</td>
<td>700</td>
<td>&quot;</td>
</tr>
</tbody>
</table>
Note the gradually increasing interval between refills. There was during this time a slight reappearance of signs in the right chest, but no toxaemia. Exercise 3 to 4 miles daily.

July 25th
-1\(\frac{1}{2}\) -6 800 c.c.s Air

Aug. 3rd
-2 \(\frac{3}{4}\) 900 "

" 14th
-6 -2 900 "

Very slight reaction (Temperature 100.2 for two days after this refill). Patient left the sanatorium on 6th September to continue artificial pneumothorax at home.

His progress since then has been:

(Extract from patient's own letter)

"I feel frightfully fit now and am putting weight on. My sputum is considerably reduced and I seldom cough, but I smoke more. I am sleeping very well but not sweating. There are no crepitations on my right side - so I am told ...................."."
This is an example more of a case of Hilus Tuberculosis with the disease progressing from the Hilus along the bronchiæles to the surface. I consider that although the signs in the right chest were more marked on admission, that the truly acute disease was on the left side and that it gradually approached the surface as time went on. It would have been an interesting study to have seen the progress of the disease if Pneumothorax had not been induced. I am inclined to think that it would have been a story of repeated exacerbations of toxicity and pleurisy until some definite pneumonic patches developed with subsequent caseation. This would have meant chronic invalidism, if not a fatal issue in a longer or shorter time. By inducing the Pneumothorax— even a partial one— the inflamed pleural surfaces were separated, the lung was put at comparative rest and the patient was able to be up and about and to proceed home for treatment. This latter factor was unfortunately necessary from a pecuniary point because I am afraid that the temperament of the patient was such as to preclude him taking the care of himself which might be a very deciding factor in the success or non-success of his case.

Another interesting fact in this case was the gradual diminution in the pressures both at the commencement and end of each refill, although gradually increasing volumes of gas were introduced. I can only explain this by the supposition that there soft pleural adhesions
between the two pleurae which were gradually stretched making the pleural cavity larger and larger. Weight in this case was also reduced markedly. His weights being:

On admission, 6/1/23. 62.200 kilogrammes

(1) 5/3/23 68.200 "
(2) 5/6/23 66.100 "

on departure 62.600

(1) Gain of 13 lbs before Pneumothorax was induced.
(2) Loss of 5 lbs after " " "
CASE OF R.A.R. No. 20.

R.A.R. age 31, admitted 12th April 1921 with a history dating back to early 1920, when he felt increasing lassitude with morning cough and sputum. He paid no attention to this until:

February 1921. Small haemoptysis, sputum positive. Patient was in business in India and was recommended to return home immediately.

Previous history. Nil
Family history. Nil

On examination there was flattening and diminished expansion of the right lung, dullness to R 3 in front and to the inferior angle of the scapula behind. Moist crepitations over same area with signs of a small cavity just over the clavicle.

Larynx. Left true cord infiltrated with small ulcer on posterior part.

Symptoms were not marked. Slight pyrexia to 99.2° p.m. Cough and sputum present with abundant bacilli.

Progress. After three weeks in bed temperature came down. He was allowed up and on to exercise with the exception of two months in early 1922 when he had an intensification of the signs. This patient gave us no cause for worry and his progress both as regards symptoms and signs was uninterrupted. In early 1922 we considered collapse therapy but matters settled so quietly under modified exercise that we decided to abandon it.

Patient left the Sanatorium on 5th July 1923 with lung quiescent and larynx healed. He arranged to go
back to business and worked in London. Lassitude returned in early December 1923. He took no notice till about the middle of December, when he developed temperature. As he was on the eve of departure for India he consulted a Doctor who said activity recommenced and impossible to proceed there.

Readmitted here on 21st December 1923. Pyrexia present (see chart) cough and sputum greatly increased, tubercle bacilli very numerous. Signs in the right lung much as on primary admission - perhaps more extensive behind. Left lung nil. After some rest in bed with no signs of lessened toxaemia Artificial Pneumothorax was induced on 18th January 1924. (see charts as to details as to amounts & pressures of refills).

I introduce this case although a recent one as it illustrates very well the following points.

(1). Under conservative treatment this patient did as well as could be expected but in less than five months of leading an ordinary life there was recrudescence of activity.

(2). The quick cessation of symptoms after collapse therapy in a case of this sub-acute type.

(3). The practically text-book sequence of events as regards spacing of refills and the expected reactions in a case of this type.

(4). Two months after induction of Pneumothorax cough and sputum are practically nil, tubercle bacilli are absent from the sputum, pyrexia gone and he is able
to walk from a half to one mile morning and afternoon. We have had to go slowly with pressures and physical activities as he has rather an easily displaced mediastinum which causes a certain amount of dyspnoea.
THREE CASES ILLUSTRATING THE TREATMENT OF PERSISTENT PLEURITIC PAIN BY ARTIFICIAL PNEUMOTHORAX.

...... Nos. 21, 22, 23.

E.D., age 30, admitted 20th, July, 1921, with a history of having had to nurse her mother for some time, with the result she suffered from 'nerves' and insomnia after her mother's death. She felt very weak and lost a lot of weight and had a short dry cough. Tuberculosis was diagnosed by a London Consultant and she was sent here. Nothing of note in her previous history, except a note of some cardiac complication following diphtheria. Family history, nothing to note.

She was a highly-strung nervous patient with very dark rims round eyes - these were probably artificial.

On examination there was very little to be made out in her chest beyond a little weakening of the breath sounds over the left base behind. X-rays showed little. Perhaps the root shadows were a little more marked than usual. She had pyrexia ranging from 99 a.m. to 100 p.m. A week after admission she developed definite friction in left lower lobe. On admission her heart was regular though the sounds were rather forcible in character, later it became irregular and there were systolic murmurs at both aortic and pulmonary areas with accentuation of the mitral first sound.

The pleurisy continued in spite of rest till September so we decided to separate the two layers of the pleura with Ozygen. This was successfully done by
introducing 300 c.cs of gas, but before we could give her another injection the friction spread to the pericardium causing extreme irregularity of the cardiac action so we had to desist - this irregularity happened only at intervals with no apparent cause - at the time there were blowing systolic murmurs in all areas and some dilation of the heart. As we were rather in the dark regarding the diagnosis of her condition we called in a Consultant who made a diagnosis of aostitis.

January 1922. Oedema of the feet and ankles set in. The pleurisy keep recurring at frequent intervals so we decided to again do a partial Pneumothorax notwithstanding the cardiac complications and it was started on 28th February 1922.

<table>
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<tr>
<th>Date</th>
<th>02</th>
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<th>04</th>
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<tbody>
<tr>
<td>Feb. 28th</td>
<td>-4</td>
<td>-4</td>
<td>-2</td>
</tr>
<tr>
<td>March 3rd</td>
<td>-4</td>
<td>-4</td>
<td>-0</td>
</tr>
<tr>
<td>4th</td>
<td>-4</td>
<td>-4</td>
<td>-0</td>
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Lung pretty well collapsed and pleural pain gone. The heart condition also improved to a great degree, the extra systoles sometimes appeared but the murmurs disappeared. Pulse rate was slower, regular in time and force. Pyrexia came down a little.

We continued the collapse refilling at about weekly intervals and bringing the pressure up to about neutral.

April 12th 1922. Heart's action quite regular now: temperature lower; no pain; allowed to sit up. We discontinued the Pneumothorax on 16th May 1922 and she felt
ever so much better. As the lung re-expanded, however, temperature again rose and the pleurisy and cardiac irregularity returned so the only indication was to collapse the lung again; this was done on 21st June, 27th June and 11th July. After this patient developed severe neuritis of right arm. Her story goes on as above till she left here on 11th July 1923. Whenever we let the left lung re-expand pleurisy returned so we kept collapsing it as indications presented themselves. She had recurrent attacks of neuritis also recurrent attacks of cardiac irregularity. Headaches and insomnia were prominent features, and lassitude was very marked.

We came to the conclusion that the condition was not tubercular after injection of blood into a Guinea pig proved negative. We explored every avenue which might prove a source of infection, including the teeth and throat, and we had blood cultures done and bacteriological examination of stools and urine, all with negative results.

The symptomology and complications in this case would provide a Thesis in itself and I only introduced it to show the effects of Pneumothorax as a palliative treatment in recurrent pleurisy. The after history of this case is much the same as when she was a patient here - chronic invalidism with varying complications from time to time. Pneumothorax has been discontinued, or at least I understand so.
TO ILLUSTRATE TREATMENT OF PAIN. CASE No. 2.

M.A.B. age 50, admitted May 7th, 1922.

HISTORY. September 1921, Attack of pleurisy - the pain of which had remained ever since.

January 1922. Four small haemoptyses occurred; confined to bed for a month.

March, 1922. Haemoptysis recurred, but quickly stopped; pleuritic pain still persisted.

On admission the signs were coarse leathery friction over the middle and lower lobes of right side - no intra-pulmonary signs. There was a small lesion at the apex of the left lung where crepitations were audible after coughing, and probably the site of the haemoptyses.

Symptoms. Temperature and pulse-rate were normal; pain was very acute; short dry cough was present.

Progress. Ten weeks in bed with no amelioration of signs or symptoms and the pain was interfering with the patient's rest at night. All the usual palliative measures were tried, including counter irritation, strapping etc. We then decided to try the effect of separating the pleural layers and this was done on October 10th, 200 c.c's O₂ being introduced. Pain still present. On October 13th 300 c.c's O₂ given. Pleural rub now inaudible. Pain still present but much relieved. October 18th 300 c.c's O₂. Pain entirely gone. November 1st 400 c.c's O₂ given.

There were no reactions after any of the injections. Pain remained absent when she left the Sanatorium on February 8th 1923.
July 1923. Patient had remained entirely free from pain to this time. Of course this condition is a very rare one as it is so seldom that we see pleurisy persist for such a time. Adhesion between the two layers of pleura efficiently stops and pain as a general rule.
TO ILLUSTRATE TREATMENT OF PAIN: CASE No. 3.

Mrs. A., age 26th, admitted January 20th '20, with a history of an acute attack of pleurisy with effusion at the end of November 1919. Tapped once, amount not known. She was acutely ill at this time and as soon as able to be moved was admitted here. Guinea pig inoculation of fluid was positive.

Family history was interesting as at the time of the onset of her illness her husband was acutely ill here with Tuberculosis and she was visiting him daily.


Symptoms. There was much pain at base and in axilla. Short dry cough present; slight temperature to 100 p.m.

Progress. After one week patient was allowed up. On 11th February, acute pleurisy with pyrexia - two days in bed. February 24th, attack repeated - 10 days in bed before temperature became normal. Pain had persisted practically since admission.

March 10th: -10 7
-4 0
300 c.cs O2

Reaction of temperature took place.

March 12th: -6 2
2 42
200 c.cs O2

Temperature remained at 101 for four days (she had pretty extensive mediastinal emphysema which caused a lot of discomfort).

March 18th: -6 1
-2 0
300 c.cs O2

No reaction. Pleuritic rub gone, no pain.
April 20th 1920. Pain still absent; no pyrexia. Unfortunately her husband died and she left the Sanatorium at this time. She went to the South of England and carried on treatment for about another six months and became very well. She had no recurrence of her pleuritic pain and no pyrexia.

Early 1924. Patient perfectly fit and married again.

Three injections of Ozygen cured her of the pleuritic pain which had been present practically continuously since she was tapped over three months previously.

I think these three foregoing cases prove the value of Artificial Pneumothorax in treatment of persistent pleurisy.
ILLUSTRATIVE CASE OF FAILURE TO FIND AN EFFICIENT SPACE.

No. 24.

B.S., age 21. admitted 28th September, 1923 with disease chiefly confined to right lung. History went back to December 1922, when she had an attack of pleurisy with effusion; 3 months in a Nursing Home.


August, 1923. Developed pyrexia but this subsided.

September 1923. Admitted here.

October 28th 1923. Allowed up, temperature down, feeling better.

November 20th 1923. Pyrexia developed again - digestion troublesome - signs in right lower lobe became more intense so Artificial Pneumothorax was decided on. The X-ray picture did not foretell any difficulty in gaining entrance, neither did the physical signs.

(1). Feby. 2nd 1924. First attempt failed.

"""" 2nd attempt \(-10 -5\) \(+0 +1\) 200 c.cs;

Evidently a very small pocket. A slight reaction occurred.

(2). Feby 10th 1924. \(-6.3\) \(+1.2\) 226 c.cs \(O_2\).

Entrance effected in mid-axillary line between ribs 4 and 5.

(3). Feby, 14th, 1924. \(-6.2\) \(+6.10\) 100 c.cs \(O_2\).

Note the small amount of gas required to bring pressure up to a high positive one. Evidently still smallerculus.

(4). Feby. 16th, 1923. 1st entrance ineffectual

"""" \(-5.1\) \(+8.10\) 200 c.cs \(O_2\)
Evidently in this case the chest was divided into numerous small loculi and that no communication existed between them.

After these 6 - 7 attempts collapse therapy was given up.

Diagram showing sites of puncture in case No. 24.
CASE OF NATURAL PNEUMOTHORAX E.M.H. No. 25

E.M.H. age 35, admitted 13th December 1921.

HISTORY. 1918. Cough and hoarseness. Tubercle bacilli found in sputum. From 1918 - 1921 she had no specific treatment for her illness besides travelling from one reputed health-resort to another with no restrictions as to her activities, but she remained moderately according to her account until she developed broncho-pneumonia in August 1921. After that she was confined to bed until her admission here.

PREVIOUS HISTORY. Nothing to note.

FAMILY HISTORY. Nil.

On admission patient was immediately put on typhoid rest as she was extremely ill, emaciated, troublesome cough with copious sputum, night sweats, dyspnoea - even at rest. Pyrexia ranged to 103 at night.

On examination, dullness, moist crepitations on inspiration and expiration were found all over the left lung with an area of acute softening at the left base. The right lung was also acutely infected but to a lesser extent. The temperature was rather of the inverse type tending to be higher at mid-day - always a bad prognostic sign. Conditions continued to go from bad to worse - there was definite spread in the right lung with excavation in both lobes of the left. Symptoms remained unabated. By March 1922 things looked very grave. Temperature ranged between 99 and 104, night sweats often twice and thrice nightly, heart failing and we
expected the end to come at any time.

April 16th ushered in by a sudden jump of temperature, marked pain in the left side with dyspnoea and displacement of the heart, a natural pneumothorax Pneumothorax occurred. On examination it proved to be fairly complete with amorphic breathing faintly present all over the left side, marked diminution in the audibility of crepitations, and peculiarly enough a marked decrease in the signs of the right chest. The signs gradually came back during May but the symptoms during that time had never abated very much.

May 29th, another Pneumothorax developed, and after the initial shock had passed off with its concomitant rise of temperature, increased dyspnoea and pulse rate, the temperature showed signs of coming down and there was a much smaller swing. The signs again became less marked. By July the temperature was ranging between 97.6 and 101. Pulse rate down to between 90 and 100, and she felt much better. Sweating became a much less marked feature.

August. Temperature 97.8 to 100. Moisture in lungs distinctly less but air in pleural cavity absorbing. Improvement continued and she began to put on flesh. Catamenia returned and she was distinctly less toxic. The signs in the left chest were only audible after coughing though there were still a good deal on ordinary breathing in right chest.

Left here on October 30th 1922 much against our
wishes as I considered her only chance was to be under strict supervision and perhaps to continue the Pneumothorax.

May 3rd 1923. Her Doctor wrote saying she was much the same as on her return. Temperature remained up to about 100 - 101 p.m. but "She was able to get up and about" and even was allowed to go for a sail on the Clyde. He concludes by saying that "She is not losing much ground but the disease is still active. Her weight remains up".

I bring in this case because I think it is of interest because it shows the amnestic effects which are possible by compression of the lung even in a case so markedly advanced as this one. In April I would have said that this patient only had a few weeks at the most to live. However, there she is in May 1923 able to sail down the Clyde. Unfortunately she was a stupid patient and very much troubled with over anxious and fussy relatives who evidently considered they knew better than we did about treatment, and they inadvisedly took her home. With typhoid rest and a continuance of the Pneumothorax a very satisfactory result might have been attained, but Pneumothorax would not have been possible until July 1922 or thereabout and by that time her parents were beginning to fuss about taking her home and we knew if we started the treatment that it would never be satisfactorily completed.

February 1924. Her Doctor again writes "Her lung
condition is much as it was when she left you. She is able to go about a little and has taken two or three trips to Glasgow for the day. One could not say she was any better or any worse, but there is still acute disease".
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**SUMMARY.**

1. Treatment by Pneumothorax is one of the greatest advances made in the treatment of Pulmonary Tuberculosis.

2. The simpler the apparatus the better. Saugmann's needle is best as a general rule.

3. Local anaesthesia should always be used.

4. Oxygen should be used for the first few fillings, then air.

5. Cases suitable. In four hundred cases of Pulmonary Tuberculosis I have recommended the operation in 40, or 10%.

6. Fluid developed. Operation performed in 33 cases, fluid developed in 12, or 36%. The fluid required withdrawal and replacement by gas in 9, or 27%. Emphysema developed in three of these effusion cases, or 9%.

7. Entrance was unobtainable in 3 cases, or 7.5%.

8. Results in my cases with Summary.

   Cases advised in 400 - 40.

<table>
<thead>
<tr>
<th>Group</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>Dead</td>
</tr>
<tr>
<td>Group 2</td>
<td>Operation for persistent pleuritic pain</td>
</tr>
<tr>
<td>Group 3</td>
<td>Failure to find pleuritic space</td>
</tr>
<tr>
<td>Group 4</td>
<td>Refused operation</td>
</tr>
<tr>
<td>Group 5</td>
<td>Done well and finished treatment</td>
</tr>
<tr>
<td>Group 6</td>
<td>Done well but continuing treatment</td>
</tr>
<tr>
<td>Group 7</td>
<td>Results unsatisfactory</td>
</tr>
</tbody>
</table>

Group 1. Five were in extremis and the operation was only tried as a last resort. One case was tried in order to stop a very copious haemorrhage. In another
case the operation was done first on one side and then on the other. One case died after the first injection from heart-failure. In only one of these five could the result justify the attempt, the patient's symptoms were much relieved for two months when she died of heart-failure in her sleep. Of the three remaining cases two died of acute spread of disease to the other lung, and one died after an operation for empyema.

Group 2. In all three cases relief was effected and in two cases lasted. The other case is fully described in another part (Case 23).

Group 3. Of the three cases two are dead. The third was only attempted recently - see Chart No. 24.

Group 4. Refused operation. Only two of these cases are now alive. One of the other cases left the Sanatorium and in two months developed a natural Pneumothorax and died.

Group 5. All these cases except two are described in the Paper. Cases Nos. 4, 15, 5, 12, 6, 23.

Group 6. All these cases except one are described. Cases Nos. 11, 20, 17, 18, 9, 3, 1, 2, 13.

Group 7. All are described in the text. Cases Nos. 10, 14, 8.

(9). In future I shall recommend Pneumothorax in a much higher proportion of cases at an earlier stage as I am convinced that the earlier the treatment the better the ultimate results and the less chance of intercurrent complications.
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(10). Injections should be small and at more frequent intervals. Caution should be particularly practised in cases with movable mediastinum. Not more than 200 to 300 c.c.s should be introduced at the primary operation and that very slowly.