A

Thesis

For

The Degree of

Doctor of Medicine

Of

The University of Edinburgh

By

James Williamson Fraser
DECLARATION

I hereby declare that the following thesis is a record of work carried out entirely by myself at Glenlomond Sanatorium, by Kinross.
PNEUMOPERITONEUM

IN THE

TREATMENT

OF

PULMONARY TUBERCULOSIS

A REVIEW OF 100 PATIENTS
INTRODUCTION

Pneumoperitoneum is the insufflation of air and oxygen into the peritoneal cavity; it was originally used in the treatment of tuberculous peritonitis and has now been used during the past fifteen years as a collapse measure in pulmonary tuberculosis. According to Banyai (9) the use of pneumoperitoneum can be traced back to 1872 when Spencer Wells operated upon a woman for an "Ovarian Cyst". After that, a variety of reports appeared discussing the value of pneumoperitoneum in tuberculous peritonitis and tuberculous enteritis. These reports were mainly favourable and, as a method of treatment, pneumoperitoneum did have a certain vogue.

In this connection, however, Fishberg (40) states: "we are in the dark as to how oxygen in the peritoneum will decrease peristalsis, and heal tuberculous ulcerations on the intestinal mucosa". He quotes Banyai who accounts for the successful results obtained in treating intestinal tuberculosis, by stating that of the different hypotheses two seem to be the most compatible with the results obtained.

(1) Oxygen increases all activity in all body tissues effecting a better local and inumological response.

(2) Injected oxygen, due to its chemical properties and increased intra-abdominal pressure, will cause irritation of the peritoneum and intestinal mucosa with subsequent hyperaemia.

Fishberg was obviously not impressed by these suggestions and considered that he was on "safer ground" in accepting Banyai's suggestions "that the relief of the patient with advanced pulmonary tuberculosis from anoxaemia and the action of oxygen as a mild cardiac or respiratory stimulant may explain the immediate euphoria seen in some of our patients independently of the local effect of oxygen". Many years later, the mental aspect of the value of any treatment was mentioned by Hurst, Maier and Dwork (49) in discussing pneumoperitoneum in pulmonary tuberculosis therapy: they stated "it is our feeling that pneumoperitoneum alone has some value because of the positive psychological effect which it has in some cases of bilateral extensive tuberculosis, and also in the preparation of the patients for surgery".
Fishberg concludes that "from the reports as to results attained it appears that symptomatic relief has been obtained in a certain proportion of cases. Considering the fact that we are dealing in intestinal tuberculosis, not with a local lesion of the gut, but with concomitant tuberculous, usually caseous, changes in the lung, larynx, etc. it is obvious that at least the treatment can be considered palliative".

It was not, however, until 1933 that Vajda discussed the possibility of the use of pneumoperitoneum in the treatment of pulmonary haemorrhage. Banyai (7) in the following year reported on the use of pneumoperitoneum in the treatment of pulmonary tuberculosis. He demonstrated the possibilities of using an artificial pneumoperitoneum in combination with phrenic nerve block in order to raise the diaphragm and so to create an "air splint" below the diaphragm when collapse therapy could not be carried out by other means. It seemed conceivable to him that the elevation of the diaphragm may exert a favourable influence upon the healing of pulmonary tuberculosis by:-

(1) Limiting the excursion of the diaphragm.
(2) Decreasing the intra-pleural pressure which will, in turn, increase the blood supply of the pulmonary tissue.

In this communication Banyai mentioned that the use of pneumoperitoneum in the treatment of intestinal tuberculosis had led to symptomatic improvement in the forty-four patients with advanced pulmonary tuberculosis. Thereafter, he had treated another one hundred cases, and "in many cases symptomatic relief was so striking that a more extensive use of pneumoperitoneum has been carried out since".

Fremmel in 1937 (43) accidentally induced pneumoperitoneum in two patients who were having artificial pneumothoraces induced into the lower intercostal spaces in the mid axillary line. After twenty-four refills, one of those patients had an established pneumoperitoneum; both patients appeared to have done well. Fremmel recommended pneumoperitoneum for pulmonary tuberculosis except for "chronic indurated tuberculosis of several years standing where the influence of pneumoperitoneum would probably be negligible".

Several other articles appeared in the literature of other countries at this time stating how pneumoperitoneum had been accidentally induced instead of an
artificial pneumothorax in the treatment of pulmonary tuberculosis. Those patients' clinical improvement give some force to Banyai's argument that pneumoperitoneum could be used as an independent procedure. Banyai, himself, (8) in the first article to be published in this country on pneumoperitoneum wrote that "the prompt relief from constitutional and localising symptoms I have seen in many cases are similar to that observed in artificial pneumothorax, which suggests that the mechanical and biological effect of these two methods is similar. This does not mean that artificial pneumothorax and pneumoperitoneum are interchangeable operations".

Thereafter, several articles appeared giving favourable comments on the use of pneumoperitoneum in pulmonary tuberculosis. Trimble and Wardrip (78) pointed out that with all the available collapse measures (from pneumothorax to thoracoplasty) "there is still a large group of patients for whom we have had little to offer in the way of an active approach to their therapeutic problem. This group is made up of persons with fairly extensive bilateral pulmonary disease, and on whom an artificial pneumothorax cannot be established because of adhesions and who cannot well tolerate, either because of age, disability or for some other reason, any of the more drastic types of collapse therapy". These authors were favourably impressed on eighty cases although, statistically, the results were not remarkable because pneumoperitoneum "had been employed in instances where for the most part there was practically nothing else to offer in the way of treatment".

The failure of many artificial pneumothoraces to collapse properly by limiting adhesions, was also mentioned by Joannides and Schlack (51). They point out how some adhesions may be cut to give a satisfactory closure of cavities. In other cases, adhesions are so extensive that more radical measures are needed. "Phrenic neurectomy alone, in a large number of those patients, may be of great assistance. Those in whom cavities are too rigid or too extensive or in whom the diaphragm did not rise satisfactorily, demanded more drastic procedures for satisfactory collapse. Of the latter group, many went from bad to worse because they presented contra-indications to thoracoplasty." For those cases, Joannides and Schlack recommended the use of a paralysed diaphragm or pneumoperitoneum. They did not, however, give any results to support their claims.

Further supports in favour of the use of pneumoperitoneum, when artificial pneumothorax was
ineffective, was forthcoming from Trimble, Eaton and Moore (80), who stated that the value of pneumoperitoneum was "apparent in the large group of patients in which artificial pneumothorax has failed (either because of inability to find a free pleural space or because only a small ineffective collapse could be obtained), and in which for various reasons more drastic types of collapse therapy are not indicated at the time. A phrenic crushing plus pneumoperitoneum is often the next logical step. This may bring the disease to a point where surgery is possible and may even bring about an arrest". In addition to the failed pneumothorax group, they further indicated the use of pneumoperitoneum in the "large group of patients with pulmonary tuberculosis having extensive or advanced bilateral lesions still in the exudative stage in which no other form of collapse therapy is available". Trimble and his co-authors used artificial pneumoperitoneum on 152 cases with distinct benefit in many cases.

Rilance and Waring (68) also considered pneumoperitoneum as a "procedure of merit". They used it on 55 patients; in eighteen the procedure was mechanically ineffective and, of the remainder, there were thirty-four with cavities (with a total of forty-one cavities) in whom there was cavity closure in nineteen (fifty-six per cent.).

Fowler (41), too, pointed out the value of pneumoperitoneum. He considered that its true value appeared to lie in its ability to improve the patient's general condition and alter the course of the pathologic process so that a more direct approach by radical surgery becomes safe. "In many cases an adequate arrest of the disease can be obtained through pneumoperitoneum alone, thus justifying its existence." Fowler goes on to state how the "value of pneumoperitoneum can be enhanced manifold by changing the mode of attack when the peak of improvement has been reached to the appropriate type of radical surgery".

In this country, several reports have appeared on the use of pneumoperitoneum. Thus, Clifford-Jones and Macdonald (21) consider that pneumoperitoneum has a place in the collapse therapy of pulmonary tuberculosis. But "what the place is we have not as yet sufficient evidence to define with exactitude". They did not regard it as a substitute for other forms of collapse therapy but rather as an additional measure likely to prove valuable in particular cases.

Edwards and Logan (36) were in substantial
agreement with this statement when they stated that a "certain number of patients with a type of lesion which can be suspected to respond to a rise of the diaphragm will benefit from the quicker and higher rise obtained following the reinforcements of phrenic paralysis by pneumoperitoneum". In fifty cases of poor prognosis they obtained "success" and "improvement" in twenty-five (i.e. fifty per cent. gave what might be considered a good clinical result).

In reporting on the use of pneumoperitoneum from India, Mallick, Malhotra and Mohammad (55) were of the opinion that pneumoperitoneum is a very useful therapeutic aid at our disposal in certain selected cases. They went on to state that after very careful clinical studies of bilateral pulmonary cases it would appear that the induced method of collapse therapy, though not superseding in efficiency any of the more direct methods of pulmonary collapse, perhaps with the exception of phrenic evulsion, has a great palliative and, in certain cases, even a curative value. In one hundred and fifty-six cases they have an approximately fifty per cent. satisfactory response.

To those favourable comments must be added those of Drury and Duffy (35) who, in a series of 28 selected cases, had "very encouraging results"; Keers (33) who considered that pneumoperitoneum had a place in the method of treatment of certain selected cases of pulmonary tuberculosis, and Gilmore (44) who found the "courses followed by his patients to have been most impressive".

More recently, Whitehead (85) stated that the efficiency of pneumoperitoneum in "certain selected cases is not generally appreciated", and Aronvitch, Caswell and Zadi (3) felt "that there are definite benefits to be derived from the use of pneumoperitoneum therapy, and that by its means, collapse of tuberculous lesions can be secured and sputum converted in selected patients who would otherwise die or require extensive major surgery".

During the past few years the most enthusiastic comments yet made on pneumoperitoneum have appeared. In general, the numbers of patients discussed by various authors have been in the region of fifty (with the exception of Mallick's Series of one hundred and fifty-six patients and Keir's seventy-four). But various American authors have now amassed impressive figures on which estimates might be tentatively made.
on the value of pneumoperitoneum. In this country, Hounslow (quoted by Kayne, Pagel and O'Shaughnessy) has an equally impressive total of treated patients since 1941.

**List of Authors and Patients Treated**

<table>
<thead>
<tr>
<th>Authors</th>
<th>Patients Treated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crow and Whelchel (1945)</td>
<td>546</td>
</tr>
<tr>
<td>Anderson and Winn (1945)</td>
<td>110 (negroes)</td>
</tr>
<tr>
<td>Mitchell, Hiatt, McCain, Eassom &amp; Thomas (1947)</td>
<td>474</td>
</tr>
<tr>
<td>Trimble, Eaton, Crenshaw, Gourlay (1948)</td>
<td>407</td>
</tr>
<tr>
<td>Kayne, Pagel and O'Shaughnessy</td>
<td>501</td>
</tr>
</tbody>
</table>

Mitchel et al. maintain that "despite the size of the bibliography, there is all too little concrete evidence on which to weigh the effectiveness of the procedure". Nevertheless, from the list of authors mentioned above, a sufficiently large number of patients have been treated and followed up for long enough to make those authors' remarks illuminating.

Thus Crow and Whelchel state that "Our experience has led us to believe that phrenic nerve interruption and pneumoperitoneum can be effective in a much larger number of patients than any other method of collapse therapy", and later they state "We have been forced to the conclusion that, unless further experience reveals some results not heretofore seen, the widespread proper use of temporary phrenic nerve interruption and pneumoperitoneum will mark a much greater advance in the treatment of pulmonary tuberculosis than did the beginning of the extensive employment of artificial pneumothorax".

Anderson and Winn consider their "results have been remarkable" in treating 110 negro patients. They go on to state that after giving an extensive trial to pneumoperitoneum in an appreciable series of both white and coloured cases "we believe that if it is fully applied to suitable cases, the results will be as good or better than those seen with artificial pneumothorax".

Mitchell et al. state that "adequate pneumoperitoneum is a valuable collapse procedure in the treatment
of pulmonary tuberculosis. Our immediate or long-term results amply confirm this. In the latest edition of Kayne, Pagel and O'Shaughnessy (52) it is stated that nearly fifty per cent. of admissions to Clare Hall Hospital are found to require pneumoperitoneum during the course of their treatment, and of these, at a conservative estimate, at least fifty per cent. show significant improvement as a direct result.

It is thus evident that a large number of authors have been favourably impressed by the therapeutic possibilities of pneumoperitoneum in pulmonary tuberculosis. There have been, however, a variety of conflicting opinions, as was bound to occur in any new method of treatment. Thus it is only during the last twenty years that the definitive indications for the extensive use of artificial pneumothorax have been evolved despite the fact that Forlanini published his original paper in 1895 and his results from the more extended use of artificial pneumothorax, in 1906.

Some institutions make use of pneumoperitoneum either in small or large measure; others find it of use for a small number of patients only; others again, apparently, have never practised this method of treatment at all. Mitchell et al. state in this connection "while over ninety per cent. of those writing on the subject apparently consider pneumoperitoneum a definitely useful procedure, that it is not so judged by most tuberculous physicians is a fact made obvious both by widespread personal comment and avoidance of the procedure". These remarks apply particularly to Drolet's article (34) on collapse therapy which appeared in 1943. He stated that between 1937 and 1941 only forty-four out of ninety-nine hospitals reported using pneumoperitoneum, and that a total of one thousand, four hundred and thirty-five patients had been treated in those hospitals, compared with a yearly average pneumothorax total of sixteen thousand to seventeen thousand.

Drolet goes on to quote the remarks of certain Tuberculosis Physicians, thus:

Overholt: "Pneumoperitoneum is a procedure ... too indirect .... to find much of a place in the treatment of pulmonary tuberculosis".

O'Brien: "Pneumoperitoneums are occasionally used .... but the popularity they had a few years ago, in California, has subsided".
Matson: "Pneumoperitoneum definitely has a place but not the place which was claimed for it three or four years ago. For certain unilateral cases, without bronchial obstruction where artificial pneumothorax is impossible and the case a poor surgical risk, pneumoperitoneum in combination with a phrenic crush is a really good procedure".

If one considers again, however, that Drolet's Figures refer to the period 1937-41, there is less occasion for surprise, for it is only during the last few years that pneumoperitoneum has become an accepted form of collapse therapy in this country, and even now, it is not generally applied. For example, Hounslow's Figures date back to 1941, when pneumoperitoneum was originally used at Clare Hall Hospital. However, it is only in 1944 that the first mention of pneumoperitoneum, as a therapeutic procedure, is made in the medical statistics of the Brompton Hospital Reports (18). At that time, five patients were treated; this represented 0.6 per cent. of the total in-patients suffering from pulmonary tuberculosis; by 1947, the number being treated by pneumoperitoneum had risen to fifty-one (7.9 per cent.)

This increased use of pneumoperitoneum by the Brompton Hospital added force to Trimble's remarks (80) "we know of no instance where therapeutic pneumoperitoneum has been used adequately on enough patients over a sufficient period of time and the procedure not considered to have value. We know of a number of instances, however, where but a handful of patients have been inadequately treated and the procedure then to be considered without merit".

Even so, Rafferty (67) states that "Pneumoperitoneum is no longer widely used in pulmonary tuberculosis", but he did agree with Fowler (41) that pneumoperitoneum had some value in improving the general condition so as to make the patient a candidate for some other forms of treatment.

An even more redoubtable opponent of the use of pneumoperitoneum in the collapse therapy of pulmonary tuberculosis was the late Max Pinner. In his book (63) he mentioned pneumoperitoneum "for completeness' sake" and stated of the suggestions to increase the rise of paralysed diaphragm by inducing pneumoperitoneum that "the results are not convincing; neither are those of pneumoperitoneum as an independent procedure".
In reviewing Trimble and Wardrip's Paper (1937) on pneumoperitoneum, Pinner briefly dismisses it by commenting that the 'reader will find the optimistic opinion concerning the therapeutic procedure. The more pessimistic attitudes reach rarely the printed state'. Despite the fact that he was here reviewing an article published in 1937, and before larger and more fully informative articles had appeared, Pinner was still reluctant to accord pneumoperitoneum a distinctive place in the treatment of pulmonary tuberculosis by 1946. For in reviewing Banyai's Book he was of the opinion that since the use of pneumoperitoneum was a 'little older than one decade (it was obviously) too short a time for definitive indications, contra-indications and results'. He quite rightly insisted at that time that the various reports then available did not contain statistically significant numbers of final results. "It will take years and careful unbiased study before pneumoperitoneum will reach a state of relative balance, such as artificial pneumothorax and thoracoplasty have apparently gained".

Hurst, Maier and Dwork (49), while granting that pneumoperitoneum as a form of therapy was having a wave of enthusiasm equal to no other treatment previously but artificial pneumothorax, were much more impressed by the potentialities of pneumoperitoneum in improving morale rather than the physical condition of the patients. They considered (what has already been stated) that pneumoperitoneum had some value because of its psychological benefits, which draws from Banyai (10) the remark that "it is hard to conceive why the value of pneumoperitoneum as a psychological effect is emphasized in particular". This "connotes a metaphysical trend of thought which has no room in sound clinical practice". However, in discussing this paper, Trimble makes the very pertinent comment that most of the patients treated by those authors had already been patients of a variety of other institutions. This, of course, immediately makes the group a selected one - a group of 'chronics' for whom it appeared little or nothing could be done, and certainly not an adequate cross section of a sanatorium population on whom to discuss the value of pneumoperitoneum.

"It does appear then that great divergence of opinion continues to exist in spite of the fact that well over a decade has passed since the procedure was first recommended." - so states Howlett (89). He goes on to remark that, until recently, it has been extremely difficult to judge to what extent this
sporadic but spreading enthusiasm for pneumoperitoneum is justified, as few large enough groups of patients had been reviewed, and the follow-up was generally too brief to permit mature judgment.

But, having decided that there is indeed some virtue in pneumoperitoneum, he believes that pneumoperitoneum still has yet to establish its claim to such extensive and enthusiastic use as is now being accorded in certain sanatoria and clinics.

It is believed, however, that reference to the results obtained by the various authors already mentioned will go some way to help establish pneumoperitoneum as a successful therapeutic procedure. Thus, in Crow and Whelchell's series of patients, the prognosis was poor, or extremely poor, in 58.7 per cent., questionable in 29.8 per cent. and good in only 11.3 per cent.; yet, closure of all cavities was obtained in 63.3 per cent. of the cases.

In the next series (2) of 110 patients, 11 per cent. are arrested, 22 per cent. are apparently arrested, 27 per cent. are quiescent and 23 per cent. are improved; there was cavity closure in 58.4 per cent.

Mitchell et al. give similar encouraging figures for they found 57 per cent. satisfactory results in 188 white patients, and 37 per cent. satisfactory results in 286 coloured patients with advanced pulmonary tuberculosis (not amenable to conventional forms of collapse therapy).

Trimble et al. give figures which are not dissimilar to those already mentioned; 57 per cent. of their patients became arrested and 13 per cent. were definitely improved.

The figures that Hounslow quoted again bear out the apparent effectiveness of this form of treatment, for he reported that 83 per cent. of patients were quiescent or improved.

It is believed then that from the results quoted by those authors, pneumoperitoneum has indeed something to offer in a collapse therapy programme.
Despite the fact that "paracentesis of the abdomen has been regarded with a certain amount of reticence by medical men" (Banyai), there is really no inherent difficulty in this procedure. This is emphasized too by Kayne, Pagel and O'Shaughnessy; indeed, in that book it is remarked that the management of pneumoperitoneum "is in many ways simpler than that of pneumothorax and that complications are fewer and less serious!"

The site of injection is largely influenced by the operator's own preference, although the majority opinion appears to favour the left subcostal route. Clifford-Jones and MacDonald were at one time in favour of using a site below the umbilicus, until they encountered one definite case of perforation of the bowel; thereafter, they induced in the left subcostal area. However, in Mallick's (et al.) report it is an area below the umbilicus that is used: Mitchell and his co-authors prefer this site; so does Gilmore.

Banyai, however, actually prefers to use the sub-phrenic route through one of the inter-costal spaces on the right. But in this there is the very real danger of inducing an artificial pneumothorax instead of a pneumoperitoneum. Indeed, this was reported by Fremmel and again by Gaetan (88). When one considers the anatomical relationships of the pleura, it is not surprising that artificial pneumothorax might be induced. In Cunningham's Anatomy (27) it is stated that beyond the mid-clavicular line the diaphragmatic reflection of the pleura is carried downwards and laterally across the extremities of the bony portions of the ninth and tenth ribs. As it crosses the tenth rib, or, it may be as it proceeds across the tenth inter-costal space, the line of the pleural reflection reaches the lowest point. This corresponds to the mid-axillary line which is generally accepted as a convenient line for the injections. It will thus be readily appreciated that a needle which does not completely traverse the pleural space into the peritoneal cavity could very easily induce an artificial pneumothorax. Especially is this so when it is realised that the pressures recorded by a manometer are closely related in both the artificial pneumothorax space and the immediate sub-diaphragmatic space encountered in the sub-phrenic approach.

Some authors discuss the use of preliminary medication in the preparation of the patient, and a variety of drugs have been suggested for the purpose.
However, with adequate explanation to the patient, pre-medication is not considered necessary, even with the most nervous patients.

The site most usually taken (and the one personally used) is below the left costal margin—generally one-and-a-half to two inches below the ninth costal cartilage and lying at the outer border of the rectus sheath. The left side is preferred to the right as there is no large viscus to be injured on this side. The skin is sterilized; then the skin and tissue down to the peritoneum (in so far as one can judge having reached the peritoneum) is anaesthetized with local anaesthetic.

The actual induction needle and apparatus are similar to those used for artificial pneumothorax refills (the type used does not matter); in this clinic, those devised by Edwards are used. His needle has a long free stilette which can be pushed gently forward once the needle is introduced, in order to see if there is any omentum or bowel in the path of the needle or liable to cause an obstruction to the free flow of air. The artificial pneumothorax needle is connected up to the apparatus and then the needle is gently, but firmly, pushed along the anaesthetized tract until one loses the feeling of resistance to the needle; it is then safe to assume that the needle is actually in the peritoneal cavity. In many cases the patient will wince as the needle passes through the peritoneum due to inadequate anaesthesia; this is always a helpful indication too.

There appears to be little danger in traumatizing the bowel by this procedure; it seems to be pushed forward by the advancing needle—always provided, of course, that the needle is not violently pushed into the abdomen. Only one case of trauma to the bowel has been noted (21). To avoid this complication, all injections should be away from the site of abdominal operation scars which might possibly have a loop of bowel adherent.

Clifford-Jones and Macdonald remark that the most frequent cause of failure to induce a pneumoperitoneum is when the needle is not inserted far enough; particularly in patients with thick abdominal walls. One has indeed seen this type of patient and realises the depth to which the artificial pneumothorax needle must be pushed before the peritoneum is reached. This difficulty is not nearly so common using the left sub-costal route as any of the para-umbilical routes; using these latter routes for refills, patients have been encountered who required specially long needles.
Of course, the converse is also true, that some patients have a very thin abdominal wall, particularly those patients who have had an upper abdominal operation; it is quite surprising just how thin such abdominal walls can be.

In contradistinction to the induction of the artificial pneumothorax, there are no manometric readings to indicate that the needle has indeed entered the peritoneal cavity. The intra-abdominal pressure is initially atmospheric, and thus, there is no free swing on the manometer. However, once one is certain that the needle is in the peritoneal cavity, air is allowed to flow. No matter how often a pneumoperitoneum is induced, the added precaution is always taken of frequently turning off the apparatus in order to read the manometer which begins to register a pressure as soon as air has entered the peritoneal cavity. Provided this needle has been correctly introduced, the first 100 ccs. of air into the peritoneal cavity will suffice to register a manometer swing of plus 2 plus 3 or plus 4, with expiration and inspiration (these, it should be noted, are in the reverse direction to the manometer readings seen with artificial pneumothorax).

It is possible for the needle not to reach the peritoneum, e.g. to slant sideways and be pushed along between the muscle layers: to the experienced operator, however, the sensation is appreciated. Still, a needle so placed might be taken as being intra-peritoneally, and if air is allowed to flow, it will immediately gather between the muscle layers: allowing an uninterrupted flow of air can lead to several hundred ccs. to gather in these muscle or fascial layers. Turning on the manometer, however, will immediately register a much higher pressure than would be expected, and a reading in the region of plus 10 plus 12 would be found from this air pocket. The patient, too, will also experience pain. This procedure must be repeated, of course, if there is any doubt as to where the needle is actually lying.

Provided the air is indeed entering the peritoneal cavity, 500 ccs. are allowed in at the induction and the pressure recorded; this usually comes to plus 3 plus 4 but may be slightly higher or lower. At the same time as the pneumoperitoneum is being induced, several authors discuss the advisability of percussing the liver; its loss of dulness would then indicate that air was indeed intra-peritoneal; this seems an unnecessary refinement which might only confuse some operators.
At the time of induction, patients sometimes complain of tightness across the upper abdomen; with a freely moving diaphragm, however, this is not a frequent occurrence. Rather more frequently, indeed it is almost constantly, patients complain of pain in the shoulders either at the induction or very soon afterwards. This, of course, is an excellent indication of a successful induction, as it is due to the irritant action of air on the under-surface of the diaphragm and leading to a referred pain in the third and fourth cervical dermatomes - the corresponding cutaneous distribution of the phrenic nerve. Of course, if there has been a preliminary phrenic crushing, this aching pain will only be confined to the unparalysed side. This shoulder pain has been mentioned by some writers as a complication of pneumoperitoneum; it is so constantly present after induction of pneumoperitoneum that it could reasonably be taken as a normal accompaniment of the induction. In any case, it wears off in a week or two, and is more of an annoyance than anything else.

The first refill is given in three days' time, and usually amounts to 700 ccs.; the usual precaution of ensuring that the needle is again in the peritoneal space being observed. Then further refills are given within the next ten days, and the patient goes on to weekly refills (two refills would suffice before weekly refills are started). The weekly refills consist of (generally) 800-1,000 ccs.; depending on the individual; usually 800 ccs. weekly is enough. Sometimes this drops to 700 ccs. or even 500 ccs. with children. By the time the weekly refill stage is reached, the abdomen is entered very easily, and almost invariably there is an initial manometric reading in the region of plus 5 plus 6; it may be more, or it may be less. Whereas, in the conduct of an artificial pneumothorax, the manometric pressures are all important, with pneumoperitoneum these have not the same significance, except that the pressures are not generally taken above the figure plus 8. Pressures above this are usually associated with unparalysed diaphragms; otherwise, these pressures are seen in patients who are rather "blown up". This effect may be seen in "satisfactory" pneumoperitoneum where rather too much air may have been given, but, more usually, those high pressures are associated with either a fixed diaphragm or with basal adhesions; in either case, the pneumoperitoneum is usually ineffective. In many cases, however, there may be no reading when the needle is introduced. The operator can produce one by pressure on the lower abdomen, when the manometer may rise up to a figure (e.g. plus 6), but gives no swing. In other cases, even this does not occur, and one continues the refill knowing from experience that the needle is actually in
the peritoneal cavity. Again, in some cases, especially if refills are given on the right side and in the presence of a paralysed right hemi-diaphragm, and in association with some collapse of the corresponding lung, the monometric pressure more closely tends to follow the intra-pleural negative pressure which is transmitted to the abdomen. Readings such as $-1.0$ or $2.0 - 1$ or $-1 + 1$ (inspiration : expiration) are then seen.

Refills are given at the site of induction at the left sub-costal area (unless, of course, this was initially done on the right), and always with local anaesthetic. Some physicians (Clifford-Jones and Macdonald; Edwards and Logan; Mitchell et al.) do not use any anaesthesia once the pneumoperitoneum has been established, but enter the space directly, as is done in artificial pneumothorax refills. Refills are given every week; only rarely do we find a patient who will last fourteen days without some drop in the height of the diaphragm; those patients who have had pneumoperitoneum established for some time can last fourteen days without any loss of height of their diaphragm, but fairly recent inductions will not last. The larger refills (e.g. $1,300$ ccs.), every fourteen days, only succeed in making the patient feel very distended, and are not used except in the long-established pneumoperitoneums, when those patients seem to accept the large refills without complaint and with little change in the level of the diaphragm.

Various suggestions have been made (notably by Banyai) that patients should be treated in the semi-recumbent position or, according to others, with the patient flat and with the paralysed diaphragm side uppermost to allow the air to rise up further on that particular side, with the possible action of raising still further the paralysed diaphragm. This thesis never seemed very sound from the available evidence. Now, Howlett (89) quotes Wright to the effect that when a patient changes from the erect to the recumbent attitude, the lung volume at mid-capacity (i.e. the air content of the lungs at the end of an ordinary quiet respiration) is reduced by twenty-five to fifty per cent. With a pneumoperitoneum, somewhat similar findings were obtained, and with the addition of phrenic paralysis (on two patients only), there was further decreased volume to a marked degree (fifty per cent. over the expected level in both positions).

Thus, there would appear to be sound physiological reasons for bed rest in promoting relaxation which, as will be mentioned later, is so important in
healing of diseased tissues. Most patients in this hospital are kept several months on bed rest, and only when there is clinical and X-ray evidence of satisfactory progress, are they allowed up. With continued progress, they are eventually discharged from the sanatorium to attend as out-patients, usually, as has been stated, at weekly intervals; rarely every fortnight, and certainly never longer.

In every case, the height of the diaphragm is controlled by screening prior to refills being given. It has already been pointed out that manometric pressures have no real value, and Jarman (50) stated "I cannot persuade myself that true readings are ever obtained during pneumoperitoneum refills". Be that as it may, manometric pressure readings are the only figure obtainable to give some record of events.

The use of an abdominal binder has been recommended by some writers, and not really considered necessary as a routine, by others. Banyai recommends a binder as he considers that it "increases the rise of the diaphragm in addition to that caused by any given amount of air." Mallick and his co-authors also recommended a binder; they found that there was an additional elevation of the diaphragm of 1/2", and the binder "helped to maintain a high level of diaphragm with a small quantity of air at longer intervals". Edwards & Logan and Mitchel et al. also recommend the use of a binder; the latter state "we have recently been able to demonstrate that a slight further elevation and slight further limitation of motion on both quiet and deep respiration of one or both diaphragms may be obtained".

We do not consider a binder necessary, however, except in the loose abdominal walls of parous women; then these patients can be helped by a well-fitting corset. However, in most women and men patients the good tone of their abdominal muscles seems adequate to maintain a high diaphragm. Hawkins and Thomas (47) agree with this.

Duration of Pneumoperitoneum

At the present time, no adequate number of patients has been followed for long enough to really base any estimate on how long pneumoperitoneum should be continued. Thus, Mitchel et al., up to 1946, had forty-two patients who had received treatment for two years, fourteen over three years, eight over four years and four over five years.
Trimble et al. could only present forty-one arrested patients, up to 1946, in whom therapy had been voluntarily discontinued, and all but seven of those patients were treated for three-and-a-half years or more.

From those exceedingly small numbers of patients, it is then quite impossible to base any opinion as to how long treatment should continue, and, at present, those are the only two series of published cases covering a long enough period of time for any evaluation of results.

In general, however, it is difficult to imagine that the duration of pneumoperitoneum would be very much different from that with artificial pneumothorax. References by Rafferty (67) on the duration of collapse in artificial pneumothorax vary between three and four-and-a-half years, and these are selected groups of patients who had survived the numerous hazards of artificial pneumothorax therapy. It cannot be imagined that, in general, pneumoperitoneum therapy (as the sole procedure) should last for less than three years. Of course, when other collapse measures are indicated, collapse by pneumoperitoneum should be of considerably shorter duration and depend on the clinical course of the patient.

In the final summing-up, of course, one can only say that the proper duration of pneumoperitoneum is not yet known; but must agree with Edwards & Logan "where a lesion can be controlled by pneumoperitoneum and by no other method, it will be necessary to maintain treatment for as long as artificial pneumothorax".
RATIONALE of PNEUMOPERITONEUM

Many writers set out to explain the good effects of pneumoperitoneum by referring to the high diaphragm seen in pregnant women, and the beneficial effect this appears to have on their tuberculosis. Trimble and Wardrip state "We have all noted pregnant tuberculous women who, during the latter months of gestation, have showed a very definite improvement in their pulmonary lesions; only to have these lesions become very much worse following on delivery". There was apparently "considerable speculation as to whether the good effect noted during the latter months of pregnancy was the result of hormonal changes or whether it was from the mechanical effect of elevating and splinting the diaphragm". On the appearance of Banyai's original article, Trimble and Wardrip decided to reproduce the mechanical effect of pregnancy by the use of pneumoperitoneum.

Banyai, too, considered that pregnancy caused the diaphragm to rise into the thorax with temporary improvement, the patient only to be subjected to widespread dissemination of the disease at the end of pregnancy.

No doubt, the observations were correct in so far as the benefit to a pregnant woman's tuberculosis was concerned, but the argument was basically wrong for, as Cohen (22) has recently shown, the diaphragm alters very little in position (one or two inches) during the pregnancy.

Nevertheless, from this erroneous argument, the whole theory of the functioning of pneumoperitoneum has been built up; for the basis of the pneumoperitoneum is the raising of one diaphragm into the chest and so creating the optimal state of affairs for the diseased area to collapse and to so improve that it will heal by this method alone, or to put the lung into an optimal state for further collapse measures to be considered.

Collapse by pneumoperitoneum does not differ, in the end, from collapse by any other method - the manner of its happening is alone different. As Pinner remarks "the unifying principle in all collapse measures is the reduction of lung volume". In pneumoperitoneum an attempt is made to bring this about by raising the diaphragm either in the paralysed or unparalysed state, thus allowing healing to take place in the diseased lung by the relaxation induced.
That, in effect, is purely the result of the mechanical action of raising the diaphragm.

In the normal healthy chest, there is the well accepted fact of a negative intra-pleural pressure which changes with the various phases of respiration, i.e., these are associated with changes in the thoracic cavity which, on inspiration, enlarges in all directions, only to return to its former position on expiration.

As demonstrated by Keith (quoted by Best & Taylor) four distinct mechanisms are at work in inspiration. But, of these, the most important is the diaphragm and its associated ribs (6 - 10). The increase in the vertical diameter of the chest is due to the downward elongation resulting from the descent of the diaphragm. This "piston action" of the diaphragm is responsible, during deep breathing, for sixty per cent. of the total amount of air breathed. Best & Taylor state that the total diaphragmatic surface is 270 sq. cms., and assuming an overall descent of 1 cm., the thoracic cavity will be increased by 270 ccs.; this means a corresponding amount of air taken into the lungs.

The excursions of the diaphragm are influenced by:-

(a) The upward pull of the sub-atmospheric intra-thoracic pressure.

(b) The abdominal viscera: in the standing position, the weight of the abdominal viscera exerts a downward pull and so aids the descent of the diaphragm, but hinders its ascent; the mean or mid-position (i.e. at the end of an ordinary quiet respiration) is therefore taken up at a lower level than in recumbency, when the viscera exert an upward pressure.

(c) The abdominal muscles: these, when lax and with the body in the standing position, allow the viscera to subside to a lower level, and so increase the downward pull on the diaphragm.

With a normally acting diaphragm, the effects of pneumoperitoneum may, to some extent, depend on increased intra-abdominal pressure, thus overcoming the pull of the abdominal viscera (see above). With
phrenic paralysis; however, the rise of the diaphragm may be due to the latter's space filling qualities rather than due to any upward pressure exerted by the air (35). When the paralysed hemi-diaphragm moves up in the thorax by reason of the negative intrathoracic pull, the effect is to cause a potential increase in the size of the abdominal cavity. This may be adjusted by the abdominal viscera occupying the increased space created, or may be by compensatory retraction of the non-rigid anterior abdominal wall. The limited elevation of the diaphragm thus achieved may be enhanced by pneumoperitoneum, which may act merely by reason of the space filling qualities of the air; this then allows the maximum rise of the paralysed hemi-diaphragm (35). As stated by Rilance and Waring, the force bringing about the elevation of the diaphragm is that exerted by the pressure differential that exists between the abdomen and pleural cavities.

One other factor has to be considered, viz., the sub-diaphragmatic negative pressure. As already stated, the abdominal viscera, themselves, particularly the liver, exert a downward pull on the diaphragm. The liver mass occupies most of the sub-diaphragmatic space; it has two ligaments, the coronary and triangular ligaments, running to the posterior abdominal, but these ligaments do not attach the liver to the diaphragm: this "attachment" is purely a capillary attraction between the under surface of the diaphragm and the superior aspect of the liver. There is created then a negative pressure in the potential sub-diaphragmatic space resulting from the intra-pleural negative pull and the weight of the viscera. By allowing air into the abdomen to occupy the sub-diaphragmatic space, this negative pressure is overcome and the viscera fall away into the abdomen, producing marked visceroptosis (as can be seen in any film of a satisfactory pneumoperitoneum). On screening some patients after only one or two refills, the liver may be seen "attached" to the under surface of the liver, but after several further refills, the sub-diaphragmatic force is overcome. This release from the pull of the abdominal viscera must enhance the diaphragmatic rise.

Whether or not this explanation is right, there is no doubt that the thoracic cavity is reduced in size at the same time as there is a corresponding increase in the abdominal cavity, due to the upward rise of the diaphragm (most marked on the paralysed side). This volumetric change in the lungs allows the lungs to retract and so promote healing.
The action of pneumoperitoneum in bringing about the marked elevation of the diaphragm, cannot be explained on the basis of pressure changes alone for, as has been pointed out (68), maximum elevation is practically always obtained before any appreciable change in the intra-abdominal pressure is noted. The introduction of large quantities of air and attempts to maintain high intra-abdominal pressure does not result in any further material rise of the diaphragm. Clifford-Jones and Macdonald also emphasise that the increase in pressure within the abdomen does not necessarily mean a corresponding increase in volume - a good diaphragmatic rise, for example, may be found with a pressure of +4, whereas in another case, a pressure of +10 or +12 gives a much less marked degree of elevation.

With a corresponding high rise in the diaphragm, there is also a limited diaphragmatic movement. Brian and Ricen (86) have stated that "the combined use of phrenic crushing or pneumoperitoneum are so effective because they accomplish two definite objectives, both of which have been demonstrated to play an important role in pulmonary collapse therapy. The elevated paralysed leaf can compress the lung to approximately one third of its total volume as well as eliminating the diaphragmatic respiratory excursions, another vital point in immobilising the lung".

This reduction in the lung volume by the use of pneumoperitoneum and usually in combination with phrenic nerve paralysis, has been emphasized by most writers. Banyai makes a great deal of it, and makes the point that the reduction in lung volume of patients with bilateral disease was rather more frequently observed on the more diseased lung, presumably due to the increased contractibility of the more involved lung tissue. Mitchell et al. also emphasize the great reduction in lung volume associated with rise of the diaphragm in their successful cases.

Reference to X-Rays shown in this report with the case histories will, in many cases, show a satisfactory reduction in total lung volume: but it is perfectly clear that this reduction is only taking place in the less involved (or even the healthy parts) of the lung. There is then a limiting factor, viz., adhesions, to prevent adequate collapse (or relaxation). Just as in artificial pneumothorax therapy, adhesions in artificial pneumoperitoneum exert a most important influence. Various authors have commented on this. Banyai, in his comments on lung volume, goes on to state that a satisfactory reduction in lung volume can be expected "with the exception of a relatively few cases, in which extensive pleural adhesions
prevent the rise of the diaphragm. However, it is considered that this pleural factor is far more important than Banyai makes out.

Various authors have stressed that adverse effect of pleural adhesions. Davies (30), when discussing the effect of phrenic nerve paralysis, points out that pleural adhesions over the diseased area may prevent adequate relaxation of the lung which already may be showing tendencies to contract. The need for a free pleural space has also been commented on by various authors (21, 36, 46, 2), all of whom stress that in the presence of widespread pleural adhesions an adequate diaphragmatic rise is not to be expected; nor is efficient pulmonary collapse to be expected.

At the same time, it should be emphasized that where the diaphragm has been fixed by adhesions as in a previous pleural effusion, no elevation can be expected from pneumoperitoneum. Indeed, Hawkins and Thomas say that when pneumoperitoneum is induced after an effusion, this should be done early enough to prevent fixation of the diaphragm by adhesions, a point which will be emphasized when the results are examined. Abdominal adhesions, too, when widespread and with attachments to the diaphragm, will reduce (or prevent) a satisfactory rise of the diaphragm.

There is thus the need for a free (or almost free) pleural space before pneumoperitoneum can be expected to function adequately. Best and Taylor point out how the free movement of the lungs in the thoracic cavity is hinged on the downward movement of the lung root, and with it, those portions of lungs in contact with the almost stationary regions of the thoracic walls (the posterior surfaces in contact with spinal column, dorsal surface of the lung; apex, and the mediastinal surfaces) are expanded indirectly; "i.e. when other parts of the lung move out of the way". The double pleural layer is thus provided for this free movement of the lungs, "permitting free gliding movements". In the presence of widespread adhesions or pleural symphisis, this normal movement is lost, and with it, one of the most important factors in satisfactory pneumoperitoneum collapse therapy. O'Connor (91) has pointed out the necessity of this free pleural space.

Whenever the diseased area of lung shows signs of collapsing, its continued collapse can only occur if there is a free movement of the rest of the lung upwards into the thoracic cavity. This is especially seen in the upper lobe cavities when their progressive swinging round towards the mediastinum can actually be watched from week to week. But in any other part of the lung a free pleural space can, with equal certainty, be expected to show a satisfac-
tory collapse, provided the lung factor itself permits this. In "collapse", the true nature of events is probably a relaxation - the diaphragm "flowing upwards" to take the place of lung tissue already collapsing and becoming airless. The consequence of this collapse in diseased tissue is to create an increased intra-pleural negative pressure, and also an increased sub-diaphragmatic negative pressure. As already stated, the pneumoperitoneum allows the diaphragm to rise into the thorax in response to this negative pull of the intra-pleural pressure by neutralizing the sub-diaphragmatic pressure.

It seems to be this mechanism more than the reverse which accounts for satisfactory pneumoperitoneum. By the reverse mechanism, of course, is implied that the rise of the diaphragm is the primary factor leading to collapse of the diseased lung segment. Ellison and Tittle (37), in discussing close of tuberculous cavities, state "when one half of the diaphragm is paralysed it is pulled up by recoil of elastic tissue above, and with pneumoperitoneum because of a posterior push from below". There may indeed be a combination of those two mechanisms at work.

In the end, of course, one looks for cavity closure in the satisfactory results. The mechanism of cavity closure has been expounded by Coryllos, who showed that occlusion of the bronchus was ultimately followed by absorption of the air contained in the cavity; this resulted in closure of the cavity. In a discussion on healing in tuberculous cavities, Auerbach and Green (5) showed that occlusion of cavities probably took place at the broncho-cavity junction, and was due to a combination of intrinsic and extrinsic factors. The intrinsic factors are either a caseous plug blocking the lumen of the bronchus, or an endobronchitis. The extrinsic factor is the gradual pinching of the bronchus by contraction of the pericavitary and peribronchial fibrous tissue. These authors go on to state that collapse therapy may aid in the process of cavity occlusion by distortion of the bronchus; and if the extra-bronchial pressure is raised above the intra-bronchial, an actual collapse of the bronchial walls may occur, this causing an apposition of the granulating surfaces, thereby facilitating their union.

In a discussion on cavity-closure, Pinner (62) states that although bronchial occlusion is one possible mechanism of cavity healing, it is not the only one "for bronchial occlusion must, of necessity, lead to rather rapid disappearance of the cavity lumen". Cavities that contract slowly over a period of months are most unlikely to close because of
bronchial occlusion. "In such cavities - a slow fibrosing process must occur, in which retraction of scar tissues plays an important part." This slow fibrosing process no doubt follows on atelectasis in peripherally located areas of disease. Loosli (quoted by Drinker) showed how the alveolar pores can be obstructed in an inflammatory process, and with this too, there is an obstruction to collateral respiration between the alveoli through those pores. In addition, the smaller draining bronchi to those diseased areas are liable to be blocked by exudate and also, in many cases, by an associated tuberculosis of the bronchi. The combination of those two factors is enough to completely obstruct local respiration with, in consequence, lobular (or may be even lobar) collapse. It is a physiological fact that the bronchi themselves lengthen and dilate with respiration; in the presence of exudation and swollen mucous membranes, the lumen of the bronchi may be so obstructed as to lead to complete obstruction, and with it, atelectasis. This may particularly happen in the ordinary quiet respiration associated with bed rest. There is then created the conditions for healing; as has been shown in a recent article by Cuthbert and Nagley (28), who demonstrated contraction and fibrosis may occur around peripherally located atelectatic areas. These authors demonstrated in post-mortem specimens the actual closure of small bronchi in diseased areas of lung tissue.

Barnwell, Littig and Culp (13) have pointed out that in the production of atelectasis by collapse therapy, there is relaxation of the lung and shortening of the bronchi. In paralysis of the diaphragm, they state that there is an actual shortening of basal bronchi between the root of the lung and the lesion - at least, on inspiration. In upper lobe lesions, the shortening of the distance between the root and the lung periphery is effected by an elevation of the root. They also maintain that an additional factor is the distortion of the bronchi by reason of a change in the direction of their axes. The high position of the diaphragm assumed in pneumoperitoneum may be enough to cause this distortion.

Thus it appears that in collapse therapy by pneumoperitoneum there are at least two factors at work - the closure of the bronchi by kinking, and here the raised diaphragm may partly account for the kinking and a local fibrosing process around the actual diseased tissue. Of these, probably the local factor is much more important. "Bilateral lesions, cleared simultaneously, although the paralyzed diaphragm was considerably more elevated and,
theoretically, should have shown a more unilateral effect. This observation would lead us to feel that the degree of rise of the diaphragm is not the most significant factor in the retrogression of such lesions²⁸. Case (18) illustrates this same point.
THE USE OF PHRENIC NERVE PARALYSIS

Most writers on pneumoperitoneum have recommended its use in conjunction with phrenic nerve paralysis; it has been pointed out that the additional rise to be obtained by adding pneumoperitoneum to a paralysed diaphragm is almost double that of diaphragmatic paralysis alone (78). Brian and Ricen (87) state that pneumoperitoneum was found to be only 25% effective without preliminary phrenic crushing, and Rilance and Waring consider that pneumoperitoneum only acts by its ability to make phrenicectomy a more effective procedure. Gilmore stresses the added elevation to be obtained in a paralysed diaphragm by the use of pneumoperitoneum.

It has been suggested (21) that an interval of four weeks should be allowed after the phrenic crush before a pneumoperitoneum is induced, so that the diaphragm will attain its maximum rise, since by this method the additional rise of the diaphragm due to pneumoperitoneum can be more easily assessed. On the other hand, Edwards and Logan suggest that such a delay is entirely unnecessary, and they induce a pneumoperitoneum within a few days of paralysing the diaphragm. As Cutler states, the good results of phrenic nerve interruption become evident within the first three to six months, or not at all. That being so, the enhanced rise of the paralysed diaphragm usually obtained with the addition of pneumoperitoneum should be sought as quickly as possible. In discussing the use of phrenic paralysis, Potter, Berry and Bortonne (66) state that the higher the diaphragmatic rise, the more likelihood there is of getting cavity closure. It does seem apparent then that the immediate use of phrenic paralysis and pneumoperitoneum is to be preferred.

Recent papers, however, have begun to raise doubts about crushing the phrenic nerve before inducing a pneumoperitoneum. It is stated by Crow and Whelchel that "from their experience pneumoperitoneum should be established, if possible, before the phrenic nerve is interrupted"; they point out that the presence of sub-diaphragmatic adhesions (or even basal pleural adhesions) may limit the rise of the diaphragm. Under these circumstances, the preliminary induction of a pneumoperitoneum will demonstrate if there is the possibility of eventually having a good rise of the diaphragm. So many cases are seen in which this end is not achieved, and the preliminary phrenic crushing then becomes an unnecessary operation.
At the outset of this series of cases, the usual routine was followed of paralysing the hemi-diaphragm prior to the induction of pneumoperitoneum. These usually followed one another within a week or more. Gradually, however, as diaphragms have been encountered which are quite fixed, especially after pleural effusions, or at the most, have only limited movement, the tendency has been to induce the pneumoperitoneum first, and follow this by phrenic paralysis if it seems likely to be followed by a good rise of the diaphragm. Many cases similar to those reported here would not now have a phrenic crushing because of the presence of abdominal adhesions or a partially fixed diaphragm.

From another point of view, too, this is a preferable method of attack; it has recently been mentioned in various articles that a temporary phrenic crush may eventually be associated with a permanent paralysis of the hemi-diaphragm. In the reviews on the results of phrenic nerve paralysis by Cutler and by Potter et al., no mention is made of any resulting permanent paralysis. Cutler does mention that he has seen no ill-effects resulting from a high diaphragm, but one feels that this is an end result to be avoided whenever possible. Two Case Histories (20,68) in this series show high diaphragms following on phrenic crushes; in one there is a residual basal bronchiectasis and, in the other, there is a lowering of his vital capacity. Another Case (62) shows the limiting effect of a diaphragm only partially recovered in the presence of a contra-lateral spread; the onset of fluid in a recent artificial pneumothorax added a considerable strain on this girl's respiratory reserve.

Crow and Whelchel and Mitchell et al. state that a certain percentage of diaphragms remain permanently paralysed following crushing. The former give 5 per cent. of their cases remaining with paralysed diaphragms; the latter give 42 per cent. complete and 9.5 per cent. partial failure to recover normal function. Hardy et al. have stated that among 143 cases 5 per cent. have partial and 10 per cent. have complete paralysis, with failure to regain normal function after one phrenic crush; following two crushes, the number in each instance rises to 12 per cent. A recent text-book (52) gives no figure for permanently paralysed diaphragms, nor does Rafferty. Pinner quotes "somewhere around 10 per cent.", and points out too that "one should be extremely hesitant to recommend crushing of the phrenic nerve in any patient who is a potential candidate for thoracoplasty". As one of the main
indications for pneumoperitoneum is to bring certain patients into a suitable state for thoracoplasty, the high incidence of permanently paralysed and with impaired function of diaphragms becomes an urgent problem.

Trimble et al. use pneumoperitoneum almost without phrenic crushing; in the initial period 1935-1939 phrenic operations were done on twenty-seven (49 per cent.) of a total of fifty-five pneumoperitoneum patients. During the last five-and-a-half year period, phrenic operations were done on only thirty-six (10 per cent.) of a total of three hundred and fifty-two patients, and then only for specific and rather narrow indications. They quote 57 per cent. arrested and 13 per cent. definitely improved - a total of 70 per cent. in four hundred and seven patients. Nevertheless, Mitchell's (et al.) figures are inclined to favour the use of phrenic paralysis with pneumoperitoneum, rather than pneumoperitoneum alone; they give 51 per cent. with phrenic paralysis as against 37 per cent. without.

It has been shown (78) that air in a pneumoperitoneum tends to gather under the paralysed diaphragm rather than the intact one which tends to massage air over to the paralysed side. It is a common observation on screening pneumoperitoneum patients that the paralysed diaphragm is always at a much higher level than the unparalysed one. Reference to the X-Ray photographs appended will demonstrate this point. With the unparalysed (or partly recovered one), as is seen in Case 31, the ordinary X-Ray film on deep inspiration appears to show both diaphragms more or less at the same level as they were prior to induction of pneumoperitoneum. With quiet breathing, however, as obtained in adequate bed rest, screening shows a very much reduced diaphragmatic movement. Thus, in the unparalysed state, the diaphragm can fulfil the same function; as has already been quoted in Rationale, there is a limited diaphragmatic movement to help eliminate the "concertina action" undesirable in collapse therapy.

Under certain circumstances (e.g., with extensive bilateral disease), the diaphragms of both sides at the same level may be a desirable objective; as has already been made clear, the relief of sub-diaphragmatic pull by a pneumoperitoneum may suffice to give sufficient relaxation to diseased lung tissues; bilateral disease might then get maximum benefit with the use of unparalysed diaphragms and pneumoperitoneum.

In this series 67 diaphragms which have been paralysed six months or more ago and which have been
seen recently, show that 10 (15 per cent.) are still paralysed, and 27 (40 per cent.) only show a partial recovery. Of course, a certain number of those will have shown complete recovery in another six months' time. But, equally, a certain number will remain paralysed. There does appear to be a certain amount of muscle degeneration of the diaphragm after phrenic paralysis, and this may be hastened by the presence of a pneumoperitoneum. Hardy et al. quote Stanbury to the effect that the diaphragm shows gross and microscopic evidences of muscle degeneration four months after phrenicectomy. That there is indeed some loss of function in those previously paralysed diaphragms can be seen on screening, when the sluggish action of those diaphragms can be compared with the normally acting opposite one.

In this connection, quite a few of those diaphragms recovering function demonstrate a disassociated paralysis, such as has been mentioned by Fox (42). Case 31 shows this on the right side, although the line of demarcation between the lateral, functioning half and the medial, paralysed half, is not so clearly shown as in the reported case.

It would appear desirable then, that an adequate survey should be made of the use of pneumoperitoneum without the use of diaphragmatic paralysis, more especially when on screening some patients with a paralysed diaphragm, the opposite diaphragm is seen to be over-acting. If there is a lung lesion associated with this over-acting diaphragm, there must be little opportunity for diseased tissue to receive adequate rest from this "pumping action" of the over-acting, normal diaphragm.
30.

COMPLICATIONS

A variety of complications are met with in pneumoperitoneum therapy in the same way as these are encountered in artificial pneumothorax, but they are not so serious nor crippling to the patient, and it has been stated "compared with pneumothorax therapy, we have no hesitation whatever in asserting that the inherent risks are quite strikingly less frequent in materialising, and less formidable in degree" (52). In contradistinction to artificial pneumothorax therapy, pneumoperitoneum is easily administered and is not associated with the same liability to serious accidents, provided of course, that care is duly taken in giving refills. These accidents have been reported, and were probably inevitable in any programme of treatment. However, with a more thorough appreciation of technique, and probably with a little added care, the number of really serious accidents appears to be negligible.

All the reports on large series of patients show few deaths directly attributable to pneumoperitoneum, and those reports must cover many thousands of refills when one considers the large number of patients reported on in several of the recent papers. In this series of patients, one major complication due to pneumoperitoneum as such has been encountered in over three thousand refills: this was a patient with lobar atelectasis; two cases of tuberculous peritonitis were seen, but, as will be mentioned later, doubts arise as to whether tuberculous peritonitis can reasonably be due to pneumoperitoneum.

In the reported series of cases, the more important complications are given as follows:

1. Air embolism.
2. Mediastinal emphysema and spontaneous pneumothorax.
3. Accidental pneumothorax.
4. Peritoneal effusion.
5. Tuberculous peritonitis.
6. Obliterative peritonitis.
7. Perforation of the bowel.
8. Various abdominal herniae.
Air embolism in pneumoperitoneum may be a transient attack, or it may be immediately fatal. An unusual case is described by Waring and Thomas (83) of a patient who died four days after his last refill following a bout of vomiting; post-mortem showed a typical air embolism. Other deaths have been described by Aslett and Jarman (4), Simonds (76) and Bailey (6). In Simmonds' patient there was evidence of damage to the liver; in Bailey's patient no source was found for the air embolus which reached the heart. Aslett and Jarman could not give a satisfactory explanation for the death of their two patients. The larger American series in each case report one death from air embolism; in one (58) it is stated that there was a definite breach of technique.

Transient attacks of air embolism have been reported. Although not due to air embolism, another death is reported in this section (24); it was due to trauma to a large omental vein, and the abdomen was found to be full of blood.

Simmonds describes cases which were due to mediastinal emphysema — the air leaking through the histi of the diaphragm to reach the mediastinum. One patient also developed a spontaneous pneumothorax with almost complete loss of her pneumoperitoneum. A similar case is described by Banyai and Jurgens (9) where there could have been no possibility of injuring the pleura at the time of the injection. Both these authors quote Macklin, who demonstrated how the mediastinum could be seen to widen when air was insufflated into it, only to find relief by rupturing through into the pleura with formation of a spontaneous pneumothorax.

An alternative route which air might take was remarked on by Laird (54) who, at thoracoscopy of patients with a combined pneumoperitoneum and artificial pneumothorax, saw air bullae over the diaphragmatic pleura. The rupture of one of these bullae could easily provide a channel for air into the pleural space with formation of spontaneous pneumothorax.

Accidental pneumothorax has already been mentioned as a result of using the inter-costal approach in giving refills.

Peritoneal effusion (pleural effusions in artificial pneumothorax) is by far the most important complication encountered in pneumoperitoneum therapy. Its frequency varies in published reports and, according to different observers, it is found in
3.8 per cent. (41), 2 per cent. (36), 3.4 per cent. (58), 1 per cent. (79) and 8 per cent. (2) of all cases. In simple effusions of small quantity, some authors recommend continuing the refills, as the fluid will usually be absorbed in periods of weeks or months.

Trimble, Eaton and Moore (80) have examined the peritoneum in patients who had pneumoperitoneum, and who eventually came to autopsy; they state that the local effects of air in the peritoneal cavity had been quite benign and compared favourably with local effects of air in the pleural cavity. Ascites was observed: this did not require any treatment; even when present in large amounts, it was not necessarily indicative of an inflammatory reaction in the peritoneum. In the series of twenty autopsies, six cases had ascites, divided equally between those with a normal peritoneum and those showing a chronic inflammatory reaction. These authors showed that in those reactive peritoneums, there was a gradual thickening, as shown microscopically, which could be considered as due to air in the peritoneum. But even after thirty-three months, the inflammatory changes may be minimal. Fowler saw no gross changes in the peritoneum or viscera in five cases that came to autopsy.

In this series five (5 per cent.) of peritoneal effusion were seen; in all cases the pneumoperitoneum was abandoned, as it was not considered advisable to continue refills in the presence of fairly large ascites. All the patients were completely asymptomatic, the fluid levels being noted on routine screening. In no case was an aspiration necessary.

One of these cases was associated with extensive abdominal adhesions which limited the rise of the diaphragm. Another developed in a man who was known to have had tuberculous abdominal glands (Case 68); the recurrence of fluid, however, was not associated with any symptoms. The wisdom of inducing a pneumoperitoneum in the known presence of abdominal tuberculosis has been questioned, but the risk was considered justified in the end by cavity closure.

(5) Tuberculous peritonitis has been seen in the reported cases in approximately 3.5 per cent. of patients. Those cases with this complication have usually been clinically ill and necessitated termination of pneumoperitoneum. Cohen (23) has reported on a series of cases, in which typical tuberculous peritonitis was seen in 3.58 per cent.; he considers that pneumoperitoneum might have something
to do with the onset of tuberculous peritonitis; it is difficult to accept his statement, however, that at least three of his four reported cases had no evidence of pulmonary activity. It has been suggested that tuberculous peritonitis is pre-existing, and probably is in the majority of cases (2).

The probability is that tuberculous peritonitis will occur no oftener with pneumoperitoneum than it will in the tuberculous patient without pneumoperitoneum therapy (80). This is probably an accurate assessment of its frequency. Three (3 per cent.) cases are reported in this series (13, 40, 100) and in two of these, it is really doubtful if pneumoperitoneum should be considered the causative factor. In Case 13, this patient had clinically continued activity of the right lung, but X-Ray did not reveal any cavity; sputum, however, was positive after gastric lavage, and she later developed left pleural effusion. The patient, Case 100, had a tuberculous consolidation which was associated with positive sputum; he developed a large mass in the ileo-caecal region; this was palpable after aspiration. Of the third patient reported here, Case 40, little or nothing is known; she died at home without having been seen, and is included in this group because she was reported as having a peritoneal effusion: she had a bronchiectatic left lung of tuberculous origin.

Trimble et al. report that when large peritoneal effusions occur as a complication of pneumoperitoneum therapy, tuberculous peritonitis should be suspected.

(6) Peritoneal adhesions are seen in the majority of patients with pneumoperitoneum (Anderson and Winn quote 71 per cent.); but these cause no difficulty. However, in a certain number of patients, there are progressive adhesions (obliterative peritonitis) which make refills more difficult, and finally lead to the abandonment of the pneumoperitoneum. Two cases of this nature were seen in this series.

(7) Perforation of the bowel was reported by Clifford-Jones and Macdonald; it was confirmed by culture of faecal organisms. Keers (53) has also reported a case.

(8) Abdominal herniae have been variously reported, but these can be easily controlled by a properly fitting truss. A series of recent reports have appeared, giving accounts of scrotal pneumocele. One reports this occurring whilst the induction was being given (70), and a second (59) occurred at the second refill, and was controlled by a properly applied
In both cases, the scrotum was swollen in five hours or so, and remained so for the next week or more. Two other reports record this complication (57, 61). All reports are agreed that this complication is due to air opening up the unobliterated tunica vaginalis and herniating into this potential space.

In this series, no gross abdominal herniae were seen; one patient had a weakened abdominal wall from an upper abdominal operation, but no true herniation. Several patients have been seen with small umbilical herniae from air. These have caused no upset nor any real discomfort.

Among other reported complications is pleural effusion; in this series, this was seen in one of the patients who developed tuberculous peritonitis (Case 13); pleural effusion should probably not be considered as a complication of pneumoperitoneum, as it is found no oftener than among any tuberculous patients.

A much more serious complication was seen in patient 74, who developed a lobular atelectasis, and empyema and then a massive atelectasis (Figs. 14-18). This condition followed on a proved case of tracheobronchitis. It has already been mentioned in the literature by Brian and Ricen.

### Table 1.

<table>
<thead>
<tr>
<th>List of Complications Encountered in Pneumoperitoneum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peritoneal effusions ..... 5</td>
</tr>
<tr>
<td>Tuberculous peritonitis ..... 3</td>
</tr>
<tr>
<td>Obliterative peritonitis ..... 2</td>
</tr>
<tr>
<td>Lobar atelectasis ..... 1</td>
</tr>
<tr>
<td>Severe abdominal pain ..... 1</td>
</tr>
</tbody>
</table>

These major complications in all cases necessitated termination of the pneumoperitoneum. They amount to 12 per cent. of the total cases, and compare with Trimble et al. 6 - 7 per cent., and Mitchel et al. 4.7 per cent.

No other notable complications were encountered.
It has been suggested (36) that loss of weight was a most notable feature - nineteen patients lost up to eighteen pounds in weight, and the comment is made that "loss of weight occurred irrespective of other clinical features, and was associated with a characteristic feeling of fullness and discomfort soon after starting a meal which had been approached with a good appetite. This has been seen in this series too, but then, mostly in patients who were probably having large refills; they immediately began to regain the few pounds of weight they lost when the amount of air was cut down at refills. This seems more a problem of management than an actual complication."
INDICATIONS

Fowler gives the following indications for pneumoperitoneum:

(1) The far advanced case for whom no other procedure is applicable.

(2) Bilateral pulmonary tuberculosis with adhesive pleuritis preventing the use of artificial pneumothorax.

(3) Unilateral tuberculosis with adhesive pleuritis too acute and inadequately stabilised to make thoracoplasty advisable.

(4) Predominantly basal lesions.

(5) To enhance the effect of a paralysed diaphragm.

(6) Uncontrolled haemorrhage.

Broadly speaking then, there are apparently two groups of patients which have been recommended for pneumoperitoneum - the "failed artificial pneumothorax" group and the far-advanced. In many of the latter, pneumoperitoneum has probably been given more as a solace to the physician's conscience and to boost patients' morale, rather than with any precise indication for any end result. Considering the comparative lack of complications, pneumoperitoneum is always something that can be tried.

Another indication has been suggested by Barnes (11) who recommended pneumoperitoneum in post-partum patients to keep up the level of the diaphragm. The patient she reported was not conclusively shown to be an active tuberculosis in the first instance; secondly, it is difficult to see how the lax abdominal walls following immediately after pregnancy could possibly give enough support to 3,000 ccs. of air, even with a binder applied. It is even more difficult to imagine a successful pneumoperitoneum following after Caesarean section (18): the authors claim that pneumoperitoneum can be induced at the time of operation "and its maintenance for a month or two is a relatively simple matter".

More precise indications can be given by reference to the Case Histories and X-Ray plates in the Appendix. From the relevant histories, the following indications are given.
1. **Unilateral tuberculosis**, where an artificial pneumothorax has failed, and the next step would be a thoracoplasty. Pneumoperitoneum should be tried as an intermediate procedure.

2. **Unilateral disease**, in which artificial pneumothorax has been given but insufficient relaxation is found by reason of apical or diaphragmatic adhesions.

3. Predominantly **unilateral disease**, where pneumoperitoneum has been tried as a primary procedure; mostly, in order to help a patient with acute exudative disease to stabilise before using other collapse measures, either artificial pneumothorax or thoracoplasty.

4. **Bilateral disease**, which is either too acute or too advanced for any other immediate therapy. The patients in this category might be classified in three groups:

   (a) Those on whom, if one side is improved sufficiently, major surgery might be considered on the other.

   (b) Those on whom pneumoperitoneum is tried in combination with artificial pneumothorax on the other side.

   (c) Those on whom pneumoperitoneum is tried as the primary procedure.

5. To close empyema spaces, facilitate absorption of fluid and help re-expanded "unexpandable lungs".

   The results are analysed according to the site of disease principally indicated for attack; the various lung zones are then individually considered. Where more than one zone is involved, this is discussed in a separate group; many patients could probably be discussed under "one zone", but the zonal distribution of disease has been followed.

   Certain patients in this analysis have extensive disease involving more than one zone, but some are included in the analysis of "one zone" because it was precisely that one zone for which treatment was indicated, in order, in most cases, to prepare for later thoracoplasty, for example, of the upper zone when the pneumoperitoneum has been indicated for basal disease.
I. Unilateral disease where artificial pneumothorax has failed.

(a) Upper Zone (Cases 16, 50, 85).

In all those cases if pneumoperitoneum is not used, the next step, logically, would be thoracoplasty. However, it is believed that the intermediate stage of pneumoperitoneum will, in many cases, give a satisfactory result, obviating the use of major surgery. This has probably been achieved in Cases 50 and 85: in those patients there was not an entirely free pneumothorax space, but in neither case was there a complete pleural symphisis; the lung was adherent in greater or less degree opposite the site of disease. The artificial pneumothorax, of course, was completely ineffective.

The other case in this group (16), despite a satisfactory rise of the diaphragm, had thick walled cavities hidden in the apex; these were only shown up by tomogram. This patient has now had a thoracoplasty done elsewhere. It is probable that tomography in the first place would have suggested an immediate thoracoplasty after the complete failure of a widely adherent artificial pneumothorax.

(b and c) No cases are included in this group of Mid or Lower Zone cavities not affected by primary artificial pneumothorax attempted before pneumoperitoneum.

(d) Two or more Zones involved (Cases 10, 62, 63).

An initial attempt at artificial pneumothorax was unsuccessful because of widely adherent lung in two cases and, in Case 63, a large tension cavity appeared after an artificial pneumothorax was induced; in each, pneumoperitoneum seemed indicated.

Case 10 gave an apparently satisfactory result, until recent tomograms showed up a small residual cavity. This man was considerably improved by pneumoperitoneum, and he appeared to be clinically well.

The next patient (63) had a large tension cavity which did not respond to pneumoperitoneum.

Patient 62 had disease in upper half of right lung (Fig. 1) which was seen to be associated
with a thickened inter-lobar septum. Despite a satisfactory rise of the right diaphragm; however, the diseased area, itself, altered very little in position, as is seen in Fig. 2; this might have been expected from the previous history of pleurisy on the right side. The evidence of tracheo-bronchial tuberculosis gradually became obvious in this patient (Fig. 2) and led to the final picture (Fig. 3) of a left hydropneumothorax and a large right tension cavity. The fresh spread of disease to the left lung would have been an indication for pneumoperitoneum; only the right diaphragm had not, at that time, completely recovered function; a pneumoperitoneum, under those circumstances, would probably have only succeeded in raising the right diaphragm.

2. Pneumoperitoneum used in combination with artificial pneumothorax (Cases 42, 43).

In patient 42 a left upper zone cavity (Fig. 4) has been partly controlled by an artificial pneumothorax and phrenic crush (Fig. 5); however, the cavity is still seen lying near the hilum in left mid-zone (cavity seen in second interspace). The addition of a pneumoperitoneum (Fig. 6) was enough to close the cavity; confirmed by Tomogram.

The second patient (43) shows the use of pneumoperitoneum applied to an artificial pneumothorax, which was showing signs of having to be abandoned soon; pneumoperitoneum was indicated before the lung becomes adherent to the chest wall, with the possibility thereafter of it being quite firmly fixed.

3. Pneumoperitoneum has been used as a primary procedure in unilateral disease, either in the belief that this will, itself, suffice to control the lung disease, or to improve a patient sufficiently for major surgery. This particularly applies where disease involves two or more zones of the lung.

(a) Upper Zone disease (Cases 54, 58, 68, 74, 83).

In those patients pneumoperitoneum was used as a primary form of treatment in the belief that it would, itself, suffice as it has done in 58 and 68; the former patient, however, has had an extension of disease to the left; the latter remains well four months after pneumoperitoneum
was terminated because of peritoneal effusion. He has a persistently high diaphragm.

Other two patients (54, 83) had pneumoperitoneum induced primarily to give the upper lobe disease the opportunity to retract in the manner already discussed. This indication in acute disease, however, is not acceptable in the more chronic type of disease where there has been an opportunity for the lung to become widely adherent over the whole diseased area.

In Case 54, the original X-Ray (Fig. 7) did suggest an adherent lung with a thickened inter-lobar septum; however, pneumoperitoneum was given a trial; despite a good rise of the diaphragm (Fig. 8), the diseased upper-zone has altered its position very little after nine months. This gave no indication, however, of the true extent of the disease, as seen by a later tomogram (Fig. 9).

In Case 74, disease that originally appeared susceptible to pneumoperitoneum (Fig. 10) - recent soft nodular infiltration in a youth - eventually produced a train of circumstances associated with tuberculous tracheo-bronchitis (Figs. 11 - 15); this class of patients will be discussed further.

(b) Middle Zone disease (Cases 12, 20).

Two patients had primary pneumoperitoneum for disease in the apex of the right lower lobe; neither one was started in this sanatorium. Both have terminated treatment, and both remain well. Case 20 has a high, poorly functioning right diaphragm with basal bronchiectasis (Figs. 16, 17).

(c) Lower Zone disease (Cases 23, 26, 30, 75, 81, 82).

Disease in this situation is generally accepted as one of the prime indications for pneumoperitoneum. In all the patients mentioned there was the specific indication - to close a basal cavity or to improve infiltration; in Cases 23, 26, 75 and 82, it was hoped that this would suffice to control the disease; in Cases 30 and 81, the indication was to control or improve the basal disease to permit radical surgery for upper lobe cavities.

Patients 23 and 26 appear to have adequately controlled lesions, although in Case 23, this is by no means certain (Figs. 18, 19). This
women, however, has remained perfectly well during the past eighteen months.

The next two patients in this series, 75 and 82, have not had basal cavities closed by pneumoperitoneum. In patient 75, there was a fairly large cavity lying behind the heart, and which was eventually seen by tomograms: pneumoperitoneum, although reducing its size, did not close this cavity; it was, however, successfully attacked by a left artificial pneumothorax; no adhesions were present. The impression remains that a bronchial factor was present, limiting the application of pneumoperitoneum.

This was probably also present in the next patient, 82, who had a cavity at the left base in some fairly dense disease (Fig. 20). A pneumoperitoneum did not close the cavity, and an eventual artificial pneumothorax (Fig. 21) was also unsuccessful, the cavity being held out by adhesions.

Cases 30 and 81 were intended to improve the right and left lungs respectively, for eventual thoracoplasty; in Case 30, this was a vain hope really for the patient had thick-walled cavities, probably of some considerable duration, and these, naturally, resisted collapse. However, it was hoped that in the next patient, 81, a preliminary induction of a pneumoperitoneum would improve the left basal disease sufficiently to ensure a more satisfactory result; there was not, however, any material benefit from pneumoperitoneum.

(d) Two or more Zones involved
(Cases 13, 91, 100)

With patient 13 there was a past history of right pleurisy, and an established pneumoperitoneum never really altered the position of the diseased upper lobe which was obviously adherent. However, radiologically, the lesion appeared controlled, although clinically, it was not. She eventually developed a tuberculous peritonitis and the pneumoperitoneum was abandoned. Thereafter, gastric lavage gave positive result, and X-Ray eventually showed up the cavity in the right infra-clavicular area. Another adherent lobe was seen in Case 100, who had a tuberculous consolidation of the right, middle and upper zones (Fig. 22); a pneumoperitoneum was quite ineffective in causing any
real rise of the inter-lobar septum and, in addition, a cavity had appeared in the infraclavicular area. This man also developed a tuberculous peritonitis with a quite obvious ileo-caecal mass.

Patient 91 shows a satisfactory response to pneumoperitoneum. She had a large peripherally located cavity which only became obvious after artificial pneumothorax was induced (Fig. 25): its size could not be guessed from the original film (Fig. 24). Such a cavity, of course, is a complete contra-indication to artificial pneumothorax, as cutting adhesions in those peripherally located cavities frequently results in empyema, not only because of the situation of the cavity but because much of the blood supply to the wall of such a cavity is carried in those adhesions from the chest wall. A pneumoperitoneum induced then is known to be associated with a more or less free pleural space, and this seems an absolute indication for such therapy. It seems, in the end, that a satisfactory result should follow despite the present positive sputum, there is apparently cavity closure radiologically (Fig. 26) and by tomogram. It is hoped that sputum conversion will result and clinical signs will disappear.


(1) Improving one side sufficiently for major surgery on the other.

(a) Upper zone disease (Case 79).

This patient has had a satisfactory result from pneumoperitoneum, where no gross cantation was present. Continued improvement on this side would now make this man a candidate for right thoracoplasty.

(b) Mid-Zone disease (Cases 46, 87).

Both patients had advanced bilateral disease on one side, with recent infiltration in the mid-zone of the other lung. In one (46) the pneumoperitoneum has been abandoned in favour of an artificial pneumothorax; in the other, the pneumoperitoneum has been continued, but it is not completely certain that the disease has been really controlled.

(c) No patient was seen with lower zone disease in this category.
(d) Two or more zones involved  
(Cases 18 and 40).

In patient 18 the pneumoperitoneum was induced to control disease in the lower half of the left lung; to allow an eventual thoracoplasty when the right side had been stabilised by bed rest (Fig. 27). However, after a period of two years, the very successful result shown in Fig. 28 was obtained. Despite the fact that the right diaphragm was not paralysed, there was sufficient relaxation to allow the diseased right upper lobe to collapse against the mediastinum; at the same time, the left basal disease had cleared. This is a most successful result.

Patient 40 had been recommended for left thoracoplasty when she developed disease in the upper half of the right lung; an artificial pneumothorax was tried, but the lung was adherent over the apex. A pneumoperitoneum thereafter appeared to give satisfactory control of this apical lesion. However, the patient took ill at home, and died there without an accurate diagnosis having been established: she was considered to have had "tuberculous peritonitis".

(2) Pneumoperitoneum used in combination with artificial pneumothorax on the opposite side.

(a) Upper zone disease (Case 96).

This girl was originally seen with extensive bilateral disease (Fig. 29), with marked acute exudative disease on right. She had a period of bed rest at home; waiting admission to the sanatorium; on admission, X-Ray (Fig. 30) shows that disease is now confined on right to the upper zone; there has apparently been considerable retraction upwards by the healing, diseased lobe. This process was assisted by pneumoperitoneum, and has resulted in further "swinging inwards" towards the mediastinum although, at present, the cavity has not yet closed; in addition, a recent artificial pneumothorax has been established to attempt control of the mid-zone left disease (Fig. 31); at present, this is only of limited value from adhesions.

(b) No patient was seen with mid-zone disease in this category.
(c) Lower zone disease (Case 94).

This patient had a basal cavity; it was considered that pneumoperitoneum was indicated for this lung to allow sufficient improvement before further treatment could be considered. Recent X-Rays no longer show a definite cavity but it remains doubtful if this is really closed. She had an artificial pneumothorax to control left lung disease; it is as yet too early to say whether this will do so.

(d) Two or more zones involved (Case 34).

This boy had acute exudative disease on the right, and lighter mottling on the left (Fig. 32). Pneumoperitoneum was induced in an attempt to stabilise the right lung and left artificial pneumothorax induced to control the disease on the left. Progress has continued satisfactorily, up to a point, but the adherent inter-lobar septum has apparently prevented further retraction of the right upper lobe, and a cavity became obvious in the infra-clavicular area (Fig. 33). This cavity, like so many more, is much larger than it at first appeared when tomogram was done (Fig. 34).

(3) Pneumoperitoneum indicated as a primary procedure. This is a large group commonly seen in sanatorium practice, for whom, in many cases, little or no active treatment can be offered. All those patients have disease extending for more than two zones; except Case 93 for whom the mid-zone cavities on the left offered a precise indication for treatment; these appear to have closed.

Cases 31, 71, 78, 88 and 98.

Of these cases, 31 and 98 were acute exudative lesions in young girls with marked disease on one side and less obvious disease on the other. Fig. 35 shows the extent of the lesion in case 31 on admission; there is a large thin-walled cavity lying in the right upper lobe, with slight mid-zone mottling. In view of the acute nature of the disease, artificial pneumothorax was contra-indicated and pneumoperitoneum was induced. The present appearance is shown in Fig. 36 after phrenic paralysis had been done on both the right and left sides. As yet, the disease is not finally controlled and some other method of collapse is indicated.
Patient 98 showed much the same initial response on the left side, but this did not prevent a fresh lesion from advancing in the left mid-zone; for this she had an artificial pneumothorax, which is not really technically satisfactory; this has been induced now with considerable less risk than would have been so as an initial artificial pneumothorax.

Patient 88 had extensive disease in the upper half of the right lung, with lighter mottling on the left. Right artificial pneumothorax was unsuccessful; pneumoperitoneum was then indicated, and this gave enough relaxation for the diseased right upper lobe to retract upwards towards the clavicle. Clinically, the disease is not yet controlled, but tomogram has shown that the large cavity has closed; small "honey-combed" cavities, however, remain.

Patient 78 had extensive infiltration on the left, with hilar cavities (Fig. 37); there was also right mid-zone infiltration. The left lung disease was too acute and too extensive for artificial pneumothorax. Pneumoperitoneum was thus indicated; a satisfactory rise of the left diaphragm was finally obtained (Fig. 38), but the disease was not controlled clinically. Tomogram (Fig. 39) showed a large posterior cavity which is seen to lie in the apex of the left lower lobe (Fig. 40).

Patient 71 shows the use of a known ineffectual pneumoperitoneum, to ease a patient's cough and help her bring up sputum. This pneumoperitoneum was stopped, but the patient requested that it be started again precisely because of its ability to ease cough and sputum.

5. To close empyema spaces. No case is reported in this group for the X-Rays of the one successful case (No. 67) are no longer available.

No patient was encountered in this series with severe uncontrolled pulmonary haemorrhage.

Benatt ( ) states that severe haemoptysis is probably better controlled by pneumoperitoneum than by an artificial pneumothorax because:

(1) It is rarely impossible to induce a pneumoperitoneum.

(2) There are fewer hazards.
(3) It is unnecessary to know which lung is bleeding.

(4) Extensive collapse is not needed.

An interesting case of pulmonary haemorrhage was described by Logie, Walker and Stoddard (90), who used pneumoperitoneum to control a massive pulmonary haemorrhage from a bronchiectatic lung, where artificial pneumothorax could not be used because of a recent pleurisy. The patient was given 1,000 ccs. of air daily for ten days, and between August 8th and September 30th he had 14,500 ccs. of air. During this time the patient lost 6,000 ccs. of blood in nineteen massive haemorrhages in a period of thirty days.

The indications for pneumoperitoneum therapy may be given briefly, thus:

(1) Where artificial pneumothorax has failed.

(2) To give increased relaxation in a partially effective artificial pneumothorax.

(3) Exudative disease, either unilateral or bilateral.

(4) With the object of helping a patient with bilateral disease to advance to other collapse measures, notably thoracoplasty.

(5) To close empyema spaces.

(6) For the control of haemoptysis, where the source of the bleeding cannot be controlled by artificial pneumothorax in unilateral disease, or where the source of the bleeding is not known in bilateral disease.
CONTRA-INDICATIONS

These are few and do not cover so wide a field as those for artificial pneumothorax; indeed, contra-indications for pneumothorax become, in many cases, an indication for pneumoperitoneum. Many contra-indications mentioned by authors are not really a contra-indication to pneumoperitoneum as such, but rather against interference of any kind. Thus, generalised tuberculosis and amyloidosis have been mentioned (9), and moribund patients with extreme toxicity (79); in this group of far-advanced patients of hopeless prognosis, there is little point to using any method of collapse therapy until the patient himself shows some signs of resisting the disease successfully. It is a mistake to believe that any form of collapse therapy can, of its own, arrest a pulmonary lesion. Despite collapse measures, many patients show progressive disease.

One might mention far-advanced disease with large thick-walled cavities; just as any other form of therapy would be regarded as out of the question, one would also place pneumoperitoneum in the same category in trying to close those cavities.

It has been suggested that serious heart disease should be a contra-indication; thus Banyai states coronary disease and cardiac decompensation should be considered contra-indications; one can only agree, for there appears to be little point in instituting any form of collapse therapy in the presence of marked cardiac disease. It might be added, in this connection, that Benatt and Berg (15) have demonstrated by electro-cardiagrams that pneumoperitoneum in the normal individual does not cause any changes from the cardiac point of view.

Certain abdominal factors are contra-indications to pneumoperitoneum. These are:

1. Limited diaphragmatic movement, as seen on screening, e.g., in patients with old-standing pleurisies and empyemas, or who have extensive abdominal adhesions to limit diaphragmatic excursions. It will be seen in the analysis of results that in no patient with an empyema or pleural effusion was a satisfactory rise of the diaphragm obtained where the empyema or effusion had continued for several months before the pneumoperitoneum was induced.

2. Presence of severe abdominal disease -
tuberculous gland masses and large tumours. One patient was seen in this group with known ileo-caecal glands: despite this, a pneumoperitoneum was eventually used and was successful in closing an upper zone cavity; this patient, however, developed a peritoneal effusion. It is not considered advisable, then, to induce pneumoperitoneum in the presence of known tuberculous abdominal complications. Any acute inflammatory condition would also contra-indicate pneumoperitoneum.

Large tumours, by reason of their space-filling qualities alone, may be considered as a contra-indication to pneumoperitoneum, and pregnancy (79) has also been suggested.

In this series no contra-indication was seen, except one, viz., the complication illustrated in Case 74 of lobular and lobar collapse in the presence of tracheo-bronchial tuberculosis. In many cases, its presence might be guessed from the associated symptoms of wheezing, marked coughing and dyspnoea disproportionate to the X-Ray evidence of lung damage. In some, the X-Ray appearances may suggest this complication, as in Fig. 1; in the majority of patients, however, its presence may only be known by the trend of events, as bronchoscopic examination is, as yet, only of limited value in visualizing the main bronchi; the smaller bronchi which may, themselves, show tuberculous changes, are not seen.

Tracheo-bronchial Tuberculosis.

This is always a major problem in any programme of collapse therapy, as interference with bronchial drainage can set up a train of events such as is seen in patient 74. It has been reported that pulmonary tuberculosis is associated with tuberculosis of the main bronchi in 10 to 15 per cent. of patients; Rafferty gives the figure 11 per cent., and Salkin, Cadden and Edson (71) give 15 per cent.; at post-mortem examinations, the figure rises even higher for Salkin et al. quote 40 per cent. Shipman (74) stated that "the method of attack in the attempt to close certain cavities should not be so much collapse therapy but rather the cure of the diseased bronchi draining cavities and the attempt to relieve obstruction". Sampson (72) considers that collapse therapy should not be given, and points out that the results have been equally poor whether the patient is treated by bed rest only or by collapse therapy. He also points out that tuberculosis of the bronchi may only be seen after collapse measures are instituted. In this connection, Warren, Hammond and Tuttle (82)
recommend that all patients should be bronchoscoped if there is any doubt at all about the presence of tuberculous bronchi. Many others have recommended that all patients who are candidates for collapse therapy should be bronchoscoped. This, of course, is an ideal not yet generally obtainable in this country.

Trimble et al. use pneumoperitoneum therapy in the known presence of tuberculous bronchitis in 74 patients; the results were definitely not good, but compared with any other treatment available, the results in the minimal and moderately advanced cases were not unsatisfactory. They point out, however, that most of their work anti-dates the use of streptomycin; whether or not streptomycin is, itself, completely satisfactory, yet remains to be seen.

In this series, 4 patients (Cases 52, 62, 74 and 94) have definite clinical and X-Ray evidence of tracheo-bronchial tuberculosis; one (74) has been confirmed by bronchoscopic examination. Patient (62) showed some initial improvement as a result of pneumoperitoneum, but eventually a small cavity appeared in the partially collapsed right upper lobe (Fig. 2), and this has gradually enlarged to form a typical tension cavity which is occupying the complete right upper-zone (Fig. 3). The treatment of a tension cavity is always a problem; with super added disease on the left lung, such as this girl shows, there is little that can be done at present.

Patient 74 showed a major complication of pneumoperitoneum, in that he developed a lobar atelectasis and, later, a complete lung collapse. The first X-Ray (Fig. 10) with its "stringy" lesions might have suggested bronchial tuberculosis; but it was not so taken, and pneumoperitoneum was instituted, only to be followed by lobar collapse (Fig. 11). Later, there was an inter-lobar empyema (Fig. 12) which the patient proceeded to cough up after it had ruptured through into a bronchus. At the same time, there was an extension of the disease in the right cardio-phrenic angle (Fig. 13). Another attempt was made to cautiously institute pneumoperitoneum, but this was immediately followed by an almost complete collapse of the right lung (Fig. 14); after this the pneumoperitoneum was abandoned forthwith. This particular patient has started streptomycin and Fig. 15 already shows quite a remarkable improvement.

Of the other two patients mentioned, 52 developed a large tension cavity while under treatment, causing the pneumoperitoneum to be again abandoned. Patient 94
had a tension cavity in the left mid-zone when first seen; this was completely uninfluenced by pneumoperitoneum.

From the results with those four patients, pneumoperitoneum would seem to be contra-indicated in patients whose history, signs and symptoms, and X-Ray appearances lead to any suspicion of tracheobronchial tuberculosis.
<table>
<thead>
<tr>
<th>AGE</th>
<th>MALES</th>
<th>FEMALES</th>
<th>TOTALS</th>
</tr>
</thead>
<tbody>
<tr>
<td>10 - 19</td>
<td>6</td>
<td>10</td>
<td>16</td>
</tr>
<tr>
<td>20 - 29</td>
<td>23</td>
<td>31</td>
<td>54</td>
</tr>
<tr>
<td>30 - 39</td>
<td>13</td>
<td>7</td>
<td>20</td>
</tr>
<tr>
<td>50 +</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td><strong>TOTALS</strong></td>
<td><strong>49</strong></td>
<td><strong>51</strong></td>
<td><strong>100</strong></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>AGE</th>
<th>MALES</th>
<th>FEMALES</th>
<th>MALES</th>
<th>FEMALES</th>
</tr>
</thead>
<tbody>
<tr>
<td>10 - 19</td>
<td>3</td>
<td>1</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>20 - 29</td>
<td>11</td>
<td>12</td>
<td>16</td>
<td>15</td>
</tr>
<tr>
<td>30 - 39</td>
<td>1</td>
<td>5</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>40 - 49</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>50 +</td>
<td></td>
<td></td>
<td>1</td>
<td></td>
</tr>
<tr>
<td><strong>TOTALS</strong></td>
<td><strong>2</strong></td>
<td><strong>21</strong></td>
<td><strong>26</strong></td>
<td><strong>1</strong></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>AGE</th>
<th>U.Z.</th>
<th>M.Z.</th>
<th>L.Z.</th>
<th>2 + Z</th>
<th>Pleura</th>
</tr>
</thead>
<tbody>
<tr>
<td>10 - 19</td>
<td>6</td>
<td>2</td>
<td>8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>20 - 29</td>
<td>12</td>
<td>14</td>
<td>7</td>
<td>17</td>
<td>5</td>
</tr>
<tr>
<td>30 - 39</td>
<td>6</td>
<td>3</td>
<td>9</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>40 - 49</td>
<td>4</td>
<td>1</td>
<td>1</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>50 +</td>
<td></td>
<td></td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>TOTALS</strong></td>
<td><strong>28</strong></td>
<td><strong>15</strong></td>
<td><strong>13</strong></td>
<td><strong>38</strong></td>
<td><strong>6</strong></td>
</tr>
</tbody>
</table>
RESULTS

In this series the results of 100 consecutive patients submitted to pneumoperitoneum are analysed; according to the indications already submitted; the patients have been reviewed up to the end of February 1948.

The age distribution and sex are shown in Table 2; it is seen that there are 51 females and 49 males. The youngest in this series is 14 and the oldest is 51.

The extent and severity of the disease has been classified according to the Classification of the American Tuberculosis Association; a copy of this classification is given in the Appendix. It is considered that this gives in actual words a better picture of the extent of lung disease than does the Classification of the Ministry of Health. Table 3 shows the age and sex distribution and classification of the disease in the 100 patients of this series. It is seen that of the 51 females, 27 are classified as far advanced (F.A.), 23 moderately advanced (M.A.) and 1 has a minimal lesion (Min.). The men show comparable figures; of 49 men treated, 26 are far advanced and 21 moderately advanced; 2 are described as minimal.

In the "indications for treatment" the site predominantly selected for treatment has been shown in the thirteenth column of the tables in the Appendix. The sites have been divided according to the zones involved; thus upper, middle and lower zones represent the radiological extent of the disease being actually treated. Many patients, however, fall outside the category of only one zone requiring attention; where the "indications for treatment" extend to two or more zones, these patients have been put into a special category (2 + Z). It may appear that this further refinement brings in an added complication to the result, but it is felt that only in this way is a truly representative idea of the extent of lung involved and requiring attention to be gained. In many patients of the far advanced group, the precise indication for treatment was confined to one zone, with possibly a future thoracoplasty in mind for the other zone(s). This will account for the difference in the tables showing 39 patients in the 2 + Z column, as compared with 53 far advanced males and females.

A further group has been added - "Pleura";
this represents a limited group where pneumoperitoneum was instituted after fluid or pus had appeared in an artificial pneumothorax; of the 6 patients in this group, 3 would be classified as far advanced and 3 moderately advanced.

Table 4 shows the zonal distribution of disease, for which pneumoperitoneum was primarily indicated, with the age distribution. It is seen that 28 patients had a primary indication for treatment in the upper zone, 15 in the middle and 13 in the lower zone; 38 patients had two or more zones involved, and 6 had pleural effusions or empyema.

The results have divided up according to whether these are successful, unsuccessful or dead.

In the "successful group" there are sub-divisions into "satisfactory" and "improved". By "satisfactory" is meant an individual with no clinical signs whatever, an X-Ray with no evidence of disease, and the sputum must be negative to either successive cultures or guinea-pig inoculation after gastric lavage (G.L.).

In the "improved group" are patients who have:

(1) Improved sufficiently on pneumoperitoneum as the sole measure that they have no clinical signs and the X-Ray suggests a controlled lesion, but in whom the sputum remains positive by gastric lavage.

(2) Improved sufficiently with extensive disease to make further collapse measures a possibility, but in whom there is not yet a satisfactory clinical or radiological evidence of a controlled lesion; the sputum remains positive. These patients are placed in this group because of considerable improvement under treatment.

As has already been explained in Rationale, at least two essentials are required for a successful outcome to pneumoperitoneum therapy: (a) an adequate rise of the corresponding diaphragm, and (b) the pleural space of the affected lung must not be obliterated. The "unsuccessful group" have been sub-divided, therefore, into three categories:

(1) Patients who have shown a poor rise of the diaphragm for any reason, or a fixed diaphragm.

(2) Patients whose lung was extensively adherent opposite the diseased site,
### Table 5

<table>
<thead>
<tr>
<th>AGE</th>
<th>Satisfactory</th>
<th>Improved</th>
<th>Poor rise of diaphragm</th>
<th>Adherent</th>
<th>Lung</th>
<th>Disease not controlled</th>
<th>Dead</th>
</tr>
</thead>
<tbody>
<tr>
<td>10 - 19</td>
<td>2</td>
<td>6</td>
<td>4</td>
<td>2</td>
<td>4</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>20 - 29</td>
<td>6</td>
<td>10</td>
<td>7</td>
<td>7</td>
<td>17</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>30 - 39</td>
<td>4</td>
<td>4</td>
<td>7</td>
<td>4</td>
<td>4</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>40 - 49</td>
<td>4</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>50+</td>
<td></td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TOTALS</td>
<td>12</td>
<td>21</td>
<td>19</td>
<td>16</td>
<td>26</td>
<td>6</td>
<td></td>
</tr>
</tbody>
</table>

### Table 6

<table>
<thead>
<tr>
<th></th>
<th>Min.</th>
<th>M.A.</th>
<th>F.A.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Satisfactory</td>
<td>1</td>
<td>8</td>
<td>3</td>
</tr>
<tr>
<td>Improved</td>
<td>7</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>TOTALS</td>
<td>1</td>
<td>15</td>
<td>17</td>
</tr>
</tbody>
</table>

### Table 7

<table>
<thead>
<tr>
<th>Duration of Pneumoperitoneum in months</th>
<th>3</th>
<th>4-6</th>
<th>7-9</th>
<th>10-15</th>
<th>16-24+</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>20</td>
<td>21</td>
<td>8</td>
<td>8</td>
<td>3</td>
</tr>
</tbody>
</table>
this obliterated pleural space being known either by previous attempt at artificial pneumothorax or by the complete failure of the diseased part of the lung to alter its position despite a satisfactory rise of the diaphragm.

(3) Patients in whom no reason is offered for the failure.

Table 5 shows the results in this series of patients according to age groups. 12 patients have a satisfactory result, and 21 are improved, i.e.; 33 give a successful result. There are 61 unsuccessful results; of these 19 were due to a poor rise of the diaphragm, and 16 due to adherent lung. No explanation is offered for 26 failures. 6 patients are dead.

It is thus seen that 33 per cent. of all the patients in this group were benefited completely, or in part, by pneumoperitoneum. Most of them had artificial pneumothorax tried unsuccessfully, and in many more of those patients, the disease was in an advanced state to make the prognosis poor. Table 6 shows the original classification of the disease in those 33 successful results, and it will be seen that 34 per cent. of the moderately advanced and 32 per cent. of the far advanced cases give a successful result. Considering the fact that little prospect of treatment could be offered those patients, these appear gratifying results.

If one considers the duration of treatment in patients who have had a pneumoperitoneum abandoned (Table 7), it will be seen that 20 patients have terminated treatment within three months of the induction of pneumoperitoneum. If these are ruled out of the final analysis of results, as one rules out ineffective pneumothorax from any final analysis, it will be seen that the successful results show a considerable improvement, rising to 41.25 per cent. of the remaining cases.

One patient (20) had pneumoperitoneum stopped abroad for what was probably inadequate reason eighteen months after the induction. He has a high paralysed diaphragm and remains sputum negative on culture, eighteen months after treatment was stopped. He is not considered in the successful results.

Of the patients who have had pneumoperitoneum continued (27 in this clinic and 3 elsewhere) 23 have had pneumoperitoneum for six months or more; 2 of
those patients (60 and 71) are known to be unsatisfactory results of pneumoperitoneum, and should probably have treatment stopped, except that both find sputum and cough are eased. Of the remaining continued cases 7 are successful and 23 have been improved.

4 patients have had treatment terminated for periods of 2½ years, 6, 4 and 4 months, respectively. Those patients have remained well since the termination of treatment. Of course, it cannot be contended that those few patients allow one to make any estimate of end results.

2 patients had pneumoperitoneum used in combination with artificial pneumothorax; the function of one appeared to have been fulfilled and a peritoneal effusion terminated the other.

The over-all results are considered highly favourable. Most of those patients have had artificial pneumothorax tried; many more had pneumoperitoneum induced as the acute nature of the disease contra-indicated pneumothorax treatment; in a number of others, pneumothorax was contra-indicated by the far advanced state of the disease. In the successful group, pneumoperitoneum is considered to justify itself by the satisfactory results seen in patients 18, 31 and 91. In the improved group it is not considered that from the clinical point of view control of the disease has been obtained; in many patients, especially those with disease in the lower zone, this may never be obtained, but at least the patients' general condition has been considerably improved, and with that the prospects of at least limiting the disease to one zone. A much more important result in the improved group is the prospect of utilising some other form of therapy which appeared quite beyond the patients' reach when they were first seen. Patient 31, for instance, in the absence of pneumoperitoneum, would have been faced with a prolonged period of invalidism, in the hope that the disease would eventually settle for other collapse measures. In the "2 + zone" group, where there is widespread and far advanced disease, the successful use of pneumoperitoneum in many of those patients makes the eventual hope of a successful thoracoplasty much more of a reality than was at first considered possible.

The initial details of the 100 patients in this series are shown in the Tables in the Appendix; these Tables should be used in conjunction with the results
### TABLE 8. RESULTS ACCORDING TO ZONAL DISTRIBUTION

<table>
<thead>
<tr>
<th></th>
<th>U.Z.</th>
<th>M.Z.</th>
<th>L.Z.</th>
<th>2+Z.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>TOTALS</strong></td>
<td>28</td>
<td>15</td>
<td>13</td>
<td>38</td>
</tr>
<tr>
<td>Satisfactory</td>
<td>4</td>
<td>2</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Improved</td>
<td>9</td>
<td>2</td>
<td>1</td>
<td>9</td>
</tr>
<tr>
<td>Poor rise of diaphragm</td>
<td>5</td>
<td>2</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>Adherent lung</td>
<td>5</td>
<td>2</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>Disease not controlled</td>
<td>4</td>
<td>8</td>
<td>5</td>
<td>8</td>
</tr>
<tr>
<td>Dead</td>
<td>1</td>
<td>1</td>
<td>4</td>
<td></td>
</tr>
</tbody>
</table>

### TABLE 9.

<table>
<thead>
<tr>
<th></th>
<th>SUCCESSFUL</th>
<th>INEFFECTIVE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper third</td>
<td>22</td>
<td>32</td>
</tr>
<tr>
<td>Middle third</td>
<td>5</td>
<td>19</td>
</tr>
<tr>
<td>Lower third</td>
<td>5</td>
<td>11</td>
</tr>
<tr>
<td></td>
<td>32</td>
<td>62</td>
</tr>
</tbody>
</table>
presented in the sections in the ensuing analysis of results by zonal distributions.

Table 8 shows the distribution of the lesions according to the zones indicated and the results obtained. It is seen that 13 patients had successful results with upper zone disease, 4 with mid-zone disease, 4 with lower zone disease, and 11 when two or more zones were involved.

In Table 9, the entire series of patients has been analysed according to the results obtained in upper, mid or lower zones (the 2 + Z has been further analysed into groups). From this Table it is thus seen that in the entire series we have 22 successful results in the upper zone, 5 in the mid-zone and 5 in the lower zone.

In both Tables the "Pleura" group has been excluded, and by its exclusion, will give a different total of final results from those shown in the earlier Tables; i.e., a total of 94 patients is now obtainable.
UPPER ZONE LESIONS

Table 10 shows the age distribution and results of upper zone lesions.

<table>
<thead>
<tr>
<th>Original distribution</th>
<th>10 - 19</th>
<th>20 - 29</th>
<th>30 - 39</th>
<th>40 - 49</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Satisfactory)</td>
<td>6</td>
<td>12</td>
<td>6</td>
<td>4</td>
<td>28</td>
</tr>
<tr>
<td>(Improved)</td>
<td>3</td>
<td>5</td>
<td>2</td>
<td>1</td>
<td>13</td>
</tr>
<tr>
<td>(Poor rise of)</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td></td>
<td>5</td>
</tr>
<tr>
<td>(Adherent lung)</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td></td>
<td>5</td>
</tr>
<tr>
<td>(Disease not controlled)</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td></td>
<td>4</td>
</tr>
<tr>
<td>(Dead)</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td>1</td>
</tr>
</tbody>
</table>

Of the patients in this group, 18 show lesions on the right and 10 on the left lung; 7 show recent, or fairly recent disease, and the rest cavities of the more chronic type.

13 patients are classified as successful results. These represent 39 per cent. of the total successful results. The failures are almost evenly distributed among the three groups of unsuccessful results. Patient (40) is dead. Until this patient took ill, she was considered as improved, and she was due to have thoracoplasty for the diseased left lung. Her death has never been adequately explained, but she is described as having had "tuberculous peritonitis".

Of all the patients in the entire series, 35 had upper lobe cavities on the right, and 15 on the left. 16 improved results were seen on the right, and 6 on the left. Those 16 patients with cavities on the right appear to have made more positive improvement than those with cavities on the left. Patients with predominantly upper lobe lesions have, in the presence of a free pleural space, an opportunity with pneumoperitoneum to allow a "swing round" of the diseased lobe against the mediastinum to perform a "medical lobectomy". This is seen in patients 18, 91 and 96 (2 of these patients are grouped under alternative
zones but the final result can be collectively discussed here; the limiting factor of an adherent lung is seen in Fig. 33, where the thick inter-lobar septum is obviously adherent to the chest wall.

It has been pointed out by Ellison and Tittle (37) that:

1. The angle that the right main stem bronchus makes with the line of the trachea is much greater than that of the left main stem.

2. The right main bronchus (the epiarterial) rises above the point where the pulmonary artery crosses the main stem bronchus; on the left the main stem bronchus does not divide until it is beyond the point of crossing of the artery.

3. There are more lymph nodes about the right hilar than the left (and especially the tracheo-bronchial group of glands which lie in direct relationship with the epiarterial bronchus).

By the combination of all these three factors, it is considered that a marked rise in the diaphragm on the right side frequently leads to a kinking of the epiarterial bronchus, resulting in an atelectasis of the right upper lobe, giving cavity closure.
## RESULTS BY INDICATIONS

### UPPER ZONE

<table>
<thead>
<tr>
<th>CASE</th>
<th>REASON FOR ABANDONING PNEUMOPERITONEUM</th>
<th>PRESENT STATE</th>
<th>SPUTUM</th>
<th>COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 K.C.</td>
<td>Progressive peritoneal adhesions.</td>
<td>Satisfactory; no obvious cavity.</td>
<td>+</td>
<td>0</td>
</tr>
<tr>
<td>3 D.H.</td>
<td>Ineffective; adherent interlobar septum.</td>
<td>Quiescent</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>16 T.C.</td>
<td>Ineffective; cavities unclosed.</td>
<td>Thoracoplasty has been done recently.</td>
<td>+</td>
<td>?</td>
</tr>
<tr>
<td>17 R.J.</td>
<td>Ineffective - adherent R. upper lobe and cavity formation.</td>
<td>Unchanged</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>22 J.K.</td>
<td>Continues</td>
<td>Not known</td>
<td>+</td>
<td>?</td>
</tr>
<tr>
<td>24 Mrs. L.</td>
<td>Ineffective - poor rise of diaphragm; cavities unclosed.</td>
<td>Unsatisfactory</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>27 R.A.</td>
<td>Patient did not attend for refills.</td>
<td>Unsatisfactory; disease remains active.</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>33 J.P.</td>
<td>Continues</td>
<td>Satisfactory</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>39 Mrs. McG.</td>
<td>To induce R.A.P. Poor rise of diaphragm.</td>
<td>Improved</td>
<td>+</td>
<td>0</td>
</tr>
<tr>
<td>40 Mrs. M.</td>
<td>Patient was said to have peritoneal effusion.</td>
<td>Died at home 3/12/48.</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>CASE</td>
<td>REASON FOR ABANDONING PNEUMOPERITONEUM</td>
<td>PRESENT STATE</td>
<td>SPUTUM ADM</td>
<td>NOW G.L.</td>
</tr>
<tr>
<td>--------</td>
<td>---------------------------------------</td>
<td>--------------------------------------------------------</td>
<td>------------</td>
<td>----------</td>
</tr>
<tr>
<td>43 Mrs. G.</td>
<td>Continues</td>
<td>Improved; doubtful if L. disease is controlled.</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>48 W.S.</td>
<td>Ineffective - no change in disease.</td>
<td>Unchanged</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>Small cavities persist.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>50 I.H.</td>
<td>Continues</td>
<td>Satisfactory</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>54 J.W.</td>
<td>Ineffective - lung adherent and cavity persists.</td>
<td>Un satisfactory</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>58 Mrs. A.</td>
<td>Continues</td>
<td>Improved</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>65 N.K.</td>
<td>Continues</td>
<td>Improved</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>74 J.L.</td>
<td>Onset of interlobar empyema</td>
<td>Improved, with streptomycin.</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>79 J. McL.</td>
<td>Continues</td>
<td>Improved</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>80 S.W.</td>
<td>Patient lost too much weight with L. A.P. and P. P.</td>
<td>Improved</td>
<td>+</td>
<td>G.L.</td>
</tr>
</tbody>
</table>
### UPPER ZONE (contd.)

<table>
<thead>
<tr>
<th>CASE</th>
<th>REASON FOR ABANDONING PNEUMOPERITONEUM</th>
<th>PRESENT STATE</th>
<th>SPUTUM</th>
<th>NOW</th>
<th>COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>83 H.L.</td>
<td>Ineffective - adherent lung.</td>
<td>Unsatisfactory</td>
<td>+</td>
<td>+</td>
<td>R. lung unchanged; spread to L. lower zone.</td>
</tr>
<tr>
<td>84 R.F.</td>
<td>Ineffective - poor rise of diaphragm.</td>
<td>Satisfactory</td>
<td>+</td>
<td>0</td>
<td>R. A.P. continued.</td>
</tr>
<tr>
<td>85 D.D.</td>
<td>Continues</td>
<td>Improved</td>
<td>+</td>
<td>+</td>
<td>G.L.</td>
</tr>
<tr>
<td>95 Mrs. M.</td>
<td>Continues</td>
<td>Improved</td>
<td>+</td>
<td>+</td>
<td>Small cavity persists in collapsed R. upper lobe.</td>
</tr>
<tr>
<td>96 B.A.</td>
<td>Continues</td>
<td>Improved</td>
<td>+</td>
<td>+</td>
<td>Satisfactory result expected (Fig.29-31)</td>
</tr>
<tr>
<td>97 I.K.</td>
<td>Continues</td>
<td>Improved</td>
<td>+</td>
<td>+</td>
<td>L. A.P. necessary.</td>
</tr>
</tbody>
</table>
MIDDLE ZONE LESIONS

Table 11 shows the distribution and results of middle zone lesions.

<table>
<thead>
<tr>
<th></th>
<th>10 - 19</th>
<th>20 - 29</th>
<th>30 - 39</th>
<th>40 - 49</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Original distribution</td>
<td>14</td>
<td>1</td>
<td>15</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Satisfactory)</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td>4</td>
</tr>
<tr>
<td>(Improved)</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Poor rise of diaphragm)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>11</td>
</tr>
<tr>
<td>(Adherent lung)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Disease not controlled)</td>
<td>7</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Dead)</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

It is seen that there are 4 successful results and 9 unsuccessful. The 4 successful results represent 12 per cent. of the total successes.

In this group there are 2 patients who have terminated treatment; one (20) 14 months ago and the second (12) 6 months ago. The former was not included in the successful results as his pneumoperitoneum was said to be ineffective when it was terminated abroad 18 months after the induction. Now he has a negative sputum (on culture) but he has a residual bronchiectasis (Figs. 16 and 17), from which he has periodic blood-stained sputum.

Cavities lying in the middle zone are, in many cases, hilar cavities and, as such, are usually occupying the apex of the lower lobe and are lying in the para-vertebral gutter; they are often widely adherent and, as a result, have little tendency to slide upwards with a possibly satisfactory result. Nevertheless, the 2 patients mentioned above had the only hilar cavities in this group. Another patient showing a mid-zone cavity is Case 42, who was treated by a combination of pneumoperitoneum and artificial pneumothorax until peritoneal effusion terminated the pneumoperitoneum (Figs. 4 - 6). Another patient (78), classified elsewhere, shows a large mid-zone cavity and its failure to respond to treatment
(Figs. 37 - 40).

Most of the patients in this group had mid-zone infiltrations associated with large cavities on the opposite lung. The primary consideration then, was to control the mid-zone lesion prior to later thoracoplasty for the contra-lateral disease; 2 patients (87 and 94) appear to have given satisfactory results.

2 patients with typical tension cavities were not helped by pneumoperitoneum; in one (52) the tension cavity came to occupy most of the mid-zone.
# RESULTS BY INDICATIONS

## MIDDLE ZONE

<table>
<thead>
<tr>
<th>CASE</th>
<th>REASON FOR ABANDONING PNEUMOPERITONEUM</th>
<th>PRESENT STATE</th>
<th>SPUTUM</th>
<th>NOW</th>
<th>COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 M.W.</td>
<td>Widespread abdominal adhesions; peritoneal effusion.</td>
<td>Quiescent</td>
<td>+</td>
<td>0</td>
<td>Control of disease from phrenic evulsion.</td>
</tr>
<tr>
<td>12 A.S.</td>
<td>Satisfactory result.</td>
<td>Quiescent</td>
<td>+</td>
<td>0</td>
<td>P.p. too recently abandoned for definite opinion.</td>
</tr>
<tr>
<td>20 R.S.</td>
<td>Apparently ineffective; abandoned abroad.</td>
<td>Quiescent</td>
<td>+</td>
<td>cult.</td>
<td>This man has a high paralysed diaphragm (Fig.16,17)</td>
</tr>
<tr>
<td>42 M.A.</td>
<td>Peritoneal effusion.</td>
<td>Satisfactory</td>
<td>+</td>
<td>0</td>
<td>L. A.P. continues (Fig.4,5 &amp; 6)</td>
</tr>
<tr>
<td>52 M.W.</td>
<td>Ineffective - developed large R. tension cavity.</td>
<td>Unsatisfactory</td>
<td>+</td>
<td>+</td>
<td>Associated disease of L. lung.</td>
</tr>
<tr>
<td>CASE</td>
<td>REASON FOR ABANDONING PNEUMOPERITONEUM</td>
<td>PRESENT STATE</td>
<td>SPUTUM</td>
<td>COMMENTS</td>
<td></td>
</tr>
<tr>
<td>-------</td>
<td>----------------------------------------</td>
<td>---------------</td>
<td>--------</td>
<td>----------</td>
<td></td>
</tr>
<tr>
<td>59 H. McI.</td>
<td>Ineffective</td>
<td>Improved</td>
<td>+</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>61 E.C.</td>
<td>Ineffective - diaphragm fixed.</td>
<td>Unsatisfactory</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>77 Mrs. H.</td>
<td>Ineffective</td>
<td>Died 22/12/48</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>87 C.C.</td>
<td>Continues</td>
<td>Improved</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>93 M.W.</td>
<td>Continues</td>
<td>Improved</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>99 J.S.</td>
<td>Ineffective - tension cavity unchanged.</td>
<td>Unsatisfactory</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
</tbody>
</table>

Patient has moved elsewhere.
R. lung shows further deterioration.
R. lung has improved too.
LOWER ZONE LESIONS

Table 12 shows the distribution and results of lower zone lesions.

<table>
<thead>
<tr>
<th></th>
<th>10 - 19</th>
<th>20 - 29</th>
<th>30 - 39</th>
<th>40 - 49</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Original distribution</td>
<td>2</td>
<td>7</td>
<td>3</td>
<td>1</td>
<td>13</td>
</tr>
<tr>
<td>(Satisfactory)</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>(Improved)</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Poor rise of diaphragm)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Adherent lung)</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
<td>9</td>
</tr>
<tr>
<td>(Disease not controlled)</td>
<td>1</td>
<td>3</td>
<td>1</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The satisfactory results in this group represent 12.4 per cent. of the total successes.

Despite the fact that 4 out of 13 patients with lower zone lesions have shown good results, we have not been wholly impressed with pneumoperitoneum in the treatment of basal disease. Lesions in this situation are always presenting great difficulty to any physician, for thoracoplasty used for such disease means a very extensive operation with, unfortunately, little prospect of success, and a great loss of normally functioning lung tissue. Neither has artificial pneumothorax been entirely successful, for many of those lower lobes show an absorption collapse after pneumothorax has been induced, indicative in many cases of a tracheo-bronchial tuberculosis. It has been pointed out that this is three times as common in the lower lobe as in the upper (71). 2 patients (75 and 82) have had artificial pneumothorax induced after a failure of pneumoperitoneum; an element of doubt remains as to whether in both these cases there is not an associated tuberculosis of the bronchi.

In many cases there is failure to induce a satisfactory pneumothorax after the pneumoperitoneum has failed, in view of extensive diaphragmatic or pleural adhesions (Figs. 20 and 21).

Nevertheless, many authors favour the use of pneumoperitoneum in lower lobe disease (46, 69), and
consider that this is the primary method of treatment of all available collapse measures. Since basal tuberculosis provides an exceedingly complicated situation for therapy, it might be that pneumoperitoneum should be attempted in all cases before any other form of treatment is considered.

In connection with lower lobe lesions, it has been pointed out by Ellison and Tittle that lower zone lesions may not be influenced by a satisfactory rise of the diaphragm, since they consider that the cavity may simply rise up on the diaphragm "like a bubble on a wave". They also point out that cavities in the base may be entirely unaffected by the diaphragm rise; they account for this by noting that the attachments of the diaphragm are fixed and that stretching of the dome of the diaphragm upwards only results in the normal triangular space behind the dome of the diaphragm becoming exaggerated; if the lung is adherent at the costo-phrenic angle, there is no possibility of any rise of the diaphragm to obliterate this triangular space with respiration. This fact has been demonstrated radiologically by Morgenstern and Pine (60). The same appearance is suggested in Fig. 39, where, although the cavity lies at a considerably higher level than the basal cavities mentioned here, it will be seen just how the diaphragm in this patient, despite an apparently satisfactory rise, would be completely ineffective in closing the cavity indicated.
## RESULTS BY INDICATIONS

### LOWER ZONE

<table>
<thead>
<tr>
<th>CASE</th>
<th>REASON FOR ABANDONING PNEUMOPERITONEUM</th>
<th>PRESENT STATE</th>
<th>SPUTUM</th>
<th>COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>23 Mrs. K.</td>
<td>Continues.</td>
<td>Satisfactory but ? cavity closure.</td>
<td>+ 0</td>
<td>See (Figs.18,19)</td>
</tr>
<tr>
<td>26 T.A.</td>
<td>Continues.</td>
<td>Satisfactory</td>
<td>+ G.I.</td>
<td></td>
</tr>
<tr>
<td>36 F.L.</td>
<td>Continues.</td>
<td>Improved</td>
<td>+ +</td>
<td>Small cavity persists.</td>
</tr>
<tr>
<td>56 A.C.</td>
<td>Ineffective</td>
<td>Unsatisfactory</td>
<td>+ +</td>
<td>Not fit enough for L. thoracoplasty.</td>
</tr>
<tr>
<td>69 Mrs. F.</td>
<td>Continues</td>
<td>Satisfactory</td>
<td>+ 0</td>
<td></td>
</tr>
<tr>
<td>75 Mrs. I.</td>
<td>Ineffective</td>
<td>Improved</td>
<td>+ -</td>
<td>L. A.P. induced 26/10/48 to close basal cavity.</td>
</tr>
<tr>
<td>81 B.M.</td>
<td>Ineffective</td>
<td>Unsatisfactory</td>
<td>+ +</td>
<td>Started streptomycin.</td>
</tr>
<tr>
<td>82 A.P.</td>
<td>Ineffective despite good rise of diaphragm.</td>
<td>Unsatisfactory</td>
<td>+ +</td>
<td>Ineffective L. A.P. (Figs.20 &amp; 21)</td>
</tr>
<tr>
<td>86 H.S.</td>
<td>Progressive abdominal adhesions.</td>
<td>Unsatisfactory</td>
<td>+ +</td>
<td>Lung lesion is unchanged.</td>
</tr>
<tr>
<td>94 Mrs. H.</td>
<td>Continues</td>
<td>Improved</td>
<td>+ +</td>
<td></td>
</tr>
</tbody>
</table>
Table 13 shows the distribution and results of two or more zone lesions.

<table>
<thead>
<tr>
<th></th>
<th>10 - 19</th>
<th>20 - 29</th>
<th>30 - 39</th>
<th>40 - 49</th>
<th>50+</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Original</td>
<td>8</td>
<td>17</td>
<td>9</td>
<td>3</td>
<td>1</td>
<td>38</td>
</tr>
<tr>
<td>distribution</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Satisfactory)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Improved)</td>
<td>3</td>
<td>2</td>
<td>4</td>
<td>1</td>
<td></td>
<td>11</td>
</tr>
<tr>
<td>(Poor rise of</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(diaphragm)</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td></td>
<td>6</td>
</tr>
<tr>
<td>(Lung adherent)</td>
<td>2</td>
<td>4</td>
<td>1</td>
<td>1</td>
<td></td>
<td>9</td>
</tr>
<tr>
<td>(Disease not</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>controlled</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Dead)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

This is a large group of far advanced patients with disease covering more than one zone; it was considered that in the majority of those patients no satisfactory grouping was available except by the one now provided. Reference to the original X-Ray plates shows many patients with lesions predominantly in one zone, but radiologically the disease extends much further. In certain plates no exact localization to one zone is possible. However, cavitation in this group can be demonstrated in the upper zone of 34 patients, in the middle zone of 9, and in the lower zone of 2.

The 11 successful results in this group represent 33.3 per cent. of the total successful results. In this group, too, are included the favourable results seen in patients (18, 31 and 91). Those results appear quite remarkable. Patient (18) had the left diaphragm paralysed so that with satisfactory improvement on the left side a right thoracoplasty could be undertaken; there was, however, coincidental improvement on this side (Figs. 27 and 28). Patient (31) shows similar improvement, although this is not yet an end result, since other collapse measures are indicated.
Several other patients in this group show the same trend - improvement up to a degree, so that the ipso-lateral or the contra-lateral lung might be subject to a more lasting thoracoplasty. The improvement in the general condition of these patients has been quite pronounced. However, despite those successes, there is the large number of unsuccessful results. No one factor appears to be primarily responsible, but when one considers that all those patients have old-standing chronic lesions, it is not surprising that pneumoperitoneum is ineffective. Whereas, the successes are mostly in acute exudative lesions, the failures are many in the chronic type of disease.

2 patients in this group have unsuccessful pneumoperitoneums continued in deference to the patients' wishes, as cough and sputum are apparently made easier with pneumoperitoneum than without it.

One patient in this group (100) shows a tuberculous consolidation of the upper half of the right lung, with some spread into the apex of the lower lobe (Fig. 22). Although there is some clearing of the lower lobe disease, the upper lobe has not changed its position much, and has now developed a cavity (Fig. 23). Wade (81) recommended pneumoperitoneum and phrenic crush for tuberculous consolidation; others also claim successful results. But this one patient has obviously not been affected; in addition he has developed tuberculous peritonitis.

2 patients were submitted for thoracoplasty; they were improved by pneumoperitoneum but not to the point that they could be considered in the definition of "improved" patients; and, in the final analysis, their pneumoperitoneums have been considered ineffective.

One patient is a diabetic; he had a rapidly advancing lesion when first seen, but has now been stabilised for the past year on pneumoperitoneum. He refuses thoracoplasty.

One patient had pneumoperitoneum re-induced after an interval of almost 5 months; no difficulty was encountered in resuming refills, but the patient was so obviously going downhill with a tuberculous enteritis that the pneumoperitoneum was stopped. Corrigan (25) has suggested that extensive adhesions might prevent successful re-induction of pneumoperitoneum. So does Keers (53), but Anderson and Winn consider pneumoperitoneum should be re-started easily.
### RESULTS BY INDICATIONS

#### TWO OR MORE ZONES

<table>
<thead>
<tr>
<th>CASE</th>
<th>REASON FOR ABANDONING PNEUMOPERITONEUM</th>
<th>PRESENT STATE</th>
<th>SPUTUM</th>
<th>ADM</th>
<th>NOW</th>
<th>COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>4 E.C.</td>
<td>Ineffective - large cavities unclosed.</td>
<td>Extensive bilateral disease.</td>
<td>+</td>
<td>+</td>
<td></td>
<td>Thoracoplasty done on 11/3/46</td>
</tr>
<tr>
<td>7 E.D.</td>
<td>Ineffective</td>
<td>Died in October 1946</td>
<td>+</td>
<td></td>
<td></td>
<td>Far-advanced disease.</td>
</tr>
<tr>
<td>8 J. McI.</td>
<td>Poor diaphragmatic rise; severe abdominal pains.</td>
<td>Unchanged</td>
<td>+</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9 W.R.</td>
<td>Ineffective</td>
<td>Died on 17/10/47</td>
<td>+</td>
<td></td>
<td></td>
<td>Disease advanced rapidly after premature discharge.</td>
</tr>
<tr>
<td>10 A. McE.</td>
<td>Continues</td>
<td>Small cavity persists in R. upper-zone. Improved.</td>
<td>+</td>
<td>+</td>
<td>G.L.</td>
<td>Thoracoplasty is now necessary.</td>
</tr>
<tr>
<td>14 H.G.</td>
<td>Ineffective - poor rise of L. diaphragm.</td>
<td>Satisfactory</td>
<td>+</td>
<td>0</td>
<td></td>
<td>Condition settled with bed rest.</td>
</tr>
<tr>
<td>18 J.S.</td>
<td>Continues</td>
<td>Satisfactory</td>
<td>+</td>
<td>-</td>
<td>cult.</td>
<td>R. upper lobe shows excellent result (Fig. 28)</td>
</tr>
<tr>
<td>19 Mrs. J.</td>
<td>Ineffective - cavities unclosed.</td>
<td>Unsatisfactory</td>
<td>+</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CASE</td>
<td>REASON FOR ABANDONING PNEUMOPERITONEUM</td>
<td>PRESENT STATE</td>
<td>SPUTUM</td>
<td>COMMENTS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-------</td>
<td>--------------------------------------</td>
<td>--------------</td>
<td>--------</td>
<td>----------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>28 T.M.</td>
<td>Ineffective - poor rise of diaphragm.</td>
<td>Improved</td>
<td>+</td>
<td>+</td>
<td>Thoracoplasty on 28/11/47. Spread to L. lower-zone.</td>
<td></td>
</tr>
<tr>
<td>31 C.B.</td>
<td>Continues</td>
<td>Greatly improved</td>
<td>+</td>
<td>+</td>
<td>See Fig. 35, 36.</td>
<td></td>
</tr>
<tr>
<td>34 J.T.</td>
<td>Continues</td>
<td>Improved</td>
<td>+</td>
<td>+</td>
<td>Concurrently with L. A.P. Large upper lobe cavity persists (Fig. 34)</td>
<td></td>
</tr>
<tr>
<td>35 C.M.</td>
<td>Continues</td>
<td>Improved</td>
<td>+</td>
<td>+</td>
<td>Small cavity persists. Thoracoplasty refused.</td>
<td></td>
</tr>
<tr>
<td>37 McK. B.</td>
<td>Ineffective</td>
<td>Died 11/2/48</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>38 T.H.</td>
<td>(1) To induce L. A.P. (2) Ineffective</td>
<td>Going rapidly downhill</td>
<td>+</td>
<td>+</td>
<td>P.P. was stopped and re-induced.</td>
<td></td>
</tr>
<tr>
<td>41 J.K.</td>
<td>Continues</td>
<td>Improved</td>
<td>+</td>
<td>+</td>
<td>X-Ray &amp; clinically a satisfactory result.</td>
<td></td>
</tr>
<tr>
<td>49 W.S.</td>
<td>Ineffective - poor rise of diaphragm.</td>
<td>Improved after prolonged bed-rest</td>
<td>+</td>
<td>+</td>
<td>Improvement of the infiltration of both lungs.</td>
<td></td>
</tr>
<tr>
<td>51 E.B.</td>
<td>Ineffective - poor rise of diaphragm.</td>
<td>Unsatisfactory</td>
<td>+</td>
<td>+</td>
<td>Air gathered under the paralysed R. diaphragm.</td>
<td></td>
</tr>
<tr>
<td>Case</td>
<td>Reason for Abandoning Pneumoperitoneum</td>
<td>Present State</td>
<td>Sputum</td>
<td>Comments</td>
<td></td>
<td></td>
</tr>
<tr>
<td>------</td>
<td>--------------------------------------</td>
<td>--------------</td>
<td>--------</td>
<td>----------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>60 J.P.</td>
<td>Continues</td>
<td>Un satisfactory</td>
<td>+</td>
<td>Spread to L. (Fig. 1 - 3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>62 E.R.</td>
<td>Ineffective - developed large R. tension cavity.</td>
<td>Un satisfactory</td>
<td>+</td>
<td>Large L. tension cavity.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>63 Mrs. McL.</td>
<td>Ineffective - cavity unaffected.</td>
<td>Un satisfactory</td>
<td>+</td>
<td>Large R. tension cavity.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>64 M. McL.</td>
<td>Ineffective - disease unaffected.</td>
<td>Un satisfactory</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>70 Mrs. I.</td>
<td>Ineffective - lung adherent.</td>
<td>Un satisfactory</td>
<td>+</td>
<td>L. thoracoplasty required.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>71 M.Q.</td>
<td>Continues - an ineffective P.P.</td>
<td>Un satisfactory</td>
<td>+</td>
<td>(Fig. 37 - 40)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>72 W.H.</td>
<td>Continues</td>
<td>Improved</td>
<td>+</td>
<td>R. thoracoplasty indicated.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>76 G.N.</td>
<td>Continues</td>
<td>Improved</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>78 B.C.</td>
<td>Ineffective - lung adherent</td>
<td>Un satisfactory</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>83 J.O.</td>
<td>Continues</td>
<td>Improved</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>89 A.M.</td>
<td>Ineffective - poor rise of diaphragm.</td>
<td>Un satisfactory</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CASE</td>
<td>REASON FOR ABANDONING PNEUMOPERITONEUM</td>
<td>PRESENT STATE</td>
<td>SPUTUM</td>
<td>COMMENTS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>--------</td>
<td>----------------------------------------</td>
<td>---------------</td>
<td>--------</td>
<td>-----------------------------------------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>90 C.C.</td>
<td>Ineffective - poor rise of diaphragm; lung adherent.</td>
<td>Unsatisfactory</td>
<td>+</td>
<td>+ There is tracheobronchial tuberculosis.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>91 M.B.</td>
<td>Continues</td>
<td>Improved</td>
<td>+</td>
<td>+ Satisfactory result expected (Fig. 24-26)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>92 D.C.</td>
<td>Unnecessary; function fulfilled.</td>
<td>Improved</td>
<td>+</td>
<td>L. A.P. has provided adequate collapse.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>100 N.H.</td>
<td>Ineffective - lung adherent; large cavity appears.</td>
<td>Unsatisfactory</td>
<td>+</td>
<td>Tuberculous peritonitis on 26/2/49. (Fig. 22, 23)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
This small group has been excluded from statistical analysis, for in only one case was a successful result obtained (3.4 per cent. of successful cases), and continued for more than three months. Another case was known to be ineffective, and only kept going at the patient's request; it was eventually abandoned when there was spread of disease in the left lung.

In this group, fluid or pus had been present for several months before pneumoperitoneum was instituted; by that time, the diaphragm was firmly fixed by adhesions and could not be affected by pneumoperitoneum. To be effective, pneumoperitoneum would require to be instituted directly fluid was observed in an artificial pneumothorax. It will not suffice to subject the effusion to prolonged aspiration, and then to institute pneumoperitoneum in an effort to re-expand an "inexpansible lung". The thickened visceral and parietal pleurae invariably limit any help from pneumoperitoneum.
## RESULTS BY INDICATIONS

### PLEURA

<table>
<thead>
<tr>
<th>CASE</th>
<th>REASON FOR ABANDONING PNEUMOPERITONEUM</th>
<th>PRESENT STATE</th>
<th>SPUTUM</th>
<th>COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>25 H.M.</td>
<td>Ineffective – poor rise of L. diaphragm.</td>
<td>Improved</td>
<td>-</td>
<td>0</td>
</tr>
<tr>
<td>32 H.R.</td>
<td>Ineffective – poor rise of L. diaphragm.</td>
<td>Improved – lung eventually re-expanded</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>44 A.S.</td>
<td>Ineffective – R. diaphragm almost fixed by adhesions.</td>
<td>Unsatisfactory</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>45 A.H.</td>
<td>Ineffective – poor rise of L. diaphragm.</td>
<td>Unsatisfactory</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>67 J.E.</td>
<td>Continued (when last seen)</td>
<td>Not known</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>
DISCUSSION

In the use of any collapse therapy programme, the end results alone will provide a basis on which to determine the efficacy of any particular method of treatment, but so few patients have had pneumoperitoneum terminated for a long enough period, that comparison with other methods of therapy is not yet possible. Discussing therapy, Rafferty states "because of the factors inherent in the tuberculosis problem, there are not now, and may not be for some time, any statistically sound data indicating the real effects of properly administered collapse therapy on prognosis". He laid emphasis on "properly administered", since so many poor end results eventuate when alternative means of treatment could be made available.

With artificial pneumothorax therapy, a growing number of physicians are becoming aware of the limiting factors of this form of treatment; but many of these factors would never arise if pneumoperitoneum were included in the collapse therapy programme. For many, this might indicate that the pendulum has indeed swung so far to one extreme, that pneumoperitoneum is now going through a "phase of overuse and over-enthusiasm". The realisation of the bad end results associated with pneumothorax complicated by fluid or pus, precipitates many physicians into other forms of therapy. A satisfactory artificial pneumothorax is probably initiated in some 10 per cent. of all patients subjected to a complete collapse programme; those patients still have to undergo the hazards of continued pneumothorax treatment. In other patients with adhesions, there is an associated risk from thoracoscopy and adhesion section, which means a not inconsiderable risk of losing the pneumothorax by reason of effusion, blood (5 per cent.), or empyema (10 per cent.) (84). All these complications have been seen following after adhesion section, and there is always a doubt raised in one's mind whether pneumoperitoneum as an initial procedure might not have eventually produced a much more satisfactory result for many of those patients.

It has already been shown that pneumoperitoneum is associated with few serious complications, and as a form of treatment, is as easily managed (probably more so) than artificial pneumothorax; it is not associated with any disturbance of the pleura to lead to effusions and empyema.

In this series it is claimed to have justified its place as a measure to be used in a collapse therapy.
programme by reason of the good results obtained; it has improved several patients quite markedly; some patients are now candidates for thoracoplasty, others will, it is hoped, become so; several patients have refused to have thoracoplasty. Many patients with basal lesions would not be submitted to this operation in view of the known difficulties in obtaining control of basal disease. It is not maintained that pneumoperitoneum may be the final stage in any one patient's treatment; it has, indeed, been frequently mentioned that one of the main indications is to so improve a patient's general condition that further collapse measures can be used with a greater hope of success. Keers, in a personal communication, adopts this view. However, like Keers, it has been noted that "in some cases where the original objective was a comparatively limited one, the ultimate result far exceeded expectations". Several such patients have been described in this series; their condition has been so markedly improved, clinically and radiologically, that for them thoracoplasty is no longer seriously considered. In the successful cases already discussed it is apparent that in pneumoperitoneum, a reversible procedure has been instituted for an irreversible thoracoplasty.

In all treatment, the fundamental fact of cavity closure remains the basis of treatment; it has been shown by Barnes and Barnes (12) that the average duration of life in patients with recognisable symptoms (from the first onset of symptoms) was 36 months in all cases of tuberculosis. In a more recent discussion, Marshall (56) states "my experience is that if you do not close the cavity the patient will not recover".

Drolet, in 1938, stated that "the ratio of deaths to new cases of tuberculosis reported has varied little during the past 20 years". In the same article, he also states that sanatorium and surgical treatment of pulmonary tuberculosis would seem so far to have had little effect upon the case fatality rates of the entire tuberculosis population. Commenting on this paper Amberson (1) states that Drolet's conclusions might be restated thus "it is not demonstrated that that treatment of advanced tuberculosis has had a very significant effect on case fatality rates". He goes on to point out that 80 per cent, of all admissions to sanatoria are in an advanced state; in most cases, then, "it can hardly be said that the treatment applied is calculated to be curative".

By 1943, Drolet had revised his original opinion, and then considered that collapse measures, especially thoracoplasty, undoubtedly achieve results in far advanced cases of tuberculosis that were never attained
before. Potter (65) considered that the death rate in patients with and without collapse therapy is decidedly lower in the former group. He points out that the death rates are overshadowed by deaths of hopeless and nearly hopeless far advanced cases admitted to institutions.

In this series, 53 far advanced patients were given pneumoperitoneum, and 18 of these have improved sufficiently for them to be considered as successful results; of this number, 8 have been considered, or will be considered, for thoracoplasty; 3 have refused the operation. It may be contended that these results might equally well have been obtained by bed rest alone; no doubt many would. However, it is considered that patients improved by pneumoperitoneum will now require a much more limited operation than originally seemed likely; in addition, pneumoperitoneum has stabilised those patients in a much better general condition than would otherwise have been the case.

Fales and Beaudet (39) discussed cavity closure in 1934, and maintained that a large percentage of cavities could be healed by strict bed rest alone, and quoted impressive figures; most notably 66 per cent. of 29 exudative lesions were healed within 10 months. More recently, Grenville-Mathers (45) found 45 per cent. cavity closure after 10 months in 37 patients. There is no doubt, therefore, that cavities can heal by bed rest as the sole measure; but pneumoperitoneum does "speed up" this process and gives a higher percentage of successful cavity closure. It is not intended that this should be done in the absence of bed rest; it has already been stressed that patients should be kept in bed until clinical signs have gone or, in the more chronic cases, are showing considerable clinical improvement. Bed rest and pneumoperitoneum are not two competing forms of therapy; each is necessary to the other, and both are essential in the same programme. Davies (31) has stated "there is much to be said for the sanatorium physician who.....puts his patient to bed ......watches his response to sanatorium regime and balances the pros and cons before putting the patient up for ancillary treatment". He goes on to remark that his most pleasing successes are those healed by sanatorium treatment alone.

Patient (97) in this series illustrates the value of bed rest alone; this girl was in bed at home for 7 months, awaiting admission to the sanatorium (Fig. 29). On admission, it is seen (Fig. 30) that there has been considerable retraction of the diseased upper lobe behind the clavicle; but with the
addition of pneumoperitoneum, there has been a quite dramatic swing towards the mediastinum after 3 months. This girl is believed to be entering the "improved" category when, if needed, a very much more limited thoracoplasty would be required than might have ever been considered possible.

In discussing the use of primary thoracoplasty, various authors argue its value from the standpoint of artificial pneumothorax. Rafferty, for example, wonders if there is any logic in the routine use of (or attempt at) artificial pneumothorax in all cases requiring major collapse therapy. He considers that this attitude is based largely on habit and precedence. Shipman (75) also favours the use of primary thoracoplasty in preference to artificial pneumothorax. None of those authors considers the possible use of pneumoperitoneum in this connection. From the evidence presented in the results, it is maintained that its routine use, prior to thoracoplasty, except in thick-walled cavities, should be considered. This is in complete agreement with Crow and Whelchel's comment - where artificial pneumothorax has failed "we are of the definite opinion that thoracoplasty should not be resorted to immediately......provided the lesions are largely exudative, and that cavities have thin, or moderately thin, walls. The use of phrenic interruption and pneumoperitoneum will effect a complete cure in many such patients, without the necessity of resorting to thoracoplasty".

It is really those acute exudative lesions that show the most dramatic results with pneumoperitoneum; it is precisely those acute lesions that are contra-indicated for artificial pneumothorax since they are so frequently complicated by effusions and empyema. It has been stated that artificial pneumothorax is not always the procedure of choice, merely because major collapse therapy is indicated (67). Numerous authors have commented on the adverse effect of an artificial pneumothorax complicated by effusion, and all tuberculosis physicians would consider the shrunken hemithorax following on empyema, a state of affairs to be avoided at all costs. Shipman quotes Cournand and Richards to show considerable impairment of pulmonary function following uncomplicated artificial pneumothorax. Birath (17) has shown more dramatically the end results of pneumothorax treatment and the effect of fluid in reducing respiratory efficiency. He pointed out that at the end of pneumothorax, the pleural surfaces may become adherent and cause an inefficient ventilation of relatively large parts of the lungs. This end result, in so far as can be seen at present, is not seen with pneumoperitoneum.
As has been indicated, large thick-walled cavities are not likely to respond to pneumoperitoneum; Potter (65) has pointed out that those cavities resist even manual compression at post-mortem. Huge apical cavities, cavities lying under thick visceral pleura with an adherent lung, or peripherally located cavities with widespread adhesions are not likely to respond to pneumoperitoneum. As has been emphasized already, wherever the pleural surfaces are widely adherent, a successful pneumoperitoneum cannot be expected. Tension cavities due to bronchial stenosis are not successfully treated by pneumoperitoneum. Indeed, bronchial stenosis is an unwelcome complication which is considered to contra-indicate pneumoperitoneum.

In the actual management of pneumoperitoneum, patients should be frequently assessed to determine the value of the treatment; three-monthly reviews are essential. X-Ray control is readily obtained with pneumoperitoneum, and it can be readily appreciated which lesions are satisfactory and those that are not; a widely adherent lung is easily detected, and where there is a thickened inter-lobar septum, it will act as an obvious indicator as to how much actual collapse (if any) has occurred. With dense lesions, the use of Tomography is strongly recommended to demonstrate cavities which are frequently not obvious in the straight X-Ray film, or whose size is not quite appreciated. Probably one of the most satisfactory features in pneumoperitoneum is the fact that in most patients the progress of the disease can be followed with the stethoscope.
SUMMARY

1. The literature on pneumoperitoneum has been reviewed, and it shows a decided trend in favour of the use of pneumoperitoneum as a collapse measure.

2. Pneumoperitoneum is a relatively simple procedure to use.

3. An account has been offered of the mode of action of pneumoperitoneum in the control of lung disease. It is considered that pneumoperitoneum acts by "relaxation", and that the rise of the diaphragm is not, of itself, the predominant factor in the results obtained.

4. The phrenic nerve need not necessarily be paralysed to obtain results, and in view of the number of paralysed diaphragms which may result following on phrenic crushing, it is considered that pneumoperitoneum should, in the first instance, be used without phrenic paralysis. If indications become very definite, phrenic nerve paralysis can be added to provide more adequate collapse. In this series 10 per cent. of patients still have paralysed diaphragms.

5. There are few complications associated with pneumoperitoneum. The following have been found in this series:-

   Peritoneal effusion ... 5
   Tuberculous Peritonitis ... 3
   Obliterative Peritonitis ... 2
   Lobar Atelectasis ... 1

6. The indications for the use of pneumoperitoneum therapy can be summarised as follows:-

   (a) Where artificial pneumothorax has failed.
   (b) To give increased relaxation in a partially effective artificial pneumothorax.
   (c) Exudative disease, either unilateral or bilateral.
   (d) With the object of helping a patient with bilateral disease to advance to other collapse measures, notably thoracoplasty.
(e) To close empyema spaces.

(f) For the control of haemoptysis.

7. From a review of the cases presented here, few contra-indications were found, except a very important one - tracheo-bronchial tuberculosis - which is deemed to be an absolute contra-indication to pneumoperitoneum therapy.

8. The results of the patients reviewed here show 41.25 per cent. successful.

9. Pneumoperitoneum is considered to be a procedure of value, and should have a place in any collapse therapy programme.
REFERENCES

(18) BROMPTON HOSPITAL REPORTS, 1944 to 1947, statistics.


(21) CLIFFORD-JONES, E. and MACDONALD, N., 1943, 'Pneumoperitoneum in the collapse therapy of pulmonary tuberculosis', Tubercle, 24, 97.


(23) COHEN, R.C., 1948, 'Peritoneal effusion as a complication of artificial pneumoperitoneum', Lancet, ii, 1006.


(30) DAVIES, H.M., 1933, 'Pulmonary Tuberculosis'.


(32) DRINKER, C.K., 1945, 'Pulmonary oedema and inflammation'.


(36) EDWARDS, P.W. and LOGAN, J., 1945, 'Pneumoperitoneum in pulmonary tuberculosis', Tubercle, 26, 11.


FISHBERG, M., 1932, Pulmonary Tuberculosis (London).


FOX, W., 1948, 'Dissociated paralysis of the diaphragm following phrenic crush and pneumoperitoneum', Thorax, 3, 15.


HEAF, F. and RUSBY, N.L., 1948, Recent advances in respiratory tuberculosis.


LAIRD, R., 1945, 'Comments on total thoracoscopy', Tubercle, 26, 149.


(60) MORGENSTERN, P.; and PINE, I.; 1948, 'Pulmonary cavities below the diaphragm', Am. J. Radiology, 59, 67.


(63) PINNER, M.; 1945, Pulmonary Tuberculosis in the Adult.

(64) PINNER, M.; 1946, Book Reviews, Am. Rev. Tuberc., 54, 589.

(65) POTTER, B.P.; 1941, 'Results of collapse therapy', Am. Rev. Tuberc., 43, 184.


(67) RAFFERTY, T.N.; 1945, Artificial Pneumothorax in Pulmonary Tuberculosis.


(87) BRIAN, E.G., and RICEN, E., 1941, 'Collapse therapy of pulmonary tuberculosis by pneumoperitoneum', ibid, 39, 391.


(89) HOWLETT, K.S., 1948, Editorial, ibid, 58, 134.


Grateful acknowledgment is made to Dr. B. W. Anderson, Medical Superintendent, Glenlomond, by Kinross, for his permission to make use of the material on which this work is based, and for providing every facility to make it possible.
APPENDIX

CASE HISTORIES

SCHEMA OF CLASSIFICATION OF PATIENTS

TABLES WITH DETAILS OF 100 PATIENTS
I.H.: female, age 24; admitted 20/10/47

This patient had a history of cough for seven months; she had also felt tired and had lost weight. She began to cough up sputum too, which was blood-stained for one day.

Clinically, there was a lesion at the right upper zone. The sputum was T.B. Positive and the B.S.R. was 43. Her weight was 9 sts.

23/10/47. X-Ray showed fibrotic infiltration of the right upper lobe with small cavities lying below the clavicle.

28/10/47. A right sided artificial pneumothorax was induced, but it was adherent over the apex and was contra-selective; it was abandoned forthwith.

4/11/47. The right phrenic nerve was crushed with paralysis of the right hemi-diaphragm.

7/11/47. A pneumoperitoneum was induced.

14/5/48. X-Ray showed a considerable improvement of the diseased area of the right upper lobe, but there were still suggestive cavities at the apex.

The pneumoperitoneum was kept up. By September the right hemi-diaphragm was beginning to recover function.

29/11/48. The right phrenic nerve was re-crushed with resulting paralysis of the hemi-diaphragm.

21/1/49. X-Ray no longer shows an apical cavity; the right hemi-diaphragm has risen 6.5 cms.

Tomograms have been done to confirm the absence of cavitation.

This patient has remained well as an out-patient; she has no sputum and this has been confirmed by gastric lavage and guinea pig inoculation. Her weight has only risen 4 lbs. to 9 sts. 4 lbs. and the B.S.R. is 12.
D.D.: male, age 18; admitted 10/6/48
        discharged 30/11/48.

This man was seen in June 1948, when he com-
plained of tiredness and a recent haemoptysis. He
had some cough and sputum thereafter, and lost some
weight. Clinically, there were a few signs at the
left apex.

3/6/48. X-Ray (prior to admission). There
was light infiltration of the upper third of the left
lung with a cavity 3.5 cms. in diameter. There was
some light mottling of the left mid zone.

Sputum was T.B. Positive; B.S.R. 30 and weight
was 10 sts. 10 lbs.

15/6/48. Left artificial pneumothorax was
induced, but this was ineffective, as the lung was
adherent opposite the cavity. The pneumothorax was
forthwith abandoned.

6/7/48. The left phrenic nerve was crushed,
with paralysis of the left hemi-diaphragm.

13/7/48. A pneumoperitoneum was induced.
Thereafter, there was a good rise of the hemi-
diaphragm (11 cms.) to the third left interspace.

13/2/49. X-Ray shows that the cavity at the
left upper zone is no longer visible. Tomograms
confirm cavity closure.

This man's general condition is very good. He
has no sputum and B.S.R. is 2. His weight has gone
up to almost 13 sts. 7 lbs. Recent gastric lavage
and guinea pig inoculation produces a T.B. Positive
sputum; however, the pneumoperitoneum will be kept
going meanwhile to allow the disease to settle down,
if possible.
T.C.: male, age 37.

This man was discharged from the R.A.F. in August 1946. He had an apical infiltration on the right side with a small cavity.

March 1946. A right artificial pneumothorax was induced in an R.A.F. hospital, but this had to be abandoned in view of the lung being widely adherent over the apex. The cavity remained.

February 1947. A pneumoperitoneum was induced in another hospital.

August 1948. This patient attended Glenlomond Sanatorium for refills. Initial X-Rays showed a satisfactory rise of the right hemi-diaphragm (7 cms. rise). He said he was well with no symptoms; he had no cough or sputum.

26/3/48. Tomograms showed a large apical cavity on the right side.

This man was an out-patient and it has not been possible to do a gastric lavage and guinea pig inoculation. He has now gone back to his original hospital where a thoracoplasty has been done.
Mrs. S. McL: age 26; admitted 28/11/47

This patient was first examined after her baby
had died of tuberculous meningitis. She had com-
plained of a persistent cough after a "cold" in
June 1947, when the baby was then six months old.
After this she developed sputum and felt easily tired.

Clinically there was widespread disease on the
left side. The sputum was positive and the B.S.R.
was 20.

28/11/47. X-Ray showed widespread broncho-
pneumonic disease of almost the whole of the left
lung and right apex. A cavity was probably lying
at the extreme apex.

2/12/47. Left artificial pneumothorax was
induced.

After this a large cavity was seen to appear in
the collapsed left lung; this cavity was held out
by uncuttable adhesions. The pneumothorax was
abandoned after one month.

27/1/48. The left phrenic nerve was crushed
with paralysis of the left hemi-diaphragm.

30/1/48. A pneumoperitoneum was induced.

Despite a good rise of the diaphragm of 6 cms.
the large left apical cavity was entirely unaffected.
The pneumoperitoneum was therefore abandoned on

This left lung would require a thoracoplasty,
but in view of the disease on the right, and the
fact that the patient is mentally unsuited for major
surgery, she was discharged home with her condition
unchanged.
A. McE: male, age 33: admitted 1/2/46
discharged 29/10/46.

This man coughed up blood two months prior to
admission; this happened on one day only and was not
repeated. He had no cough or sputum although he
had lost some weight. The sputum by direct
examination was negative and the B.S.R. was 21.

7/2/46. X-Ray showed a chronic fibroid lesion
at the right apex with cavity formation; some
lighter shadowing on the left side.

26/2/46. Right artificial pneumothorax was
tried but as the lung was widely adherent at the
apex it was forthwith abandoned.

5/3/46. The right phrenic nerve was crushed
with resulting paralysis of the right hemi-diaphragm.
Three months later there was no great change in the
appearance of the lesion.

4/6/46. A pneumoperitoneum was induced.
The pneumoperitoneum has been kept up since and the
patient has been attending as an out-patient for
refills, weekly, since 29/10/46.

The disease appeared to have healed up con-
siderably. During the past two years the patient
has been working and he has had no symptoms at all:
no cough or sputum.

18/11/48. X-Ray showed dense fibrotic lesion
of the right apex but no cavity was seen. However;
on screening, there was the impression that a cavity
was being seen behind the clavicle.

10/2/49. Tomograph films were taken and these
showed a small round cavity persisting in the dense
disease.

As a result of a gastric lavage and guinea pig
inoculation, the sputum is now seen to be positive.
Fig. 1 18/12/47. Opacity of R. upper lobe.

Fig. 2 10/6/48. Diseased area unaltered in position with a small infra-clavicular cavity.

Fig. 3 17/2/49. R. upper lobe cavity and L. hydro-pneumothorax.
E.R.: female, age 20: admitted 30/12/47.

This girl was admitted with acute exudate disease of the right upper lobe.

18/12/47. X-Ray (Fig. 1) showed an opacity of most of the right upper lobe and a lateral film showed that this was due to collapse of the posterior apical segment of the upper lobe.

The B.S.R. was 25 and the Sputum positive. Her weight was 6 st. 4 lbs.

13/1/48. An artificial pneumothorax was induced in an attempt to collapse the right lung, despite the original X-Ray appearance. This had to be abandoned as the lung was widely adherent over the apex.

27/1/48. A pneumoperitoneum was induced prior to a right phrenic crush as there was a history of pleurisy some years previously and X-Ray showed some filling up of the costo-phrenic angle: this was especially noticeable on the X-Ray after the artificial pneumothorax was induced.

27/1/48. There was apparently a freely moving right hemidiaphragm so the right phrenic nerve was crushed with resulting paralysis of the hemidiaphragm.

The patient's general condition improved and the moist signs heard on admission disappeared by April 1948.

10/6/48. X-Ray (Fig. 2) showed that despite the good rise of the hemidiaphragm to the fourth costal cartilage (a rise of 6 cm.) the right upper lobe had not altered its position since the original film taken on admission; in addition, a small cavity had now developed below the clavicle. The pneumoperitoneum was abandoned as being quite ineffective and the patient was considered for thoracoplasty. This right upper lobe cavity progressively enlarged until it occupied most of the lobe, before thoracoplasty could be arranged. This disease now became obvious clinically.

At the beginning of December 1948 there were clinical signs of extension of the disease to the left side.

2/12/48. X-Ray confirmed a mid zone infiltration of the left lung. In view of the already paralysed right hemidiaphragm, which had not yet completely recovered function, an attempt to control the left lung disease by an artificial pneumothorax was made on 7/12/48.

However /


However, this was quickly succeeded by a large pleural effusion on the left side. During this time the patient was exceedingly dyspnoeic and ill. The effusion has been partly cleared up by frequent aspiration.

This girl has now a quite obvious tuberculous tracheo-bronchitis; she has a pronounced wheeze over the right upper lobe and frequent spasms of coughing; she has difficulty in bringing up thick tenacious sputum.

17/2/49. X-Ray (Fig. 3) shows the large right upper lobe cavity and the left hydro-pneumothorax.
Fig. 4 18/12/46. Moderately large cavity in L.upper zone.

Fig. 5 10/9/47. L. A.P. with unclosed mid-zone cavity.

Fig. 6 23/10/47. A.P. and P.P. giving cavity closure.
M.A.: female, age 28: admitted 17/12/46
         discharged 23/10/47.

This girl had been under observation since
14/9/44, when she had a haemoptysis with clinical
and X-ray evidence of disease of the upper half of
the left lung. She refused admission to the
Sanatorium. The patient remained well after that,
and after six months' bed rest at home, the original
cavity on the left side had disappeared. She was
periodically X-rayed, until there was seen to be
reactivation of the disease on the left side; she
was then admitted. The sputum was positive and the
B.S.R. was 10.

18/12/46. X-ray showed a moderately large
cavity in the left infra-clavicular area. (Fig. 4).

19/12/46. Left artificial pneumothorax was
induced. It was not really a technically satisfactory
pneumothorax despite two attempts to divide adhesions.

15/5/47. X-ray showed a persistent mid zone
cavity in the collapsed left lung. In an effort to
control this cavity, it was decided to crush the
left phrenic nerve.

27/5/47. The left phrenic nerve was crushed with
paralysis of the left hemidiaphragm. As a result of
the paralysis, the left hemidiaphragm has risen
4.5 cms.; despite this rise, a further X-ray on
10/9/47 (Fig. 5) showed that a cavity persisted in
the left mid zone.

23/9/47. Pneumoperitoneum induced, giving a
total rise of the left hemidiaphragm of 9 cms. The
artificial pneumothorax and pneumoperitoneum were
simultaneously used to obtain cavity closure, as seen
on X-ray 23/10/47 (Fig. 6). However, by 6/2/48, it
was obvious that there was considerable pleural
thickening and the pneumothorax was taking very much
smaller refills. Nevertheless, it was possible to
keep the pneumothorax with small refills.

30/9/48. There was a peritoneal effusion on
screening and this was present two months later, so
that the pneumoperitoneum was abandoned on 15/11/48.

This patient has remained sputum negative and
Tomograms confirm cavity closure.
Mrs. G., age 27: admitted 17/12/46  
discharged 23/10/47.

Clinically and by X-Ray, this patient had left upper zone infiltration with a large infra-clavicular cavity; there was also disease of the right mid zone. The sputum was positive and the B.S.R. was 40. Her weight was 6 st. 10 lbs.

19/12/46. A left sided artificial pneumothorax was induced, but there were adhesions which necessitated adhesion section twice.

20/1/47. Adhesions were divided.

13/2/47. The left phrenic nerve was crushed in an effort to help close the cavity in the left lung which remained held out by adhesions.

12/4/47. Second adhesion section; after this, several ounces of blood were aspirated from the pleural space. The artificial pneumothorax was kept up until a purulent effusion developed on 7/8/47; this was T.B. negative on culture. After this effusion the lung began to creep out and developed a thickened visceral necessitating abandonment of the left artificial pneumothorax on 20/10/47.

23/9/47. In view of the possibility of ultimately having to abandon the left artificial pneumothorax, a pneumoperitoneum was induced.

14/10/47. The left phrenic nerve was re-crushed. There was a satisfactory rise of the left hemidiaphragm of 7.5 cms.

5/11/48. X-Ray showed the left hemidiaphragm to be opposite the fourth rib, but it remained doubtful whether the disease in the left lung was really controlled.

Tomograms seemed to show a small cavity remaining in the left infra-clavicular area.

The right lung disease had improved considerably by the time the patient was discharged; however, moist sounds still remain on this side; it has nevertheless shown no sign of deterioration.

During this time the patient's general condition has remained very well. She has put on seven or eight pounds in weight and states that she has no sputum. A recent gastric lavage and guinea pig inoculation, however, was positive.
Mrs. M.A.: age 22; admitted 18/12/47.

In early 1947 this woman developed bronchitis which did not clear up properly; her cough and sputum persisted in spite of treatment. She lost some weight.

19/3/47. X-Ray showed a mid zone cavity on the right together with an apical cavity and a pleural effusion on the left. She was kept in bed at home for the next seven months until she was admitted to sanatorium.

On admission, there were signs in the upper half of the right lung and more extensively at the left base.

The sputum was positive and the B.S.R. was 23. The weight was 7 sts. 7 lbs.

18/12/47. X-Ray showed an improvement since the previous film; the right upper zone disease showed a more chronic character and the mid zone cavity had now retracted upwards to lie opposite the second costal cartilage. Despite marked clinical signs, there was only some light infiltration at the left base.

6/1/48. A pneumoperitoneum was induced.

14/1/48. The right phrenic nerve was crushed with paralysis of the right hemi-diaphragm.

Thereafter, the right hemi-diaphragm rose to the third costal cartilage (a rise of 10 cms.) and there was progressive improvement of the disease in the right upper zone; an improvement which has been maintained.

24/6/48. X-Ray showed more obvious infiltration at the left base. Clinical signs at the left base persisted during this time, and remained quite obvious despite an improvement in X-Ray appearances four months later.

Her general condition improved and she appeared very well. The sputum was negative and the B.S.R. 7. Her weight was 8 sts. 4 lbs.

17/11/48. Gastric lavage and guinea pig inoculation was negative. This was repeated but before the result could be obtained, fresh sputum was obtained.

24/2/49.
24/2/49. For fourteen days, this patient had been bringing up a lot more sputum which was now T.B. Positive: clinically, there was no change in the signs at the left base. X-Ray showed some honey-combing.

Tomograms showed dense disease with a cavity at the left base.
R.M.: male, age 44: admitted 16/2/48

This man was admitted to another hospital on
25/12/47 with "appendicitis"; he was eventually
operated on a week later and found to have enlarged
ilio-caecal glands. Two days after this he developed
"pneumonia". On recovering from this, his chest
was X-Rayed.

Patient had been feeling unwell for eight months;
he was easily tired and had a poor appetite; no
noticeable loss of weight. Whilst in hospital he
had a cough and sputum which was blood stained, in
which, however, no tubercle bacilli were found.

Clinically, there were no obvious signs of
disease in the lungs. His sputum was negative on
repeated examination; the B.S.R. was 6 and his weight
was 10 sts. 9 lbs.

19/2/48. X-Ray showed a small cavity above the
right clavicle.

9/3/48. The right phrenic nerve was crushed
with paralysis of the right hemi-diaphragm.

16/3/48. A pneumoperitoneum was induced.

17/9/48. X-Ray showed a good rise of the right
hemi-diaphragm (10.5 cms.) and the small cavity
already seen in earlier films was no longer present.
However, there was now a persistent effusion in the
abdomen. This was first seen on 23/7/48.

8/10/48. This effusion showed no sign of
disappearing so the pneumoperitoneum was abandoned.

After this, the patient complained of vague
abdominal pains but there was nothing to indicate
that the condition was an active tuberculous condition.
The right hemi-diaphragm has remained high opposite
the fourth costal cartilage.

2/2/49. Lipiodol X-Rays showed collapse of the
right lower lobe above the hemi-diaphragm and
crowding together of the bronchi especially in the
anterior basic segment.

This man’s general condition is good. His
weight is 12 sts. 12 lbs. (an increase of 31 lbs.),
the B.S.R. is 4 and the sputum, after gastric lavage
and guinea pig inoculation, remains negative.
Fig. 7 5/11/47. Fibro-caseous disease of R. upper zone. Cavity under the clavicle.

Fig. 8 17/9/48. R. upper zone disease unaffected by P.P.

Fig. 9 15/10/48. Tomogram shows large apical cavity.
J.W.: male, age 40: admitted 16/12/47

This man was seen as a contact of his wife who
had advanced bilateral pulmonary tuberculosis. He
denied having any symptoms. Clinically, however,
there were moist sounds at the right apex. The
sputum was T.B. Positive and the B.S.R. was 10.

5/11/47. X.-Ray (Fig. 7) (prior to admission)
showed fibro-caseous disease of the right upper lobe
with a thick walled cavity lying under the clavicle.

30/12/47. Pneumoperitoneum induced.

27/1/48. The right phrenic nerve was crushed
with paralysis of the right hemidiaphragm.

Thereafter there was an appreciable rise of the
hemidiaphragm (6 cms.) to the fourth space.

Clinically, there was some improvement of the
disease and the patient said he had no sputum. His
weight at 8 sts. 4 lbs. was much the same as that on
admission. Owing to certain domestic difficulties
this man was allowed to go home and attend as an
out-patient.

17/9/48. X-Ray (Fig. 8) showed that the
disease at the right apex had been entirely unaffected
by the pneumoperitoneum. After nine months' treat-
ment there was really little change in the X-Ray
appearances, although the cavity seen in the original
film was no longer seen.

15/10/48. Tomograms (Fig. 9) showed that
the large apical cavity has been unaffected by the
disease.

The pneumoperitoneum was abandoned forthwith.

This man had been feeling tired and lacking in energy for six months; he had lost nearly one stone in weight. He developed a cough and sputum.

Clinically, there was active disease in the upper half of the right lung.

The sputum was positive and the B.S.R. was 30. His weight was 8 sts. 5 lbs.

19/6/48. X-Ray showed a dense lesion in the upper zone of the right lung which was breaking down to form cavities.

29/6/48. A pneumoperitoneum was induced.

6/7/48. The right phrenic nerve was crushed with paralysis of the right hemi-diaphragm.

6/10/48. X-Ray. There was not a lot of change at the right upper zone despite a rise of 7 cms. of the right hemi-diaphragm.

Tomogram showed up several cavities 1 to 1.5 cms. each in diameter. These have been quite unaffected by the pneumoperitoneum.

19/10/48. A right sided artificial pneumothorax was induced, and the pneumoperitoneum abandoned.

The right lung was widely adherent over the apex and the pneumothorax was quite ineffective.

This man's general condition remains good. The sputum remains positive. The B.S.R. is 7 and weight is 9 sts. 1 lb.

Since admission, there has been a spread of disease to the left lower zone.
Fig. 10 22/10/48. Nodular infiltration with small cavities in R. upper zone.

Fig. 11 5/8/48. R. upper lobe atelectasis with P.P.

Fig. 12 11/10/48. Shows an interlobar effusion with high R. diaphragm.

Fig. 13 13/1/49. Clearing of the opacity over R. upper lobe. Fresh disease at R. base.

This boy had an "influenzal" attack prior to his first X-Ray and diagnosis of the condition: he had a cough and sputum and lost weight. Clinically, there was disease in the upper half of the right lung.

22/4/48. X-Ray showed nodular infiltration with multiple small cavities in the upper third of the right lung; there was fine mottling of the rest of the right lung and of the left lung. The sputum was T.B. positive and the B.S.R. was 35. (Fig. 10).


4/5/48. Pneumoperitoneum was induced in view of the apparently acute nature of the disease. Six weeks later an X-Ray showed a good rise of the hemidiaphragm to the fourth costal cartilage (a rise of 5.5 cms.).

5/8/48. X-Ray (Fig. 11) showed a uniform opacity over the right upper lobe; a lateral film showed this to be an atelectasis.

11/10/48. X-Ray (Fig. 12) showed that there was an inter-lobar effusion; repeated aspiration, however, did not produce any fluid. With the appearance of the effusion the pneumoperitoneum was abandoned. A Tomogram was done at this time but showed a uniform opacity only: no cavity could be seen. This condition remained stationary until the beginning of January 1949, when the patient started to cough up large quantities of pus which was T.B. Positive. Clinically there were numerous rhonchi over the right upper lobe.

13/1/49. X-Ray (Fig. 13) showed that the opacity over the right upper lobe had cleared considerably but there was now fresh disease in the right lower lobe. A cavity was now visible for the first time below the right clavicle.

In view of this fresh disease and cavity formation a further attempt was made to help the diseased right lung by inducing a pneumoperitoneum again on 18/1/49. However, this was almost immediately followed by an early atelectatic appearance of the whole lung (Fig. 14).

27/1/49. Pneumoperitoneum was forthwith abandoned.

9/2/49.
Fig. 14 18/1/49. Early atelectasis of R. lung.

Fig. 15 24/2/49. Considerable improvement is now seen at the R. upper zone.
9/2/49. Bronchoscoped by Dr. Semple, Hairmyres Hospital, because of the now definite evidence of bronchial obstruction. He reported that there is large granulomatous mass protruding into the orifice of the right main bronchus. There is little doubt that this is related to a hilar gland which has ulcerated through, and it seems that this granulation tissue was tuberculous; some of this tissue was removed for examination. The pathologist reported that this was tuberculous granulation tissue.

12/2/48. In view of the bronchoscopic examination streptomycin, on gramme daily, was started.

24/2/49. X-Ray (Fig.15 ) showed considerable improvement from the last film: there is still an atelectatic area lying behind the clavicle, but no other areas of collapse are seen; in addition, there has been some improvement of the recent infiltration in the right cardio-phrenic angle. The boy's general condition has improved too.

The B.S.R. is 11 and the sputum on direct examination has become negative.
A.S.: male, age 24.

This man was never admitted to this institution; he only attended here as an out-patient. Initial report is from another hospital.

This man took ill whilst serving in the R.A.F. in Egypt in June 1946; he had a cough and sputum. On return to this country in November 1946, an X-Ray showed up a lung lesion which was taken to be a lung abscess which did not respond to penicillin.

13/12/46. X-Ray showed two cavities in the apex of the right lower lobe. The sputum was then found to contain tubercle bacilli after bronchoscopy.

28/1/47. A pneumoperitoneum was induced.

1/4/47. The right phrenic nerve was crushed with paralysis of the right hemi-diaphragm.

5/9/47. X-Ray at this Sanatorium for the first time showed a satisfactory rise of the right hemi-diaphragm with an area of collapse at the apex of the right lower lobe.

15/10/48. This man's right hemi-diaphragm was seen to be functioning freely and as the diseased area looked quiescent, the pneumoperitoneum was abandoned.

24/2/49. X-Ray showed a small area of collapse remaining at the apex of the right lower lobe.

His general condition is good and he has no sputum. It has not been possible to do a more thorough search to confirm the absence of sputum on this man.
R.S.: male, age 25: admitted 20/5/47
discharged 8/8/47.

In January 1947 this man had an influenzal attack and three months later he began to perspire at nights. In November 1947 he had pain in the chest and in January 1948 he was admitted to hospital with a febrile illness. He was thereafter transferred to another hospital with a diagnosis of lung abscess; however, tubercle bacilli were found in the sputum.

4/2/47. X-ray showed a dense area of consolidation in the right lower lobe with some collapse. There was a cavity lying at the apex of the lower lobe. There was also consolidation of the middle lobe.

19/2/47. Lipiodol X-Rays show no bronchiectasis.

31/3/47. The consolidation of the right base is less marked. Collapse is still present, however, with raising of the right hemi-diaphragm and pulling over of the trachea. The cavity persists at the apex of the lower lobe.

8/4/47. The right phrenic nerve was crushed.

10/4/47. A pneumoperitoneum was induced.

After this the patient made good progress and his sputum was negative. On admission to this Sanatorium there were extensive clinical signs over the right lower lobe posteriorly. The sputum was consistently T.B. negative on direct examination and the B.S.R. was 45: this was 60 prior to transfer to Glenlomond.

25/5/47. X-ray showed a pneumoperitoneum with the right diaphragm opposite the fourth costal cartilage: there is dense collapse of the lower and middle lobes.

This man was then transferred to Switzerland from 8/8/47 to 6/9/48 and, whilst there, the pneumoperitoneum was abandoned in December 1947 as it was said to be ineffective. The patient was then bringing up blood-stained sputum every morning, which was T.B. Positive. It became negative, however, before returning to this country.


Extensive moist sounds were still heard posteriorly. The sputum on culture was negative and the B.S.R. was 2. The patient still had sputum with blood in it in the mornings: this was an almost daily occurrence.

13/9/48. /
Fig. 16 9/12/48. Bronchogram. Early bronchiectasis in cardio-phrenic angle.

Fig. 17 9/12/48. Lateral film shows high paralysed diaphragm and crowding of basal bronchi.
13/9/48. Tomograms showed honey-combing in the cardio-phrenic angle but no cavity was seen.

9/12/48. Lipiodol X-Rays (Fig.16,17) were done in view of the persistent signs. These show a distinct basal bronchiectasis, with crowding of the bronchi in all basic segments of the lower lobe. There is also a high right hemi-diaphragm opposite the fourth costal cartilage which only moves 3 cms. between full expiration and inspiration, as compared with the left hemi-diaphragm which moves 6 cms.

This man's general condition is very good: his weight has gone up two stones since that (10 sts. 8 lbs.) on original admission. The sputum remains T.B. negative and the B.S.R. on discharge was 2.
Fig. 18 8/5/47. L. basal infiltration and cavity.

Fig. 19 4/3/49. High diaphragm with L. basal collapse and small cavity at medial end.
Mrs. K.: age 42: admitted 6/5/47
discharged 17/1/48.

This woman had a sharp stabbing pain on the left side at the beginning of 1947: she had some cough but no sputum. She has also been easily tired and has lost two stones in weight. Clinically there was extensive disease in the left lower lobe posteriorly.

The sputum was Positive and the B.S.R. was 38. Her weight was 9 sts. 4 lbs.

8/5/47. X-Ray (Fig. 18) showed infiltration in the lower half of the left lung with a cavity (3 cms. in diameter) lying just outside the heart shadow.

20/5/47. The left phrenic nerve was crushed.

27/5/47. A pneumoperitoneum was induced.

There has been a good rise of the left hemidiaphragm to the fourth space but X-Rays have frequently shown that a small cavity persists above the left hemidiaphragm, which has risen 12 cms.

4/3/49. X-Ray (Fig. 19) shows the present position with a small cavity lying at the medial end of a collapsed area of lung on the left side.

This patient is generally well; her weight is now over 11 1/2 sts. She states that she has no sputum but it has not been possible to confirm this by gastric lavage or guinea pig inoculation.
T. A.: male, age 28: admitted 3/6/47
    discharged 24/10/47.

This man had a history of sweating at nights, loss of appetite, scanty morning cough and sputum. An X-Ray, on admission to another hospital, showed tuberculosis of the right lung.

On admission to Sanatorium, there was clinically evidence of a lesion at the base of the right lung. The sputum was T.B. Positive and the B.S.R. was 42.

5/6/47. X-Ray showed infiltration of the lower half of the right lung with an area of collapse towards the periphery in the middle basic segment. There were scattered calcified opacities throughout both lung fields.

17/6/47. The right phrenic nerve was crushed, but this did not result in paralysis of the right hemi-diaphragm.

25/6/47. A. pneumoperitoneum was induced.

30/9/47. The right phrenic nerve was re-crushed with paralysis of the right hemi-diaphragm. This re-crushing of the right phrenic nerve produced a marked rise of the right hemi-diaphragm of 6.5 cms. Pneumoperitoneum refills were kept up at weekly intervals; when an attempt was made to let this man go for fourteen days between refills at the beginning of 1948, when he was attending as an out-patient, an X-Ray showed a small cavity was developing in the collapsed part of the right lower lobe. This disappeared again when the patient was once again given weekly refills.

13/9/48. X-Ray showed a marked improvement and clearing in the diseased right lower lobe, but the collapsed area persists as a triangular shadow above the hemi-diaphragm. Tomograms of this area show that there is no cavitation.

This man's condition is excellent, and he has remained very well, doing a full day's work.

Gastric lavage and guinea pig inoculation is negative.

The patient had a febrile illness with pain in the chest five years ago. Ever since then she has never really felt well; she had not the same amount of energy. But six months prior to admission this tiredness was more marked and she had a cough and sputum. She also lost weight.

Clinically, there were extensive moist sounds over the left lower lobe posteriorly.

The sputum was T.B. Positive and the B.S.R. was 55. Her weight was 7 sts. 11 lbs.

23/4/48. X-Rays showed infiltration towards the left base but no cavity was seen.

29/4/48. Tomograms showed an irregular large cavity (3 cms. in diameter) hidden behind the heart.

4/5/48. The left phrenic nerve was crushed with paralysis of the left hemi-diaphragm.

19/5/48. A pneumoperitoneum was induced.

21/10/48. X-Ray showed that the left hemi-diaphragm had risen 8 cms. to lie opposite the fourth costal cartilage. However, this cavity, although much smaller, was not apparently closed. This was confirmed by Tomograms. The sputum remained T.B. Positive, so pneumoperitoneum was abandoned.

26/10/48. A left sided artificial pneumothorax was induced.

This gave a satisfactory collapse of the left lung with apparent control of the disease. During the past two months there has been a spread of the disease, to the right lung, necessitating further bed rest. The sputum, however, is T.B. Negative. The B.S.R. is 15 cms. and her weight is 8 sts. 12½ lbs.
Fig. 20 3/5/48. L. basal infiltration and cavity.

Fig. 21 29/10/48. A.P. and P.P. Adhesions holding out large cavity.

This patient was originally admitted to hospital with a diagnosis of "appendicitis". Prior to this, she had been feeling "off colour" for a few months. She had developed some cough and sputum, and lost a lot of weight.

Clinically, there were extensive moist sounds in the left lower lobe.

3/5/48. X-Ray (Fig. 20).

10/6/48. X-Ray showed an area of consolidation and infiltration at the left base. There was a cavity (3 cms. in diameter) lying in this dense shadow.

The sputum was positive; B.S.R. was 47 and weight was 8 sts. 12 lbs.

22/6/48. The left phrenic nerve was crushed with paralysis of the left hemi-diaphragm.

25/6/48. A pneumoperitoneum was induced.

21/10/48. X-Ray showed that the cavity had been appreciably unaffected by the rise of the left hemi-diaphragm to the fourth interspace (a rise of 8 cms.) The pneumoperitoneum, therefore, was abandoned.

26/10/48. A left artificial pneumothorax was induced. Despite a fairly good collapse, the cavity was still held out by adhesions. These could not be cut on thoracoplasty as they were in the nature of thick bands fanning out from the cavity.

29/10/48. X-Ray (Fig. 21) shows the pneumoperitoneum present with left artificial pneumothorax; the cavity is seen held out by adhesions opposite the fourth costal cartilage.

23/12/48. X-Ray shows there has been extension of the disease on the right lung, with formation of a small cavity in the mid-zone.

28/12/48. A right artificial pneumothorax was induced. The lung, however, was widely adherent and this pneumothorax was forthwith abandoned.

This girl's general condition has improved since she was admitted. The sputum remains T.B. Positive; the B.S.R. is 12 and weight is 9 sts. 10 lbs.
Mrs. A.M.: age 28; admitted 23/6/47

This woman had haemoptysis in December 1945, but she was regarded as having a peptic ulcer and treatment directed towards this non-existent ulcer. In February 1946 she had pleurisy which led to an X-Ray in a large hospital, with a diagnosis of "unresolved pneumonia". The patient did not feel well; she had a cough and sputum and was losing weight. Eventually, the sputum was examined and the true diagnosis established.

Clinically, on admission to hospital, there was obvious disease of the right lung. The sputum was positive and the B.S.R. 40.

26/6/47. X-Ray showed chronic fibro-caseous disease of the whole of the right lung; there is a large basal cavity (4 cms. in diameter) and small cavities in the right upper lobe. The left lung showed some mottling in the upper zone.

1/7/47. The right phrenic nerve was crushed with paralysis of the right hemi-diaphragm.

8/7/47. A pneumoperitoneum was induced in an attempt to close the basal cavity.

Despite a good rise of the right hemi-diaphragm to lie opposite the fourth costal cartilage (a rise of 6 cms.) the basal cavitation was not influenced in any way. At this time it was seen that there was an extension of the disease at the left apex to form a cavity.

11/5/48. X-Ray shows that the basal cavity has not been affected by the rise of the right hemi-diaphragm; in this film the basal cavity is seen lying partly above and partly below the hemi-diaphragm at the left apex.

This pneumoperitoneum was obviously quite ineffective but it was kept up as it eased this woman's cough and made her feel better. Eventually, however, with a further increase in the size of the left upper lobe cavity the pneumoperitoneum was abandoned on 10/9/48. The patient was then losing weight; the sputum which is T.B. Positive had increased in quantity. The patient was obviously going downhill.

At the end of 1947 this girl complained of pain on the left side of the chest. She had no cough or sputum. She developed these, however, before admission to hospital.

Clinically, there was extensive infiltration of most of the left lung. The sputum was positive. The B.S.R. was 40 and her weight was 8 sts. 5 lbs.

15/4/48. X-Ray showed a large apical cavity (4 cms. in diameter) with soft mottling throughout the lower half of the left lung.

8/6/48. The left phrenic nerve was crushed with paralysis of the left hemi-diaphragm.

11/6/48. A pneumoperitoneum was induced to help the left basal infiltration.

Clinically, after three months, this remained entirely unaffected.

22/9/48. X-Ray. The left apical cavity had become larger (5.5 cms. in diameter) with the formation of a second smaller cavity in the left mid zone, despite a good rise of the left hemi-diaphragm of 9 cms. The pneumoperitoneum was therefore abandoned.

7/1/49. This patient was transferred to a chest unit for a thoracoplasty on the left side; but this was not done for bronchoscopy, in view of the large tension cavity at the left apex, showed ulceration of the right main bronchus. She was then started on a course of streptomycin. Her general condition, at present, remains good and the sputum has been reduced in quantity, although it is still positive. The B.S.R. is 10 and her weight is 8 sts. 13 lbs.

6/3/44 - 1/3/45. In Sanatorium with a right sided pleural effusion. This girl was due to report back to the Sanatorium regularly for review but she was not seen again for eighteen months, when she returned for X-Ray and examination after coughing up some blood. Clinically, there was disease in the upper half of the right lung.

6/2/47. X-Ray, as an out-patient, showed infiltration in the right upper lobe with a large cavity lying between a thickened inter-lobar septum and the clavicle.

3/2/47. Re-admitted to the Sanatorium.

11/2/47. The right phrenic nerve was crushed with paralysis of the right hemi-diaphragm.

21/2/47. A pneumoperitoneum was induced.

13/11/47. Discharged from the Sanatorium. The right upper lobe cavity could not be seen by X-Ray. Despite a good rise of the hemi-diaphragm of 6.5 cms., the thickened inter-lobar septum had scarcely moved its position. At the same time, rales persisted over the diseased right upper lobe. Nevertheless, the pneumoperitoneum was kept up in an effort to control the disease.

1/7/48. Patient was seen at home with abdominal upset. This proved to be a tuberculous peritonitis.

19/2/48. Re-admitted to Sanatorium. Twenty-five ounces of straw coloured fluid was aspirated from the abdomen. No tubercle bacilli were isolated from one specimen.

The pneumoperitoneum was abandoned from the date of illness. Many moist sounds were still present in the right upper lobe but this girl constantly maintained she had no sputum. However, tubercle bacilli were isolated after gastric lavage. The B.S.R. was 43 and her weight 7 sts. 10 lbs.

13/9/48. Tomograms did not reveal any cavity in the diseased right lung.

24/9/48. This patient developed a large left sided pleural effusion which required frequent aspiration before it eventually settled down.

18/11/48. /
18/11/48. X-Ray showed a cavity had re-appeared above the thickened right inter-lobar septum.

This girl should have had a thoracoplasty but her parents were absolutely against this being done.
Fig. 22 17/8/48. Pneumonic consolidation of R. mid-zone.

Fig. 23 17/2/49. Some improvement is shown but a large cavity has appeared.
No. 100.


This man was admitted to a general hospital in July 1948 because of an unexplained pyrexia. X-Ray showed lobar consolidation of the upper half of the right lung, and tubercle bacilli were found in the sputum after repeated examination.

Clinically, there was extensive disease in the upper half of the right lung with suggestion of cavity formation. The sputum was T.B. Positive and the B.S.R. was 30.

X-Ray

18/11/48. There is a pneumonic consolidation of the right mid zone with partial lung collapse. On the lateral film the disease was seen to occupy the posterior axillary segment; and the apex of the lower lobe was also involved. There had been no change since the X-Ray was taken on 17/8/48. (Fig. 22)

29/11/48. The right phrenic nerve was crushed; at the actual time of the crushing, the patient experienced a sudden jerking sensation in his abdomen as if "some tension had been released".

14/12/48. A pneumoperitoneum was induced. Thereafter there was a satisfactory rise of the right hemi-diaphragm of 9 cms.; but the inter-lobar septum has not moved up any and it is obviously adherent to the chest wall.

17/2/49. X-Ray showed that the dense lung lesion had not changed: in addition, however, there was now a large cavity in this dense lesion. This was not present previously as Tomograms had been done. (Fig. 23). Pneumoperitoneum was abandoned.

26/2/49. During the past week this patient had been very uncomfortable and distressed. He was running a temperature in the evenings to 103°. Examination showed that he had developed a typical tuberculous peritonitis which required aspiration. On aspiration, a large mass is felt in the right iliac fossa: this may be a tuberculous gland mass.
Fig. 24 16/9/48. Light infiltration in upper half R. lung with a cavity.

Fig. 25 6/10/48. R. A.P. Large cavity in collapsed upper lobe.

Fig. 26 2/2/49. Diseased area collapsed against mediastinum. No cavity seen.
No. 91.


This patient had a history of pain in the right chest, with cough and sputum. Clinically there was active disease of the right upper zone.

The B.S.R. was 20 and the sputum was T.B. Positive. Weight was 9 sts. 1/2 lb.

16/9/48. X-Ray (Fig. 24) showed light infiltration in the upper half of the right lung, but there was dense disease with cavitation at the extreme apex.

28/9/48. A right sided artificial pneumothorax was induced.

6/10/48. X-Ray (Fig. 25) showed a large apical cavity held out by adhesions. In view of the dangerous nature of this pneumothorax it was forthwith abandoned.

19/10/48. A pneumoperitoneum was induced.
2/11/48. The right phrenic nerve was crushed.

Clinically there are still active signs of the disease at the right apex, but these are very much less than when she was admitted. The sputum remains positive.

2/2/49. X-Ray (Fig. 26) shows considerable improvement in the right lung with the diseased area swinging in towards the mediastinum. No cavity is seen and this has been confirmed by Tomograms. The right diaphragm has risen 10 cms.

This patient's condition is very much improved: her weight is now 10 sts. 11 lbs., and the B.S.R. is 5.
J. McL.: male; age 43; admitted 22/5/48
      discharged 19/1/49.

This man had several years' history of
indigestion. Some seven months prior to admission
to hospital he had a recurrence of indigestion. A
month or so later he noticed that he had an evening
pyrexia. He also had a cough. His chest was then
X-Rayed.

Clinically, there was chronic fibroid disease
on the right side. The sputum was positive and the
B.S.R. was 20. His weight was 8 sts. 9 lbs.

27/5/48. X-Ray showed dense fibroid disease
in the upper half of the right lung with multiple
small cavities; there is a very thick apical pleura.
On the left side there was light fibrotic disease
in the upper zone but no cavity was seen.

28/5/48. A Tomogram was done of the left side
and this showed honey-combing and dense upper lobe
disease: but no larger cavity was seen.

22/6/48. The left phrenic nerve was crushed.

4/6/48. A pneumoperitoneum was induced.

There was progressive improvement on the left
side.

17/1/49. X-Ray showed no change on the right
side but there has been considerable improvement on
the left side; this left hemi-diaphragm has risen
11 cms.

The absence of cavitation on the left side was
confirmed by Tomograms. This man's general condition
has improved considerably whilst in the Sanatorium:
he has put on 28 lbs. in weight and looks very much
better. Cough and sputum are much easier now: the
sputum remains T.B. Positive and the B.S.R. is now
6.

This patient's right side would need a thora-
coplasty but the patient, himself, prefers to postpone
this meanwhile.
R.F.: male, age 24: admitted 22/9/47.

This man had cough and sputum. He had also been feeling tired and had lost some weight.

Clinically, the signs were scanty. Sputum was T.B. Positive and the B.S.R. was 25.

26/9/47. X-Ray showed a large thick-walled upper lobe cavity on the right lung with some infiltration throughout most of this lung. The left lung showed some infiltration with a small cavity in the mid zone.

4/11/47. The left phrenic nerve was crushed.

7/11/47. A pneumoperitoneum was induced.

Eight months after the pneumoperitoneum was induced there was still no appreciable change in the left lung disease, despite diaphragmatic rise of 8 cms.

26/8/48. Tomograms showed up a cavity in the fibrotic lesion in the left mid zone. After the X-Ray the pneumoperitoneum was abandoned in favour of an artificial pneumothorax.

7/9/48. A left sided artificial pneumothorax was induced. The collapse of the left lung was limited by a stout adhesion opposite the site of the disease.

7/2/49. Adhesions divided.

It is not yet possible to state whether this pneumothorax is going to control the left lung disease.
No. 87.


This girl had a cough and sputum for ten months; she was also easily tired and had lost weight.

Clinically, there were few moist sounds to be heard but she appeared to have a left apical cavity. The sputum was T.B. Positive and the B.S.R. was 38. The weight was 9 sts. 2 lbs.

17/7/48. X-Ray showed a large cavity in the left upper zone with some peri-cavitary collapse; the mediastinum was pulled over to the left. There was recent infiltration in the right mid zone.

20/7/48. A pneumoperitoneum was induced.

9/8/48. The right phrenic nerve was crushed as there appeared to be a satisfactory rise of the right hemi-diaphragm, eventually of 8.5 cms.

25/11/48. X-Ray showed improvement on the right mid zone but there has been further deterioration on the left lung with another cavity appearing.

This girl lost a lot of weight (16 lbs.) and her general condition is not good. Clinically, there are obvious signs remaining on the right side in addition to those on the left.

The sputum remains T.B. Positive and the B.S.R. is 23.
Fig. 27 6/2/47. Large upper lobe cavity with infiltration in the upper half of R. lung. L. infiltration.

Fig. 28 4/3/49. P.P. with no evidence of lung disease.
J. S.: male, age 26: admitted 27/1/47

Patient had a history of pleurisy with effusion on the left side in 1943. He remained well after this until October 1946, when he developed a cough and sputum: the patient was sent to a large Out-Patient Department where the condition was diagnosed as an inter-lobar pleurisy on the right side. Cough and sputum, however, persisted and he began to lose weight.

On admission, there were extensive signs in the upper half of the right lung and he had a tuberculous involvement of the larynx.

The B.S.R. was 11 and the sputum T.B. Positive.

6/2/47. An X-Ray (Fig. 27) showed a large upper lobe cavity (5 cms. in diameter) with fibro-caseous disease; there was also light infiltration of the lower half of the left lung.

13/2/47. The left phrenic nerve was crushed with resulting paralysis of the left hemi-diaphragm.

7/3/47. A pneumoperitoneum was induced.

10/9/47. An X-Ray showed that the disease of the right lung had contracted considerably towards the mediastinum. Rt. diaphragm had risen 6 cms. Lt. 10 cms.

The present position is shown in Fig. 28(4/3/49).

There has been further collapse towards the mediastinum of the diseased area of the right upper lobe, and in addition the light motling of the left lung has cleared up completely. Confirmation of cavity closure was obtained by repeated Tomograms. Sputum is negative on culture. The patient is still attending as an Out-Patient for refills. The tuberculous larynx has healed.

This patient was attending as an out-patient for refills for a left sided artificial pneumothorax when it was seen she had a small fresh focus with cavitation at the right apex. In view of this, it was decided to abandon the left sided artificial pneumothorax.

4/1/47. The lung had re-expanded completely but there were persistent signs of active disease on the left side and she was recommended for a left sided thoracoplasty, as the small right focus had remained stationary.

25/2/47. Prior to admission to the thoracic unit, the patient was allowed home for a few days and, on her return to hospital, an X-Ray showed widespread acute infiltration of the upper third of the right lung. The sputum was T.B. Positive.

5/8/47. A right sided artificial pneumothorax was induced, but the lung was adherent over the apex and pneumothorax was abandoned.

26/8/47. A pneumoperitoneum was induced.

30/9/47. A right phrenic nerve was crushed with paralysis of the right hemi-diaphragm.

2/3/48. X-Ray showed a considerable improvement on the right side. A small apical cavity which had already been seen was no longer definitely visible and the recent infiltration had cleared up. The right lung had so far improved as to make thoracoplasty on the left side again a feasible proposition. This was being considered when the patient took suddenly ill at home in August, 1948, and died there on 3/12/48. The cause of death was never definitely ascertained although she was said to have had "fluid on the stomach".
Fig. 29 8/4/48. Infiltration and cavity R. upper lobe. L. mid-zone infiltration.

Fig. 30 12/11/48. R. upper lobe contracted behind clavicle with cavity. L. mid-zone infiltration remains.

Fig. 31 3/3/49. P.P. with R. disease collapsing against the mediastinum. L. A.P. held out by adhesions.

This girl was first seen at home on 8/4/48. She was toxic and ill, and there was, clinically, extensive bilateral disease. An X-Ray showed caseating disease of the right upper lobe with a cavity (3 cms. in diameter), and also infiltration of the left mid zone (Fig. 29). The B.S.R. was 50. The sputum was positive. Weight was 8 sts. 4 lbs.

As no beds were available to admit this girl to the Sanatorium, she was kept on strict bed rest at home. On this regime her general condition improved considerably. She was eventually admitted to the Sanatorium on 10/11/48 after seven months in bed at home. Clinically, however, there were still marked signs of active disease.

12/11/48. X-Ray (Fig. 30) showed improvement, with the diseased right upper lobe retracting behind the clavicle, but there is still a quite obvious cavity (now 2.5 cms. in diameter); on the left there has been some improvement in the extent of the mid zone infiltration.

16/11/48. An artificial pneumoperitoneum was induced.

23/11/48. The right phrenic nerve was crushed, giving paralysis of the right hemi-diaphragm. Following this diaphragmatic paralysis, there has been a marked rise of the right diaphragm to the second interspace (a rise of 8 cms.), and this diseased area has retracted behind the clavicle: the cavity in the right upper lobe has been considerably reduced in size, but, as yet, has not closed.

To control the disease on the left side, an artificial pneumothorax was induced on 18/1/49, and the adhesions were partly divided on 7/2/49; further adhesion section will be necessary.

3/3/49. X-Ray (Fig. 31) shows the present position.

Both the pneumoperitoneum and the artificial pneumothorax will be kept up meanwhile to try to obtain further improved collapse by those methods.

The patient's sputum remains positive. B.S.R. is 15 and weight is 8 sts.

This patient had pneumonia two years ago and had never been really well since; she had a cough since the pneumonia and sputum during the past six months. She has lost weight and has been easily tired.

Clinically, there was disease of most of the right lung and at the left upper zone.

The sputum was T.B. Positive and the B.S.R. was 40.

2/11/48. X-Ray showed a large basal cavity (3.5 cms. in diameter) on the right side in the posterior basic segment; there is also infiltration in the right upper zone with cavitation. On the left side there is mid zone infiltration with honey-combing and a small cavity.

9/11/48. The right phrenic nerve was crushed with paralysis of the right hemi-diaphragm.

12/11/48. A pneumoperitoneum was induced.

13/1/49. X-Ray showed the right hemi-diaphragm to have risen 8 cms. to lie opposite the third interspace; there is an area of collapse lying above the left hemi-diaphragm and this may contain the large basal cavity which is not definitely seen now.

18/1/49. A left sided artificial pneumothorax was induced to control the left lung disease.

This patient's condition has improved considerably, but it remains to be seen whether the pneumoperitoneum will be sufficient to control this basal cavity.

Her sputum remains positive and B.S.R. is 10.
Fig. 32 15/7/47. Acute broncho-pneumonic disease upper half R. lung with cavities. L. mid-zone infiltration.

Fig. 33 13/1/49. Thickened inter-lobar septum fixing lung with small infra-clavicular cavity. L. A.P.

Fig. 34 23/2/49. Tomogram shows large cavity.
J.T.: male, age 18: admitted 14/7/47.

This boy was admitted with a history of cough and sputum; he was easily tired and had lost weight. The sputum was T.B. Positive and the B.S.R. was 75. His weight was 7 sts. 10 lbs.

15/7/47. X-Ray (Fig. 32) showed acute broncho-pneumonic disease of the right upper lobe with cavitation above the thickened inter-lobar septum; there was also soft nodular infiltration of the mid zone of the right lung and light infiltration of the left mid zone.

22/7/47. Pneumoperitoneum was induced.

30/7/47. The right phrenic nerve was crushed.

Thereafter, there was a progressive rise of the right hemidiaphragm with a gradual improvement in the condition of the right lung. With a corresponding improvement in the disease of the right side, an artificial pneumothorax seemed indicated for the left side.

25/11/47. Left artificial pneumothorax was induced, and this produced a satisfactory pneumothorax.

The pneumoperitoneum and the pneumothorax have been kept up since both were initiated. This boy has now been in bed for fifteen months (ever since admission); his general condition has improved considerably; he has put on one stone in weight and the B.S.R. has fallen to 3. Nevertheless, the sputum remains T.B. positive.

13/1/49. X-Ray (Fig. 33) shows the right hemidiaphragm to be opposite the fourth interspace but the right upper lobe disease is seen above a thickened inter-lobar septum with a small cavity at the peripheral end of the septum; there is a left artificial pneumothorax apparently controlling the disease of the left side.

23/2/49. Tomograms (Fig. 34) demonstrated that the cavity is indeed much larger than would have appeared from the X-Ray; it is lying above an adherent septum.

This girl had a haemoptysis in 1947: this was said to be a nasal haemorrhage. After this she became listless and easily tired. She had a cough which was said to be due to smoking but no sputum: she did have another haemoptysis, however, four months prior to admission.

Clinically, there were scattered moist sounds over most of the left lung and in the right mid zone.

The sputum was T.B. Positive and the B.S.R. was 54. Weight was 9 sts. 5 lbs.

28/10/48. X-Ray showed infiltration with cavities (2.5 and 1.5 cms. respectively in diameter) in the upper half of the left lung. On the right there is a small mid zone cavity.

2/11/48. A pneumoperitoneum was induced.

9/11/48. The left phrenic nerve was crushed with paralysis of the left hemi-diaphragm.

15/12/48. X-Ray shows that the left hemi-diaphragm has risen 9 cms. to lie opposite the third costal cartilage; the left mid zone cavities appear to have closed.

This girl's condition is satisfactory; she states she has no sputum (gastric lavage has not yet been done) and, clinically, no moist sounds are heard.

B.S.R. is 28. Weight is 9 sts. 3 lbs.; this fall in weight has been due to too large refills for the pneumoperitoneum.
Fig. 35 8/5/47. Large cavity in R. upper lobe. L. mid-zone infiltration.

Fig. 36 4/2/47. Shows considerable improvement. R. disease persists against the mediastinum. L. hilar disease.
C. B.: female, age 15: admitted 6/5/47
discharged 18/12/48.

History of being off colour for a year: then she became listless and began to lose weight. During the few weeks prior to admission she developed a cough and sputum which was T.B. Positive.

Clinically there was pronounced disease of the upper half of the right lung and less marked disease of the left upper zone. The B.S.R. was 50. Weight was 5 sts. 6 lbs.

8/5/47. X-Ray on admission (Fig. 35) showed dense disease of the right lung with a large cavity (3 cms. in diameter) above a well defined inter-lobar septum; there was also heavy shadowing at the left root with infiltration spreading out into the mid zone of the left lung.

24/6/47. Rt. phrenic nerve was crushed, resulting in paralysis of the right hemi-diaphragm.

8/7/47. A pneumoperitoneum was induced: in three months the right hemi-diaphragm was now beginning to function normally again; at the same time, there had been considerable improvement on the right side clinically but there were still many moist sounds persisting on the left side.

23/9/47. The left hemi-diaphragm was paralysed by a phrenic crush; it quickly recovered function again and was re-crushed on 9/3/48.

This girl's general condition improved considerably; clinically she appeared to have cavity closure on the right side, but there were persistent signs on the left side. Gastric lavage and a guinea pig inoculation were done in the continued absence of sputum: this proved T.B. Positive.

4/2/47. An X-Ray (Fig. 36) does not suggest any remaining cavities but Tomograms show small honeycombed areas in both diseased areas of the lungs. The pneumoperitoneum will be kept up meanwhile but obviously some other collapse measure is indicated. The B.S.R. was 3 and her weight had gone up to 7 sts. 12 lbs. on discharge.

In May 1948 the patient developed a cold with a severe cough but she had no sputum. She had pain in the left side of the chest.

Clinically, there was active disease of most of the left lung and at the right mid zone.

The sputum was T.B. Positive and the B.S.R. was 55. Her weight was 8 sts. 2 lbs.

29/7/48. X-Ray showed active disease in the upper half of the left lung with a cavity (3 cms. in diameter under the second rib); there was also recent broncho-pneumonia patches in the upper half of the right lung.

3/8/48. A pneumoperitoneum was induced, as the disease was too active for a pneumothorax.

9/8/48. The left phrenic nerve was crushed and there was paralysis of the left hemi-diaphragm.

There was a good rise of the left hemi-diaphragm (of 9 cms.) and the disease contracted upwards behind the left clavicle. Clinical examination showed that extensive moist sounds persisted on the left.

The sputum was T.B. Positive.

16/12/48. X-Ray showed that the fresh focus in the mid zone of the left lung which had been already seen in an earlier film had now broken down to form a small cavity.

Tomograms showed that this cavity measured 2 cms. in diameter.

25/1/49. A left sided artificial pneumothorax was induced and the pneumoperitoneum was abandoned; the left lung, however, was held out by apical adhesions and the mid zone cavity was not closed.

7/2/49. The adhesions were partly divided.

24/2/49. X-Ray showed little change in the collapse and the cavity is still quite obvious in the mid zone of the left lung.

The right lung during this time has partly broken down to form a small cavity.

The patient's general condition is not good. The sputum is positive. The B.S.R. is 30 and her weight has dropped 12 lbs. to 7 sts. 4 lbs.

This man was admitted with infiltration of the right lung. Clinically, there were many rales over most of the right lung with cavity signs at the apex. The sputum was positive and the B.S.R. was 58. His weight was 9 sts. 8 lbs.

16/7/48. X-Ray showed dense infiltration of the upper half of the right lung with a large cavity (4 cms. in diameter) in the infra-clavicular area. There was also scattered infiltration of the left lung.

20/7/48. A right sided artificial pneumothorax was induced but the lung was widely adherent over the apex and the pneumothorax was promptly abandoned.

3/8/48. A pneumoperitoneum was induced.

10/8/48. The right phrenic nerve was crushed with resulting paralysis of the right hemi-diaphragm.

16/12/48. X-Ray shows that the right hemi-diaphragm has risen to lie opposite the third costal cartilage (a rise of 11 cms.) and the infra-clavicular cavity can no longer be seen. In addition an extension of the disease on the left side which had taken place since his admission has also tended to settle, although the left hemi-diaphragm has only risen 2 cms.

24/2/49. Tomograms show that the cavity is not clearly visible; there is, however, a good deal of honey-combing in the dense disease above the thickened inter-lobar fissure.

This man's sputum remains positive. His general condition has improved markedly and he looks very much better. His weight has gone up 20 lbs. His B.S.R. at present is 5.
Fig. 37 21/4/48. Infiltration of most of L lung with hilar cavity. Infiltration of R lung.

Fig. 38 3/11/48. P.P. but disease is unaffected.

Fig. 39 9/12/48. Lateral film demonstrates cavity in apex of lower lobe.

Fig. 40 9/12/48. Tomogram demonstrates large posterior cavity.

At the beginning of 1948 this girl complained of tiredness and, some short time later, she had a small haemoptysis, which led to an X-Ray and the diagnosis of the condition.

Clinically, there were extensive signs on the left lung and in the right mid zone.

Sputum was T.B. Positive; B.S.R. was 32 and weight was 8 st. 3 lbs.

21/4/48. X-Ray (Fig.37). Light scattered infiltration of the greater part of the left lung with small cavities seen in the hilar region. There was light mottling of most of the right lung.

25/5/48. The left phrenic nerve was crushed with paralysis of the left hemi-diaphragm.

1/6/48. A pneumoperitoneum was induced.

Clinically, the left lung disease was not materially affected by the pneumoperitoneum, despite a good rise of 6 cms.

3/11/48. X-Ray (Fig.38) showed some clearing of the shadows of both lungs, but, in the main, the disease had not really been affected.

9/12/48. Tomograms were taken and these show a large cavity lying posteriorly (Fig. 40).

Lateral film (Fig.39) shows cavity to be lying at the apex of the left lower lobe and to be quite unaffected by the rise of the left hemi-diaphragm.

The pneumoperitoneum, therefore, was abandoned.

14/12/48. A left pneumothorax was induced. This, however, had no effect on the large posterior cavity which appeared to be adherent to the chest wall. The pneumothorax was forthwith abandoned.

This girl's general condition has probably improved a little since admission, but the same extensive clinical signs are still present in both lungs.

Sputum remains T.B. Positive; B.S.R. is 61 and weight is 7 st. 9 lbs.

This patient had cough and sputum and had been easily tired for nine months; eventually she became too tired for her work. She lost weight.

Clinically, there was extensive disease on the right lung.

The sputum was almost a pure culture of tubercle bacilli. The B.S.R. was 80 and her weight was 8 sts. 4 lbs.

30/3/48. X-Ray showed chronic fibro-caseous disease of the right lung, with a large cavity (4 cms. in diameter) lying above the thickened interlobar septum. Another large cavity was seen below the thickened septum and occupying the apex of the right lower lobe. The left lung showed a healed primary focus.

6/4/48. The right phrenic nerve was crushed with resulting paralysis of the right hemi-diaphragm.

13/4/48. A pneumoperitoneum was induced.

Two months later it was quite obvious that the pneumoperitoneum was quite ineffective and was stopped. Two weeks later, however, the patient requested that the pneumoperitoneum should be started again as she had more cough and sputum, and this was accordingly done.

18/11/48. X-Ray. The right hemi-diaphragm has risen 5.5 cms. to lie opposite the fourth costal cartilage; the right upper lobe has contracted a little but it is now mostly excavated and, in addition, there are further cavities developing in the right mid zone. The left base now shows widespread light mottling.

Despite the X-Ray appearances, her general condition remains fairly good.

The sputum is positive. The B.S.R. is 34 and the weight remains steady at 8 sts. 4 lbs.
CLASSIFICATION OF PATIENTS ON EXAMINATION

(National Tuberculosis Association of America)

1. MINIMAL LESION:
   (a) Slight infiltration without demonstrable cavitation.
   
   (b) A small part of one or both lungs - total volume of involvement, regardless of distribution, shall not exceed the equivalent of lung tissue which lies above the second chondro-sternal junction and the spine of the fourth or the body of the fifth thoracic vertebra on one side.

SYMPTOMS:

1. Slight or none.

   Slight, or no constitutional symptoms, including particularly gastric or intestinal disturbance, or rapid loss of weight; slight or no elevation of temperature or acceleration of pulse at any time during the twenty-four hours. Expectoration usually small in amount or absent. Tubercle bacilli may be present or absent.

2. Moderate.

   No marked impairment of function, either local or constitutional.

3. Severe.

   Marked impairment of function, local or constitutional.

11. MODERATELY ADVANCED LESION:

   Lesion allowable under Moderately Advanced - One or both lungs may be involved but the total involvement shall not exceed the following limits:
   
   (a) Slight disseminated infiltration or fibrosis which may extend through not more than the equivalent of the volume of one lung.
   
   (b) Severe infiltration, with or without fibrosis, which may extend through not more than the equivalent of the volume of one-third of one lung.
(c) Any gradation within the above limits.
(d) Total diameter of cavities, if present, should not exceed 4 cms.

**SYMPTOMS**:

1. **Slight or none.**
   Slight or no constitutional symptoms, including particularly gastric or intestinal disturbance or rapid loss of weight; slight or no elevation of temperature or acceleration of pulse at any time during the twenty-four hours. Expectoration usually small in amount or absent. Tubercle bacilli may be present or absent.

2. **Moderate.**
   No marked impairment of function, either local or constitutional.

3. **Severe.**
   Marked impairment of function, local or constitutional.

**III. FAR-ADVANCED LESION:**

A lesion more extensive than under Moderately Advanced, or definite evidence of greater cavity formation.

**SYMPTOMS**:

1. **Slight or none.**
   Slight or no constitutional symptoms, including particularly gastric or intestinal disturbance or rapid loss of weight; slight or no elevation of temperature or acceleration of pulse at any time during the twenty-four hours. Expectoration small in amount or absent. Tubercle bacilli may be present or absent.

2. **Moderate.**
   No marked impairment of function, either local or constitutional.

3. **Severe.**
   Marked impairment of function, local or constitutional.
DETAILS OF 100 PATIENTS
SUBMITTED TO
PNEUMOPERITONEUM
<table>
<thead>
<tr>
<th>CASE</th>
<th>SEX</th>
<th>AGE</th>
<th>EXTENT OF LUNG INVOLVEMENT</th>
<th>CLASSIFICATION</th>
<th>PHLEMO PERITONEUM</th>
<th>DIAPHRAGM PARALYSIS</th>
<th>WORKING RISE</th>
<th>INDICATIONS</th>
<th>COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. K.C. M.</td>
<td>21</td>
<td>Upper zone L. lung with small cavity. Recent soft apical infiltration on R. lung.</td>
<td>M.A.</td>
<td>9/1/45</td>
<td>19/7/46</td>
<td>18 mths.</td>
<td>2/7/44</td>
<td>L. Peral 8 cms.</td>
<td>To control the lesion evulsion in another hospital</td>
</tr>
<tr>
<td>CASE</td>
<td>SEX</td>
<td>AGE</td>
<td>EXTENT OF LUNG INVOLVEMENT</td>
<td>CLASSIFICATION</td>
<td>INDUCED TERMINATED</td>
<td>DURATION</td>
<td>COMMENTS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>------</td>
<td>-----</td>
<td>-----</td>
<td>-----------------------------</td>
<td>----------------</td>
<td>-------------------</td>
<td>----------</td>
<td>----------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>12.</td>
<td>A.S.</td>
<td>M</td>
<td>24</td>
<td>Cavitation R. mid-zone.</td>
<td>M.A. 28/1/47</td>
<td>15/10/48</td>
<td>21 mths.</td>
<td>This man was first seen on 5/3/47. His original films are not available for comparison.</td>
<td></td>
</tr>
<tr>
<td>13.</td>
<td>B.S.</td>
<td>F</td>
<td>18</td>
<td>Upper half of R. lung with cavity.</td>
<td>M.A. 21/2/47</td>
<td>1/7/48</td>
<td>17 mths.</td>
<td>This girl developed a parietal and L. pleural effusion between July and September, 1946.</td>
<td></td>
</tr>
<tr>
<td>15.</td>
<td>J.D.</td>
<td>M</td>
<td>33</td>
<td>Infiltration L. upper-zone.</td>
<td>Min. 25/2/47</td>
<td>21/2/47</td>
<td>1 mth.</td>
<td>This man was first seen as an O.P. on 5/3/47. His original plates are not available now.</td>
<td></td>
</tr>
<tr>
<td>16.</td>
<td>T.C.</td>
<td>M</td>
<td>37</td>
<td>R. apical infil- tration with cavities in upper zone.</td>
<td>M.A. Feb. 1947</td>
<td>26/3/48</td>
<td>13 mths.</td>
<td>This man was attended as an out-patient until re-admitted on 1/3/48 with extension of disease on R.</td>
<td></td>
</tr>
<tr>
<td>18.</td>
<td>J.S.</td>
<td>M</td>
<td>26</td>
<td>R. upper lobe cavi- tation and recent infiltration of left middle &amp; lower zones.</td>
<td>F.A. 7/3/47</td>
<td>continues</td>
<td>2 yrs.</td>
<td>This patient is now going downhill at home.</td>
<td></td>
</tr>
<tr>
<td>19.</td>
<td>Mrs.</td>
<td>F</td>
<td>27</td>
<td>Extensive bilateral disease with cavi- tation.</td>
<td>F.A. 7/3/47</td>
<td>28/6/48</td>
<td>25 mths.</td>
<td>Diaphragm remains high and is only partly working. The initial attempt to crush the R. phrenic nerve failed.</td>
<td></td>
</tr>
<tr>
<td>CASE</td>
<td>SEX</td>
<td>AGE</td>
<td>EXTENT OF LUNG INVOLVEMENT</td>
<td>PATHOLOGICUM</td>
<td>DIAGNOSIS</td>
<td>INDICTIONS</td>
<td>COMMENT</td>
<td></td>
<td></td>
</tr>
<tr>
<td>------</td>
<td>-----</td>
<td>-----</td>
<td>---------------------------</td>
<td>--------------</td>
<td>----------</td>
<td>------------</td>
<td>---------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>25.</td>
<td>H.M.</td>
<td>34</td>
<td>Left pleura</td>
<td>M.A.</td>
<td>17/6/47</td>
<td>10/12/46</td>
<td>Fluid in L. A.P. for six months with collapsed lung. Lung has not yet completely re-expanded.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>26.</td>
<td>T.A.</td>
<td>28</td>
<td>Infiltration R. lower zone with segmental collapse.</td>
<td>M.A.</td>
<td>25/6/47</td>
<td>30/9/47</td>
<td>Control R. basal disease. The first L. crush because L. A.P. was ineffective for haemoptysis. The second crush to give adequate rise with F.P.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>27.</td>
<td>R.A.</td>
<td>20</td>
<td>Infiltration of L. upper zone with cavity.</td>
<td>M.A.</td>
<td>1/7/47</td>
<td>26/5/47</td>
<td>To aid absorption of fluid and obliterate R. A.P. space. Fluid for three months before P.P. was induced.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>30.</td>
<td>Mrs.</td>
<td>F.</td>
<td>R. lung with upper and lower zone cavities.</td>
<td>F.A.</td>
<td>8/7/47</td>
<td>1/7/47</td>
<td>To aid absorption of fluid in and re-expansion of the L. A.P. Fluid present for three months before P.P.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>31.</td>
<td>C.B.</td>
<td>15</td>
<td>Upper zone on R. with large cavity; L. mid-zone infiltration.</td>
<td>F.A.</td>
<td>8/7/47</td>
<td>24/6/47</td>
<td>Control R. upper lobe cavity. This man has moved elsewhere.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>32.</td>
<td>H.R.</td>
<td>21</td>
<td>R. A.P. and recent acute M.A. spread into L. upper zone with cavity.</td>
<td>M.A.</td>
<td>22/7/47</td>
<td>15/7/47</td>
<td>Control R. upper zone lesion. There is concurrent L. A.P.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>33.</td>
<td>J.P.</td>
<td>46</td>
<td>Infiltration R. upper zone.</td>
<td>Min.</td>
<td>22/7/47</td>
<td>30/7/47</td>
<td>For acute R. lung disease. There is concurrent L. A.P.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>34.</td>
<td>J.T.</td>
<td>19</td>
<td>Acute infiltration upper half R. lung and L. mid-zone.</td>
<td>F.A.</td>
<td>22/7/47</td>
<td>30/7/47</td>
<td>For acute R. lung disease. There is concurrent L. A.P.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CASE</td>
<td>SEX</td>
<td>AGE</td>
<td>EXTENT OF LUNG INVOLVEMENT</td>
<td>CLASSIFICATION</td>
<td>INDUCED</td>
<td>TERMINATED</td>
<td>DURATION</td>
<td>WORK-LIFE DURATION</td>
<td>SIDE ING</td>
</tr>
<tr>
<td>------</td>
<td>-----</td>
<td>-----</td>
<td>-----------------------------</td>
<td>----------------</td>
<td>---------</td>
<td>------------</td>
<td>----------</td>
<td>-------------------</td>
<td>--------</td>
</tr>
<tr>
<td>35. C.N.</td>
<td>M. 35</td>
<td>Upper half R. lung with cavity</td>
<td>F.A.</td>
<td>29/7/47</td>
<td>Cont.</td>
<td>18 mths.</td>
<td>22/7/47</td>
<td>R. Partly 10 cms.</td>
<td>To control R. zone</td>
</tr>
<tr>
<td>36. F.L.</td>
<td>M. 32</td>
<td>Bilateral disease both lower lobes involved. Cavitation on left.</td>
<td>F.A.</td>
<td>29/7/47</td>
<td>Cont.</td>
<td>18 mths.</td>
<td>1/22/7/47</td>
<td>L. Yes</td>
<td></td>
</tr>
<tr>
<td>43. Mrs.</td>
<td>G. 27</td>
<td>Infiltration with cavity in L. upper zone; R. mid-zone infiltration.</td>
<td>23/9/47</td>
<td>Cont.</td>
<td>16 mths.</td>
<td>1/13/2/47</td>
<td>L. Yes</td>
<td>7.5 cms.</td>
<td>Control the left disease after left A.P. induced on 13/12/46 had developed thickened pleura.</td>
</tr>
<tr>
<td>44. A.G.</td>
<td>M. 27</td>
<td>Scattered motting with small apical cavity; two large cavities in L. upper zone.</td>
<td>F.A.</td>
<td>30/9/47</td>
<td>13/11/47</td>
<td>6 weeks</td>
<td>24/9/47</td>
<td>R. Partly None Fixed To help re-expansion of R. lung which had developed an empyema after resection on 8/6/47.</td>
<td></td>
</tr>
</tbody>
</table>

**INDICATIONS**
- To control R. zone
- To control disease in L. lung
- Control R. upper lobe disease
- Control extension of disease on R. prior to thoracoplasty on L.
- Close cavity on R. side
- Close cavity in mid-zone of lung in conjunction with L. A.P.
- Control the left disease after left A.P.
- To help re-expansion of R. lung which had developed an empyema after resection on 8/6/47.
<table>
<thead>
<tr>
<th>CASE</th>
<th>SEX</th>
<th>AGE</th>
<th>EXTENT of LUNG INVOLVEMENT</th>
<th>CLASSIFICATION</th>
<th>INDUCED</th>
<th>TERMINATED</th>
<th>DURATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>45. A.H.</td>
<td>M.</td>
<td>23</td>
<td>Light mottling of each lung with some honey-combing at the left hilum.</td>
<td>M.A.</td>
<td>3/10/47</td>
<td>21/1/49</td>
<td>15 mths.</td>
</tr>
</tbody>
</table>
55. J.M. N. 24 Fibro-caseous disease of most of the L. lung; light infiltration R. mid-zone.

56. A.C. F. 21 Large apical cavity and light basal infiltration on L. Right mid-zone infiltration.

57. T.K. F. 24 Large cavity on L. upper lung; and R. mid-zone infiltration with small cavity.


60. J.P. M. 28 Active fibro-caseous disease of all L. lung; tuberculous empyema on R.


64. M. F. 23 Large cavity at R. apex with infiltration scattered throughout L. lung.

<table>
<thead>
<tr>
<th>CASE</th>
<th>SEX</th>
<th>AGE</th>
<th>EXTENT OF LUNG INVOLVEMENT</th>
<th>CLASSIFICATION</th>
<th>INDUCED</th>
<th>TERMINATED</th>
<th>DURATION</th>
<th>DATE</th>
<th>SIDE</th>
<th>WORKING</th>
<th>RISE</th>
<th>INDICATIONS</th>
<th>COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>pneumonic infiltration</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>with cavities in upper</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>half of the L. lung.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>pneumothorax.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>R. lung.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>with apical cavitation.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>disease in the upper</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>half of the R. lung.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>throughout the L. lung</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>with upper-zone cavities.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Nodular infiltration in</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>upper half of R. lung.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>73.</td>
<td>J.K.</td>
<td>F.</td>
<td>Chronic fibroid lesion</td>
<td>Min.</td>
<td>27/4/48</td>
<td>10/12/48</td>
<td>8 mths.</td>
<td>20/4/48</td>
<td>L.</td>
<td>Yes</td>
<td>Poor</td>
<td>Continue improvement of L. apical disease which was obvious clinically.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>at the L. apex.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>cavities in upper third</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>of R. lung.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>with cavitation.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Case</td>
<td>Sex</td>
<td>Age</td>
<td>Extent of Lung Involvement</td>
<td>Classification</td>
<td>Terminated</td>
<td>Duration</td>
<td>Date</td>
<td>Side</td>
<td>Working Rise</td>
<td>Indications</td>
<td>Comments</td>
<td></td>
<td></td>
</tr>
<tr>
<td>------</td>
<td>-----</td>
<td>-----</td>
<td>---------------------------</td>
<td>----------------</td>
<td>------------</td>
<td>----------</td>
<td>-------</td>
<td>------</td>
<td>--------------</td>
<td>-------------</td>
<td>----------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>77.</td>
<td>Mrs.</td>
<td>F</td>
<td>24</td>
<td>Bilateral recent exudative disease with cavitation in</td>
<td>F.A.</td>
<td>19/5/48</td>
<td>28/10/48</td>
<td>5 mths.</td>
<td>-</td>
<td>Slight</td>
<td>To help the L. mid-zone disease which had shown some tendency to regress.</td>
<td>This patient went down steadily after the onset of enteritis and died on 22/12/48.</td>
<td></td>
</tr>
<tr>
<td>78.</td>
<td>B.C.</td>
<td>F</td>
<td>22</td>
<td>Light infiltration of most of the left lung with a hilar cavity.</td>
<td>F.A.</td>
<td>1/6/48</td>
<td>9/12/48</td>
<td>6 mths.</td>
<td>25/5/48 L. Partly 6 cm.</td>
<td>Close L. mid-zone cavity and improve infiltration.</td>
<td>This man is not anxious to have a thoracoplasty done. Attends another clinic as an out-patient.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>82.</td>
<td>A.P.</td>
<td>F</td>
<td>23</td>
<td>Pneumonic patch in left L. lower zone with a central cavity.</td>
<td>M.A.</td>
<td>25/6/48</td>
<td>21/10/48</td>
<td>4 mths.</td>
<td>22/6/48 L. Yes 8 cm.</td>
<td>To close L. lower-zone cavity.</td>
<td>There has been spread to the R. mid-zone.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>83.</td>
<td>H.L.</td>
<td>M</td>
<td>26</td>
<td>Dense lesion in upper zone R. lung with small cavities.</td>
<td>M.A.</td>
<td>29/6/48</td>
<td>19/10/48</td>
<td>4 mths.</td>
<td>6/7/48 R. Yes 7 cm.</td>
<td>To control R. upper-zone disease.</td>
<td>There has been spread to the L. lower-zone.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>84.</td>
<td>R.F.</td>
<td>F</td>
<td>19</td>
<td>Infiltration with cavity in R. upper lobe.</td>
<td>M.A.</td>
<td>29/6/48</td>
<td>16/9/48</td>
<td>3 mths.</td>
<td>22/6/48 R. Partly Poor</td>
<td>To relax lung in R. A.P. as it was held out by apical adhesions.</td>
<td>Adhesions could not be cut after thoracoplasty.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>86.</td>
<td>H.S.</td>
<td>M</td>
<td>22</td>
<td>Chronic bilateral disease with recent spread to the left base.</td>
<td>F.A.</td>
<td>13/7/48</td>
<td>11/11/48</td>
<td>4 mths.</td>
<td>6/7/48 L. Partly Poor</td>
<td>To control left basal disease.</td>
<td>This man has been in sanatorium already with advanced lesion of both lungs.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CASE</td>
<td>SEX</td>
<td>AGE</td>
<td>EXTENT OF LUNG INVOLVEMENT</td>
<td>CLASSIFICATION</td>
<td>INDUCED</td>
<td>TERMINATED</td>
<td>DURATION</td>
<td>DIAGNOSIS</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>------</td>
<td>-----</td>
<td>-----</td>
<td>---------------------------</td>
<td>----------------</td>
<td>---------</td>
<td>------------</td>
<td>---------</td>
<td>-----------</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>92</td>
<td>D.G.</td>
<td>46</td>
<td>Infiltration in most of L. upper and mid-zones with a cavity at the L. hilum. R. mid-zone infiltration.</td>
<td>F.A.</td>
<td>19/10/48 1/3/49 4 mths.</td>
<td>11/11/48 R. No 6 cms. To relax lung of L. A.F. strung up by apical adhesion.</td>
<td>These adhesions were not considered suitable for section as they were close to the mediastinum.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>94</td>
<td>Mrs.</td>
<td>36</td>
<td>Large basal cavity and smaller apical cavity on R. Left mid-zone infiltration with small cavity.</td>
<td>F.A.</td>
<td>12/11/48 Continues</td>
<td>3 mths.</td>
<td>9/11/48 R. No 8 cms. Close R. basal cavity.</td>
<td>This woman had a L. A.F. induced on 18/1/49.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CASE</td>
<td>SEX</td>
<td>AGE</td>
<td>EXTENT OF LUNG INVOLVEMENT</td>
<td>CLASSIFICATION</td>
<td>INDUCED</td>
<td>TERMINATED</td>
<td>DURATION</td>
<td>DATE</td>
<td>SIDE</td>
<td>WORKING RISE</td>
<td>INDICATIONS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>------</td>
<td>-----</td>
<td>-----</td>
<td>-----------------------------</td>
<td>----------------</td>
<td>---------</td>
<td>------------</td>
<td>---------</td>
<td>------</td>
<td>------</td>
<td>--------------</td>
<td>-------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Infiltration of both upper zones with small cavities on L.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>It is considered that the L. A.P. will be necessary.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Bilateral exudative disease in upper halves of each lung; L. upper-zone cavity.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Mid-zone infiltration with cavitation.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>