THE PROBLEM of TUBERCULOUS EMPYEMA in SANATORIUM PRACTICE.

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VOLUME I.

VOLUME II:

Introduction.

It might be asked how tuberculous empyema can claim a place as a subject for a surgical thesis.

Tuberculosis, more than any other disease in these islands to-day, is the meeting ground of surgeon, physician, pathologist, radiologist, medical officer of health, and social worker.

The best chest surgeons are also good physicians and chest physicians must know the indications for, and the scope of, thoracic surgery.

The keyword in their line of attack is "rest". Each of the many surgical procedures devised against pulmonary tuberculosis, apart from lobectomy or pneumonectomy, aims at attaining local rest of the diseased part. The methods most widely used, and which have stood the test of time, are artificial pneumothorax and thoracoplasty. Although opinions still differ on the matter, it is becoming more generally realised that thoracoscopy and pneumonolysis are essential accompaniments of successful artificial pneumothorax therapy. The latter is a highly skilled and delicate surgical manoeuvre.

As empyema is the most dreaded and the most intractable complication of artificial pneumothorax it merits research. Moreover, empyema is a fairly common complication of lobectomy or pneumonectomy, and in many cases it requires extensive thoracoplastic operations for its cure.

Because empyema is such a drawback in sanatorium, and indeed in tuberculosis clinic, practice, I have chosen it for special study, and I hope to show later that cases of empyema should come under the care of thoracic surgeons at a much earlier period than many of them do at present.

It will be appreciated that in such a chronic disease as tuberculous empyema, opinions differ widely as to its exact causation and treatment. The same divergence of views is seen in the choice of cases for, and the management

of, artificial pneumothorax which is connected, so intimately, with tuberculous empyema.

This work covers an initial period of general study of the problem at Glenafton Sanatorium, Ayrshire, from January/1946 to March/1947, and then a period of intensive work, detailed investigation and observation from 1st.April/1947 to date at Baguley Sanatorium, Manchester. The latter hospital has 420 beds for pulmonary tuberculosis in addition to a military wing of 120 beds. As Deputy Medical Superintendent I have worked in the team under Dr. Trayer, Physician Superintendent, and Mr. Graham Bryce, Consultant Chest Surgeon for the region, to both of whom I am indebted for permission to carry out any research I desired.

It is my intention, as a result of personal observation, to try to make some contribution, however slight, to our knowledge of the condition. I shall try to place in correct perspective the factors concerned in its causation. Then the management of the lesion will be described, and finally I shall endeavour to show that one should adopt early surgical as opposed to persistant medical measures.

CHAPTER I.

Definition:

The bibliography on tuberculous empyema is enormous. Practically every article on artificial pneumothorax therapy or adhesion section mentions the name. The incidence and prognosis varies widely in different writers' hands because there is no strict criterion of what a tuberculous empyema is. Gibbons (1) defines an empyema as "any collection of fluid in the pleural space, containing bacteria". Woodruff's (2) definition is "any turbid pleural fluid in the presence of pulmonary tuberculosis or any fluid containing tubercle bacilli on direct smear." Coryllos (3) includes as empyemata only "turbid and pus-like" exudates.

In one series of 155 cases with clear pleural fluids throughout their illness in Baguley Sanatorium, only 26 had tubercle bacilli, and 129 showed no bacilli at any time, by direct smear and Ziehl Neelsen staining. If the 129 negative fluids had been concentrated and centrifuged they would have yielded a few more positive findings. Similarly, good culture technique or guinea-pig inoculation would have improved the yield of tubercle bacilli positive cases. This is a well established principle in tuberculosis work with pleural fluids, sputa examination, and with pulmonary lavage fluid as I have repeatedly found. Close (4) noted that by improved technique and cultural methods he found as many as 75% of 23 pleural effusions from young men of the Royal Navy to be tubercle bacilli positive. Tergolina (5) found 87% of his pleural fluids developing during artificial pneumothorax treatment to be tubercle bacilli positive by culture on Petragnani medium. I mention this to show how the definition of empyema given by Gibbons (1), etc., is not sufficiently accurate. I have included in this work only cases of pulmonary tuberculosis who had purulent pleural effusion, and my definition of empyema is "pus in the pleural cavity". This is important because the prognosis, incidence, and treatment of cases with clear pleural fluid and of cases with pus, vary enormously. There are border-line cases in which a

turbid serum or honey-like fluid is present. A good working rule which I have made is to note whether the scale markings on a new 2 c.c. record syringe can be seen through the fluid in the syringe barrel under a good light. If they cannot be discerned the fluid, if not haemorrhagic, is classed as purulent. If I have erred in my own cases as regards record purposes, it has been on the side of omitting many borderline cases from the series and classing them simply as pleural effusions.

Initial investigation:

The first part of this investigation was to scan the individual case records of 7,206 patients with pulmonary tuberculosis who were in Baguley Sanatorium between 1/1/1929 and 1/1/1948. All the empyema cases were extracted; there is no card index system at present to facilitate matters. The cases, drawn from Manchester for the most part, have mainly advanced disease on admission and are all adult cases. Children are not admitted to the Sanatorium. The total field comprises 2,157 deaths and 5,049 who were discharged or left the hospital for one reason or another.

Housing shortages, overcrowding, and poor social conditions are common in the areas from which the hospital derives most of its cases. Persons with positive sputum living in overcrowded houses with children, or patients in common lodging houses, etc., get priority of admission over early cases. This determines the case classification incidence on admission as R.B.2 - 62.0%, R.B.3 - 20.4%, R.B.1 - 2%, and all negative cases (mostly in the R.A.2 group) - 15.6% of the above total of 7,206 patients (Ministry of Health (46)). Thus R.B.2 and R.B.3 cases comprise 82.4% of all admissions for the past 20 years and this figure shows the poor material with which we are working. In view of the bad prognosis of such cases any sputum conversion is a real gain and hence attempts at active treatment may be justified.

At the very outset this work gave a correct perspective of the problem of tuberculous empyema. As regards spontaneous pneumothorax cases the number developing empyema

and the number escaping this complication were noted. No detailed analysis of them was made as the cause of the empyema is obvious.

Each of the remaining empyemas was analysed under the headings of - age at development of empyema, sex, case classification at onset of empyema, duration of empyema, blood sedimentation rate at onset, and the treatment adopted. It was noted what condition predisposed to the empyema; if an artificial pneumothorax was concerned its course was analysed under such headings as pyrexia on induction and the time of onset of empyema from induction or from thoracoscopy. If positive pressure refills were used this fact was noted and the time of onset of empyema after such positive pressure was ascertained.

The outcome of each case of empyema was determined. In a few cases where the patient had left the hospital this was impossible, although an attempt was made to obtain the information from the tuberculosis officers concerned. The X-rays of each case, where available, were studied and reduction photographs were made by myself. Apart from those in which tuberculous empyemata developed, the numbers of all other artificial pneumothoraces, with and without positive pressures, were determined, and the numbers of those with fluid effusion, sufficiently copious to warrant diagnostic aspiration or to cover the hemi-diaphragm completely, were noted. In this way it was hoped to find some common recurring factor which when analysed would yield a clue to the most prevalent modes of onset of empyema and the best way to manage this intractable condition.

No work of this sort is of any value unless it is known how many artificial pneumothoraces, radiologically similar to those developing empyema, existed and escaped that complication. Accordingly the other artificial pneumothoraces during the period in question were analysed and sorted into various groups. Table I gives the overall figures extracted from the investigation. This table will be broken up into separate parts later.

TABLE 1.	Empyemas not associated with spontaneous pneumothoraces.		Negative Pressure Artificial Pneumothoraces.		Positive Pressure Artificial Pneumothoraces.		Spontaneous Pneumothoraces.		Total Deaths.	Total Discharges	Deaths + Discharges	Total Empyemas from all causes.
Year.	A. Death Records.	B. Discharge Records.	A. No Fluid.	B. With Fluid.	A. No Fluid.	B. With Fluid.	A. Escaping Empyema	B. Developing Empyema.				
1929 - 30.	2	-	-	-	2	-	5	3	310	301	611	5
1930 - 32	6	1	3	4	1	2	4	3	305	492	797	10
1932 - 34	5	4	9	4	8	7	4	8	278	511	789	17
1934 - 36	8	3	26	15	4	7	11	5	246	575	821	16
1936 - 38	1	-	32	17	2	5	5	5	236	526	762	6
1938 - 40	2	3	43	23	2	1	3	10	181	553	734	15
1940 - 42	8	5	34	13	1	-	1	-	161	411	572	13
1942 - 44	6	10	46	22	1	2	2	3	149	530	679	19
1944 - 46	8	8	90	43	-	-	2	2	175	638	813	18
1946 - Jan. 1st. 1948.	11	13	83	56	1	1	3	3	116	512	628	27
TOTALS:-	57	47	366	197	22	25	40	42	2,157	5,049	7,206	146.

Spontaneous Pneumothorax:

In the 7,206 cases of pulmonary tuberculosis reviewed, spontaneous pneumothorax occurred in 82 cases apart from artificial pneumothorax therapy. This represents an incidence of 1.13%. Grancher and Hutinel (6) state that spontaneous pneumothorax occurs in 1 in 100 old cases of pulmonary tuberculosis. Of the 82 cases of spontaneous pneumothorax 51.2% developed purulent effusions and their subsequent course was the same as that of empyemata developing from any other cause. The remaining 40 cases of spontaneous pneumothorax did not develop purulent effusion. In 25 of those cases the lung was re-expanded with no significant pleural effusion (i.e., not sufficient to warrant aspiration or to cover the hemi-diaphragm). Eleven cases developed fluid effusion, in some patients containing tubercle bacilli, but this fluid was not described as purulent. There were four deaths directly attributable to the spontaneous pneumothorax, one of them accompanied by spontaneous haemo-pneumothorax.

The small incidence of spontaneous pneumothoraces to total hospital cases (1.13%) is explainable on the basis of the advanced type of case with which we are mainly dealing. The longer the pulmonary disease exists the more pleural adhesions form and the more difficult is it for a spontaneous pneumothorax to occur. Grancher and Hutinel (6) quote Weil as having studied 46 cases of spontaneous pneumothorax and 40 of those occurred in the first 4 months of the disease.

The percentage of empyemas due to spontaneous pneumothorax, to all empyemata in Baguley Sanatorium during the period, is 28.8%. The 42 cases of empyemata due to spontaneous pneumothorax were not individually analysed. Once they have developed, their course and prognosis are identical with those of empyemata due to other causes.

The bulk of empyemata in the series, 104 in number or 71.2% of the total empyemata, have been analysed in detail. (Table 2). Of those 93 were associated with artificial pneumothorax therapy and 11 were apparently due to causes other than artificial pneumothorax or spontaneous pneumothorax.

ANALYSIS OF EMPYEMATA - TOTAL NUMBER 146.

TABLE 2.

1	2	3	4	5	6	7	8	9	10	11	12
Year.	Numbers due to Spontaneous Pneumothoraces.	Percentage of numbers due to S.P.T.'s to yearly total due to all causes. (Column 9).	Numbers due to Artificial Pneumothoraces. A. No adhesion section within 3 months of empyema onset.	Numbers due to Artificial Pneumothoraces. B. Onset within 3 months of adhesion section,	No. of other adhesion sections in the same period.	Total empyemas due to A.P.T. therapy. (Columns 4 & 5).	Causes other than A.P.T. or S.P.T.	Period total of empyemas. (Cols. 2, 7 & 8).	Percentage of total empyemas due to A.P.T.(Col.7), to all A.P.T.'S done (Col.12.)	Nos. of all A.P.T.'S not developing empyema.	Total A.P.T.'s done. (Cols. 7 & 11).
1929 - 30	3	60%	1	-	-	1	1	5	33.3%	2	3
1930 - 32	3	30%	5	-	-	5	2	10	33.3%	10	15
1932 - 34	8	47%	7	-	-	7	2	17	20%	28	35
1934 - 36	5	31.25%	10	-	-	10	1	16	16.1%	52	62
1936 - 38	5	83.3%	1	-	-	1	-	6	1.8%	56	57
1938 - 40	10	66.7%	5	* -	2	5	-	15	6.8%	69	74
1940 - 42	-	-	10	2	1	12	1	13	20%	48	60
1942 - 44	3	15.8%	13	2	10	15	1	19	17.4%	71	86
1944 - 46	2	11.1%	7	9	16	16	-	18	12.03%	133	149
1946 - 48	3	11.1%	16	5	29	21	3	27	12.9%	141	162
TOTALS:-	42	28.8%	75	18	58	93	11	146	13.2%	610	703

* Adhesion section was first used in this period.

Therefore at this stage in the investigation it is clearly seen that the causes of empyema over a 19 year period, in this large sanatorium serving the City of Manchester, are in order of frequency - artificial pneumothorax therapy, spontaneous pneumothorax and a miscellaneous group (Table 3).

TABLE 3.

Causes.	Numbers.	% of total.
Artificial pneumothorax therapy.	93	63.7%.
Spontaneous pneumothorax.	42	28.8%.
Causes other than Artificial pneumothorax or spontaneous pneumothorax.	11	7.5%.
TOTALS:	146	100.0%.

A glance at column 3 of table 2 reveals that with increasing artificial pneumothorax therapy the general trend is for the number of empyemas due to spontaneous pneumothoraces, calculated as a percentage of the yearly empyema total, to fall.

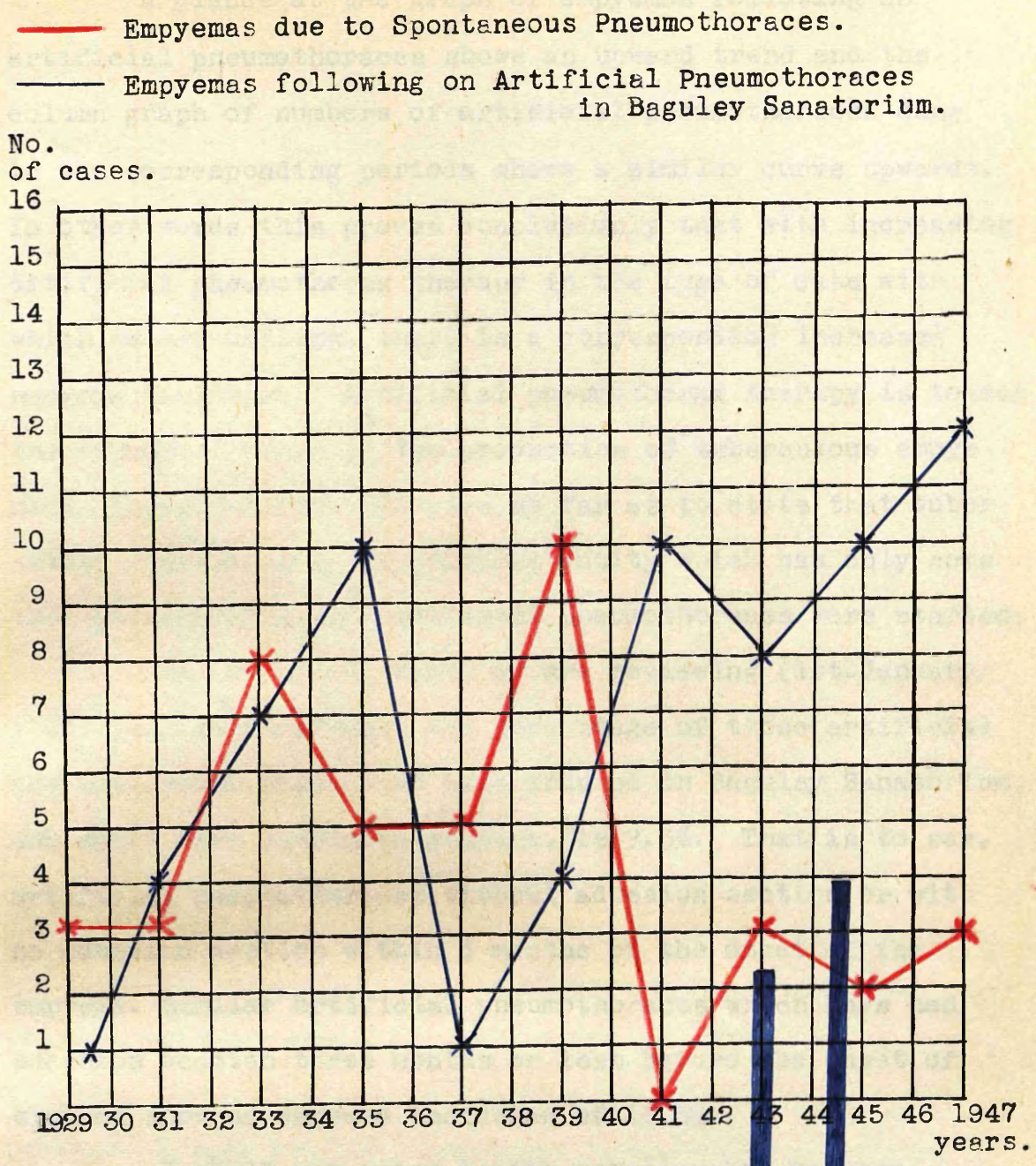
At this stage, in order to assess the true incidence rate of empyemas in artificial pneumothoraces, it is necessary to correct table 2 by subtracting from the numbers those cases who were admitted with empyemata. To include them, when their artificial pneumothoraces were commenced elsewhere, would be to give too high an empyema incidence rate in Baguley Sanatorium artificial pneumothoraces. Table 4 makes this correction.

TABLE 4.

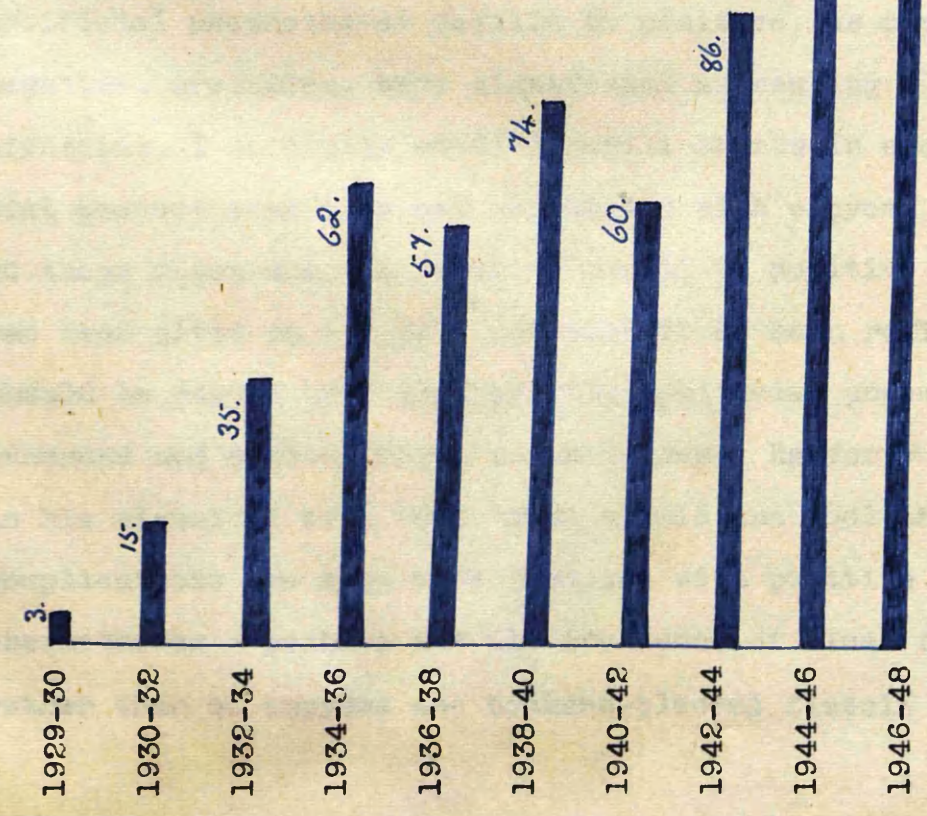
Year.	1 Total empyemas admitted.	2 Numbers of (1) due to artificial pneumothorax.	3 Corrected Baguley empyemas due to A.P.T. therapy	4 Baguley empyemas due to A.P.T. and not attributable to Adhesion Section.	5 No. of Baguley empyemas within 3/12 of adhesiotomy.	6 Total A.P.T. in the period.	7 Total Adhesion Sections in the period.
1929-30	0	-	1	1	-	3	0
1930-32	1	1	4	4	-	15	0
1932-34	0	-	7	7	-	35	0
1934-36	0	-	10	10	-	62	0
1936-38	0	-	1	1	-	57	0
1938-40	1	1	4	4	-	74	2
1940-42	3	2	10	8	2	60	3
1942-44	8	7	8	6	2	86	12
1944-46	6	6	10	5	5	149	21
1946-48	10	9	12	8	4	162	33
TOTALS:	29	26	67	54	13	703	71.

With the hospital bed state remaining fairly constant one can compare by graph the numbers of empyemas due to spontaneous pneumothoraces (column 2, table 2) with the empyemas, developing in Baguley Sanatorium, due to artificial pneumothorax therapy (column 3, table 4).

Apart from two peaks in the periods 1932-34 and 1938-40 the trend of empyemata due to spontaneous pneumothoraces is horizontal and the average is 4.4 per 2 year period.



Graph showing distribution of the total numbers of artificial pneumothoraces done (703).



A glance at the graph of empyemas following on artificial pneumothoraces shows an upward trend and the column graph of numbers of artificial pneumothoraces done in the corresponding periods shows a similar curve upwards. In other words this proves conclusively that with increasing artificial pneumothorax therapy in the type of case with which we are dealing, there is a corresponding increased empyema incidence. Artificial pneumothorax therapy is to-day the commonest cause in the production of tuberculous empyemata. Indeed Freeman (7) goes so far as to state that tuberculous empyema is a new clinical entity which has only come into prominence since artificial pneumothoraces were started.

In the period which we are reviewing (1st.January/1929 - 1st.January/1948) the percentage of those artificial pneumothoraces which have been induced in Baguley Sanatorium, and which have developed empyema, is 9.5%. That is to say, artificial pneumothoraces without adhesion section or with no adhesion section within 3 months of the onset of the empyema. Similar artificial pneumothoraces which have had adhesion section three months or less before the onset of empyema show an empyema incidence of 18.3%.

I shall now point to the relationship between empyema incidence and the incidence of pleural fluid (non-purulent) in artificial pneumothoraces.

In order to settle the question as to whether or not artificial pneumothorax refills to positive, as opposed to negative, pressures, were significant in causing pleural effusions, I carefully studied refill charts in each artificial pneumothorax case not associated with empyema and recorded those cases where a definite refill to positive pressures had been given on a single occasion or at each refill. It should be stated here that all the artificial pneumothoraces recorded had existed for 4 weeks or over. Rafferty (8) states in his classical work that "most clinicians feel that serious complications are much more frequent with positive pressure pneumothorax - perhaps not the incidence of clear fluid, but rather than of empyema and broncho-pleural fistula due to

continued tension through and rupture of unsevered adhesions". Ustvedt (9) states that a very common result of high pressure pneumothorax is the appearance of pleural exudate and mediastinal displacement. Ulmar (10) states that his experience does not bear out the impression that high pressure artificial pneumothoraces have more tendency to fluid. He thinks that pressure has very little, if anything, to do with the actual appearance of fluid. This may be so with completely free artificial pneumothoraces such as one sees in dealing with a hospital population in the R.B.1 class (the ideal state of affairs). When dealing with cases such as ours where over 80% of all admissions are in the R.B.2 or R.B.3 category, free artificial pneumothoraces are in the minority and I believe that positive pressure refills not only cause increased fluid but are definitely dangerous.

Table 5 shows the relationship of non-purulent fluid to artificial pneumothoraces with and without positive pressure refills. Table I gives the numbers in each class for the various two year periods. Only fluid of sufficient quantity to cover the hemi-diaphragm or to warrant aspiration is counted. I have not included those cases where there is a minimal amount of fluid in the costo-phrenic angle.

TABLE 5.

Negative Pressure artificial pneumothoraces without fluid.	366
Negative Pressure artificial pneumothoraces developing fluid.	197
% negative pressure artificial pneumothoraces developing fluid to all negative pressure artificial pneumothoraces.	35%.
Positive pressure artificial pneumothoraces without fluid.	22
Positive pressure artificial pneumothoraces developing fluid.	25
% positive pressure artificial pneumothoraces with fluid to all positive pressure artificial pneumothoraces.	53.2%.
Total artificial pneumothoraces excluding those which had empyema.	610.

Table 5 proves that in proportion, positive pressure artificial pneumothoraces develop half as many clear fluids again as artificial pneumothoraces with negative pressure, i.e., the ratio is approximately 1.5 to 1.

Table 6 is constructed to show, for each two year period under review, the numbers of all artificial pneumothoraces developing non-purulent fluid calculated as a percentage of the total artificial pneumothoraces done (column 3). It shows also, for the same periods, the numbers of corrected Baguley Sanatorium empyemas calculated as a percentage of all artificial pneumothoraces in the period (column 5).

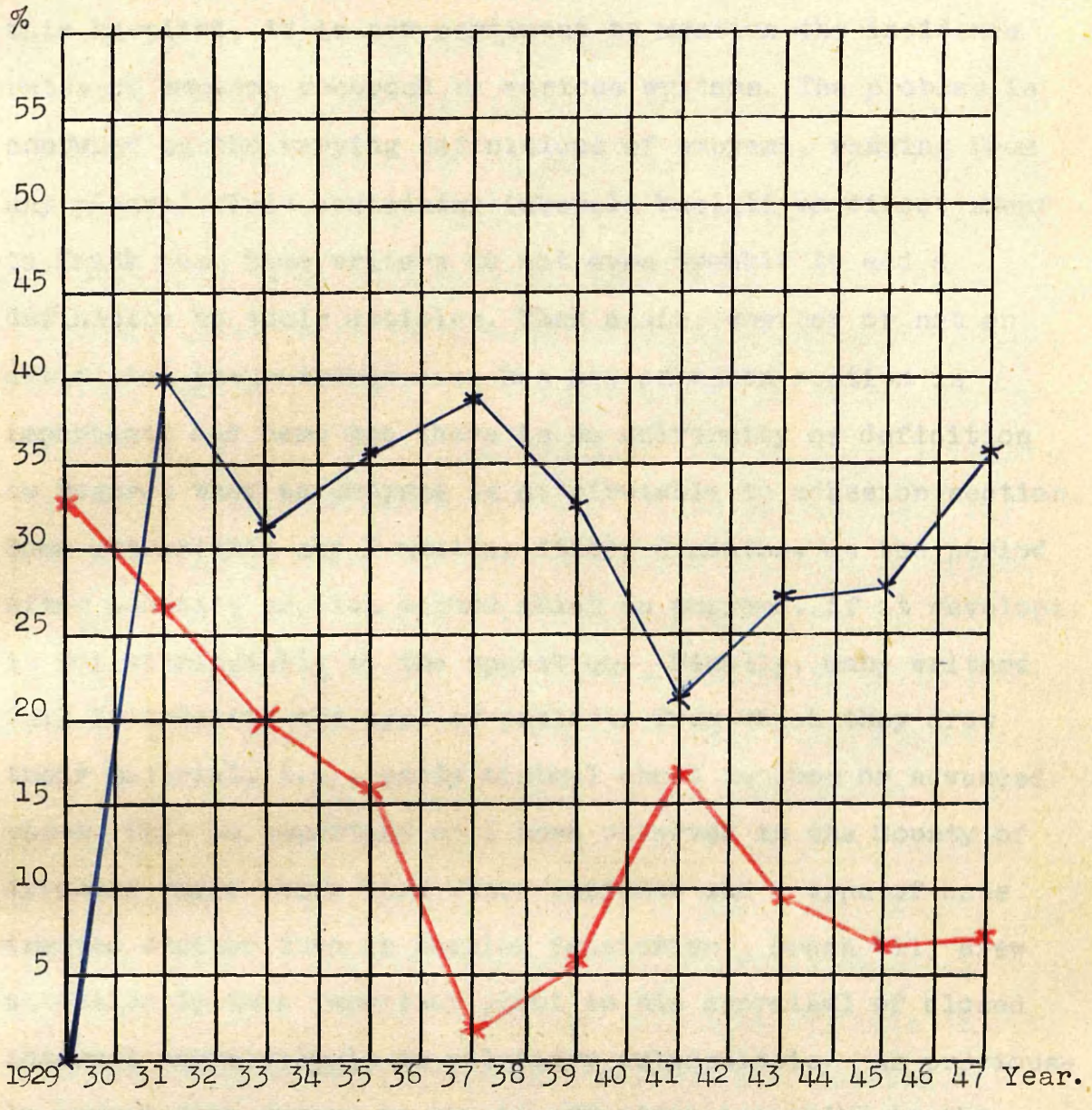
TABLE 6.

Year.	1 Total artificial pneumothoraces.	2 Artificial pneumothoraces developing fluid.	3 Percentage of (2) to total artificial pneumothoraces (1).	4 Empyemas due to artificial pneumothoraces.	5 Percentage of (4) to total artificial pneumothoraces (1).
1929-30	3	0	0%	1	33.3%
1930-32	15	6	40%	4	26.7%
1932-34	35	11	31.4%	7	20.0%
1934-36	62	22	35.5%	10	16.1%
1936-38	57	22	38.6%	1	1.7%
1938-40	74	24	32.4%	4	5.4%
1940-42	60	13	21.7%	10	16.7%
1942-44	86	24	27.9%	8	9.3%
1944-46	149	43	28.9%	10	6.7%
1946-48	162	57	35.2%	12	7.4%
TOTALS:	703	222	31.6%	67	9.5%.

As mentioned at the beginning of this chapter the dividing line between turbid fluids and thin pus is hard to determine. I have included in the empyemata only fluids definitely described as purulent. Turbid or cloudy fluids were omitted from the empyema list. Allowing for this and for the fact that over the nineteen year period studied, different medical officers, with different ideas of what is and what is not a turbid fluid, were concerned with the aspirations, it is hardly logical to construct a graph comparing columns 3 and 5 of Table 6. However, it is included as a rough guide to the relationship to artificial pneumothoraces of non-purulent and purulent fluids.

——— Percentage of non-purulent fluids to all artificial pneumothoraces.

——— Percentage of empyemas to all artificial pneumothoraces.



From year to year there seems to be no constant relationship between the numbers of non-purulent fluid cases and true empyemata. Many small but important points have to be attended to in the course of artificial pneumothorax therapy. Thus large and irregular refills, trauma to the lung surface at refills, positive pressure refills or the maintaining of an ineffective artificial pneumothorax just too long to prevent disaster, all play their part in adding to fluid complications, as does the type of case selected for artificial pneumothorax therapy. When the personal factors, of different medical officers having charge of the cases over the nineteen years, are remembered in connection with those headings, I think this is sufficient to explain the fluctuations in the percentages, of the foregoing graph.

Recorded Incidence of Tuberculous Empyema.

Having shown the magnitude of the empyema problem in this hospital, it is now pertinent to mention the incidence rates of empyema recorded by various writers. The problem is confused by the varying definitions of empyema, ranging from any pleural fluid containing tubercle bacilli on direct smear to frank pus. Some writers do not even trouble to add a definition to their articles. Then again, whether or not an artificial pneumothorax case has had adhesion section is important, and here too there is no uniformity of definition as regards when an empyema is attributable to adhesion section. Some authorities say 3 months, others 6 months, as the period after adhesion section beyond which an empyema, if it develops, is not attributable to the operation. Finally, many writers fail to indicate the type of patients from which they draw their material, i.e., early minimal chest lesions or advanced cases. This is important as I have observed in the County of Ayrshire where there were fewer empyemas and a type of case treated earlier than in Baguley Sanatorium. Drash (11) drew attention to this important point in his appraisal of closed internal pneumonolysis in pulmonary tuberculosis. As previously proved with regard to simple effusions occurring in the course of artificial pneumothorax therapy, refills to positive or negative pressures may have some bearing on empyema incidence and this factor is rarely mentioned. Jacoboeus (12) in 1923, describing his results with 130 cases of adhesion section, noted a slight effusion or absence of pleural exudate in 111 cases. A lasting exudate with pyrexia was present in 21 cases and true empyema in 14 cases (10.8%). In 1927 Hayes (13) stated that 21% of artificial pneumothorax cases developed purulent effusions. The following year Peters (14), discussing his reasons for discontinuing artificial pneumothorax in 218 cases, stated that it was because of a massive exudate in 2.8% and an empyema or ruptured lung in 8.3% of the series. In 1929 Dumarest and Brette (15) found that 86% of artificial pneumothorax cases developed pleural effusions but this figure covers all types of effusion.

Matson (16) in 1934 compared 3 methods of severing adhesions in artificial pneumothorax. With the original galvano-cautery technique of Jacoboeus, out of 136 cases he had purulent exudates following in 36 or 26.4% of the cases. Using his own electro-surgical method he had a lower empyema incidence, i.e., with an unnamed high frequency unit out of 35 cases 4 or 11.4% developed empyema. Using a Bovie high frequency unit, in 78 operations he had an empyema incidence of 2.5%. Weisman (17) in 1936 found effusions in 84% of 150 consecutive artificial pneumothoraces. 48% of those effusions were of considerable amount and 67% of his patients had far advanced tuberculosis. Weisman collected a series of reports and noted from them purulent effusion in 5% to 21% of all artificial pneumothoraces, and clear effusions in 17.1% to 90% of artificial pneumothoraces. Woodruff (2), whose definition of an empyema is "any turbid fluid in the presence of pulmonary tuberculosis or any fluid containing tubercle bacilli on direct smear", in 1937 analysed the development of 154 cases of "empyema" sent to him for treatment by 12 different physicians. While the article does not give the incidence of fluid in all artificial pneumothoraces, it lets us know the relative importance of the causes of effusion, viz:-

Developed from:-	Pleurisy with effusion	Spontaneous pneumothorax	Artificial pneumothorax	Following pneumonolysis	Infection after thoracoplasty	Other and uncertain causes	TOTAL.
Cases	12	5	121	8	4	4	154.

Again in 1937 Drash (11) in his paper on internal pneumonolysis quotes Matson as having had a 12% tuberculous empyema incidence in 480 artificial pneumothoraces. Drash himself had no cases of pyogenic or mixed infection empyema in his series; six or 2.6% of his cases developed tuberculous empyema and of those only 1 developed it within less than 5 months after operation. The longest time of development after operation was 30 months. As stated above my criterion for adhesion section as the predisposing cause of empyema is that the latter should develop within 3 months of the operation. Chandler (18)

(1937) in an analysis of 210 consecutive adhesion section operations found non-purulent effusions following operation in 41 cases and empyema in 8 cases (3.1%). Alexander (19) in 1937 cites the total empyema incidence following 111 adhesion sections as 5.4% (6 cases), while transient fluid was seen in 31 cases and persistant fluid in 11 cases. In 1938 Brock (20) described his results with 442 consecutive adhesiotomies. His table is best reproduced for clarity. The total empyema incidence is 2.8%.

Fluid formation.	Operations.	Percentage.
Nil.	232	64.5%
Very small amount.	81	22.5%
Moderate.	29	
Much or persistant.	8	
Purulent effusion (Tubercle Bacilli only).	5	1.4%
Purulent (secondary pyogenic organisms).	5	1.4%

It is noteworthy that Brock believes that all artificial pneumothoraces should be made complete so as to ensure permanent and concentric collapse. This is, in my opinion, highly important, and of course such complete artificial pneumothoraces are very rarely obtained in conducting artificial pneumothorax therapy on R.B.2 and R.B.3 cases. Brock does not state the case classification of his subject matter.

In 1941 Cutler (21) produced one of the best articles which I have seen on tuberculous empyema. He demonstrated that mechanically poor collapses give rise to a much higher percentage of empyemas than mechanically good collapses when considered in similar stages of the disease. He considered "turbid or purulent effusions positive for tubercle bacilli on direct smear" as empyemata, and he also considered the time factor in relation to onset of empyema from induction of artificial pneumothorax. As I have stated previously all those points must be considered if the article is to be complete, i.e., (definition of empyema, time factor and case

classification analysis). Unfortunately Cutler omits the factor of refills to negative or positive pressures. Cutler's tables are reproduced below. He analysed 476 artificial pneumothorax cases, each of 6 months' duration or more, and considered the incidence of tuberculous empyema without other organisms.

Group 1 were mechanically good artificial pneumothoraces where the collapsed lung was not adherent to the chest wall. 17% of this group were of 6 months to 1 year's duration and 83% were maintained from 1 to 5 years. Pneumonolysis was performed 203 times in this group.

Group 2. Mechanically poor artificial pneumothoraces. The diseased lung was adherent en masse or by numerous or very thick adhesions to the chest wall as seen by X-ray or thoracoscopy.

TABLE I.	Total cases.	Total T.E.	Per-centage	Stage 1.			Stage 2.			Stage 3.		
				Total cases	Total T.E.	%	Total cases	Total T.E.	%	Total cases	Total T.E.	%
Group 1. Mechanically effective artificial pneumothoraces	346	24	7.0	45	0	0	163	7	4.0	138	17	12
Group 2. Mechanically ineffective artificial pneumothoraces	130	35	27.0	6	0	0	40	9	22.5	84	26	31
TOTALS:	476	59	12.4	51	0	0	203	16	8.0	222	43	22

Cutler points out that of the 130 cases in Group 2, 35 or 27% developed tuberculous empyema as compared with 7% in Group 1, i.e., a ratio of 4 to 1, and he shows that this does not coincide with Matson's statement that pure tuberculous empyema occurs with constant frequence irrespective of the type of collapse obtained.

As regards the time factor an analysis of the 59 empyemata yielded the following data. In Group 1 the approximate time of onset of the pus in the pleural cavity after the induction of artificial pneumothorax was from 3 to 53 months, averaging 17.7 months, and in Group 2 from 1 to 32 months, averaging 11.3 months. Cutler concluded that if an empyema is

going to develop we may expect it somewhat sooner in the ineffective pneumothorax group as compared to the effective ones, but the interval in months between the induction of artificial pneumothorax and the appearance of empyema does not differ materially in the two groups.

Goorwitch (22) discussing closed intrapleural pneumonolysis as a contributory factor in empyema onset, in 1944, regarded the operation as attributable if the complication occurred within the first 4 post-operative weeks. In Goorwitch's own series of 55 operations there were no cases of tuberculous empyema or broncho-pleural fistula attributable. 9% developed pleural effusions not classed as empyemata.

In a general article in 1945 Vaccarezza et al (23) found the total incidence of pleural effusion in 158 artificial pneumothoraces to be 55%. That same year Allen and Kelly (24) reviewed 140 patients who had undergone artificial pneumothorax therapy. Twelve of the patients had bilateral pneumothorax. 46.9% of all cases developed clear effusion at some time during treatment but only 17 febrile effusions occurred; there were 1 staphylococcal empyema and 2 tuberculous empyemata. Again in 1945 Laird (25) writing from Clare Hall, Middlesex, made a valuable contribution to the subject. He records 455 thoracoscopies in 325 artificial pneumothoraces. Complications within 3 months of thoracoscopy were 15 pleural effusions, 12 tuberculous empyemata and 1 haemothorax. Laird attributes the low complication incidence to the fact that artificial pneumothoraces are not induced "until the acute phase of the disease has subsided and until the sedimentation rate (Westergren) is below 20 m.m. in the first hour". This may be to a large extent true, but reading between the lines of this article one can picture the early type of case being treated from the fact that in 12 artificial pneumothoraces no adhesions were found on thoracoscopy, and in 170 cases it was possible to divide <u>all</u> adhesions in 1 to 3 sessions. This is a state of affairs one does not find in dealing with R.B.2 and R.B.3 cases. (From talks with various Clare Hall medical officers my surmises on an earlier type of case being treated

there, than here in Manchester, have been proved correct). Jones (26) in November 1945 reviewed 509 adhesion section operations. In 22 cases moderate serous exudate, and in 13 cases empyema, developed. This represents an empyema incidence following adhesion section of 2.6%. Söderhjelm (27) at this time reported an empyema incidence rate of 7.5% in pneumothorax treatment. His empyema total, due to artificial pneumothorax therapy, was 113 cases. In June 1946 Joynt (28) analysed the results in 277 consecutive cases of tuberculosis having 348 adhesion section operations. 16 cases or 5.8% developed a purulent effusion. Paquette (29) in November 1946 concluded that 95% of tuberculous empyemata arise as complications of artificial pneumothorax. His 617 cases of artificial pneumothorax had purulent effusions following in 58 cases or 9.4%.

In 1947 several articles relating to empyema incidence rates appeared. Murphy (30) in 88 artificial pneumothoraces performed 96 adhesion section operations. That he was dealing with a treatable type of case can be deduced from the fact that in 48 patients (50% of total pneumonolyses) there was complete division of all adhesions seen. The fluid complications were 22% with serous accumulations of a transient nature, 2% with purulent effusion, and 2% with broncho-pleural fistula.

Gibbons (1), whose definition of empyema is "any collection of fluid in the pleural space containing bacteria", reviewed a population of 1,024 patients of whom 663 or 67.7% had artificial pneumothorax therapy. Of those latter 78 or 10.2% developed "all types" of empyema but only 10 or 1.5% developed "purulent empyema". The material with which Gibbons was working comprised 18.2% of minimal cases, 50.7% moderately advanced and 31.1% of far advanced cases.

Moore and Watt (31) in 820 adhesion section operations saw 40 cases with appreciable post-operative bleeding. I mention this article because it points to the incidence rate of empyema in haemothorax following pneumonolysis. 14 of their 40 cases of haemorrhage, i.e., 35%, developed empyema.

Watt (32) reported 820 adhesion section operations on 557 different patients. Complete pneumonolysis was obtained in 235 cases, nearly complete in 90, partial in 239, and slight in 23 (again indicating a reasonably early type of case which they were treating). Early pleural effusions occurred in 125 cases and late effusions in 83 cases. Empyemata occurred in 10% of all cases. 8% of early effusions, 24% of late effusions, and 35% of haemothoraces became purulent.

In August 1947 Kunstler (33) reviewed his results with 654 adhesiotomy operations on 500 artificial pneumothoraces. In 17 cases an empyema developed within 6 months after operation, i.e., in 3.4% of cases.

Andosca and Foley (34) reviewed the results when 612 cases of artificial pneumothorax were thoracoscoped and 533 pneumonolysis operations were performed. 17.2% of cases had considerable serous effusions. The total empyema rate was 25.1%, comprising 11.6% of tuberculous empyema and 13.5% mixed infection empyema.

Wollaston (35) reported thoracoscopy and division of adhesions in the cases of 393 patients. The lung was free from the mediastinum above the arch of the aorta in 272 cases, giving selective collapse. Selective collapse with the lung adherent to the mediastinum was obtained in 77 cases, and there was unsatisfactory collapse in 44 cases. 106 cases had sufficient effusion to require aspiration. Empyema developed in 26 cases or 1.6%. From the number of cases in which the author obtained freedom of the lung from mediastinum above the arch of the aorta, and from his statement that on discharge 336 patients were sputum negative, I conclude that he must have been dealing with an early type of case which explains his low empyema incidence.

Bayliss (36) writing from Sydney, Australia, reported an empyema incidence of 6.2% in 268 thoracoscopies with 225 adhesion sections (12 of those occurred within 8 weeks of operation and 2 over 8 but within 16 weeks). Bayliss summarises, in table form, the findings of several authorities on

empyema incidence following adhesion section, and I reproduce his table here.

Author.	No. of Patients.	Type of Empyema.					
		Tuberculous		Pyogenic and Mixed.		Total.	
		No.	%	No.	%	No.	%
Anderson and Alexander.	87	4		2		6	7.2
Brock.	302	5		5		10	3.3
Chandler (1st. series).	89	3		4		7	7.8
Chandler (2nd. series).	68	1		0		1	1.4
Drash.	230	6		0		6	2.6
Edwards & Lynn.	226	32		-		32	13.5
Goorwitch (4 surgeons).	373	-		-		31	8.3
Goorwitch (collected series).	5114	-	3.5	-	1.4	-	4.9
Matson.	249	41		-		41	16.5
Newton.	148	3		-		3	2.0

Finally Day, Chapman and O'Brien (37) in January 1948, analysing 1,000 consecutive operations of closed intrapleural pneumonolysis on 923 patients, report a total empyema incidence of 5.3%. This includes early and late cases.

On reading the foregoing pages it will be noticed how the question of empyema incidence in artificial pneumothorax therapy has come to be that of the incidence in relation to internal pneumonolysis. That is because thoracoscopy and adhesion section has become such an integral part in artificial pneumothorax treatment, and the freedom or otherwise of a collapsed lung, from adhesions, makes all the difference to the ultimate outlook both as regards sputum conversion and intrapleural complications.

I am convinced that figures of empyema incidence can be comparable only when (a) the definition of empyema is standardised, (b) the case classification of the subjects treated is analysed, (c) the time factor as to when an empyema is or is not attributable to adhesion section is constant and (d) the pressures (positive or negative) of

artificial pneumothorax refills are recorded. Very few workers fill to positive pressures now.

In addition, many personal factors such as operational technique and aftercare, and differing clinical judgments and experience, play an important part in various clinicians' hands.

Incidence of Empyema after Lobectomy and Pneumonectomy:

For the sake of completeness I will add a few references to the incidence of empyemata occurring after the removal of tuberculous lobes or lungs. Sometimes those operations are done with the deliberate intention of eradicating tuberculous foci as in one of my cases (Cases 10X, 10K and case M.G., mentioned in the section on tracheobronchitis in the major bronchi, para 'D'). Very often a lobe thought to be the seat of uncomplicated bronchiectasis is later found to have tuberculous foci.

Sweet (38) in 1946 summarised the complications in 27 lobectomies and 36 pneumonectomies for tuberculosis. In the 27 lobectomy operations he had 1 tuberculous empyema without bronchial fistula as an early complication, and 1 early staphylococcal empyema. In the 36 pneumonectomy operations his early complications were 1 tuberculous empyema with bronchial fistula and 1 staphylococcal empyema. Late complications of pneumonectomy were 2 tuberculous empyemata without bronchial fistula. That is to say, in 63 operations he had 6 empyemata or 9.5%.

Overholt, Langer, Szypulski and Wilson (39) reported on their results in 192 patients who had 200 pulmonary resections between 1934 and April 1946. This article cannot be summarised briefly and therefore I will quote it extensively. The main argument is that they have cut down the empyema incidence following operation by better technique in dealing with the bronchial stump and by using a face down, hips elevated, position with intratracheal suction of secretions during the operation. (This also prevents bronchogenic spread to the contralateral lung). The meticulous bronchial closure implies (1) division as near the main carina as possible to

leave a very small bronchial pouch which might act as a sump for secretions. This also permits of good stump retraction into the mediastinum. (2) Suture of the bronchus so as not to interfere with its blood supply, and the use of a pleural flap.

In Group 1 there was no standardised routine for bronchial structures. A "tourniquet technique" was often used and usually no pleural flap.

In Group 2 a pleural flap was used and individual ligation of the hilar structures.

Group 3 cases had this meticulous detail given to bronchial closure, and in addition Overholt's face down, elevated hips position.

The results are as shown in the following tables and they show how the empyema and broncho-pleural fistula rate has fallen.

Complications of Lobectomies.	GROUP I. 1934-Jan.1/42. 7 Lobectomies		GROUP II. Jan.1/42-31.3.44 27 Lobectomies		GROUP III. 31.3.44 - 5.4.46. 35 Lobectomies	
	No.	%	No.	%	No.	%
Tuberculous empyema.						
(a)With fistula	3	42.8	1	3.7	0	0.0
(b)Without "	0	0.0	0	0.0	0	0.0
Non-Tuberculous empyema.						
(a)With fistula.	2	28.6	0	0.0	0	0.0
(b)Without "	0	0.0	0	0.0	0	0.0
Temporary fistula	2	28.6	1	3.7	0	0.0
Permanent fistula	3	42.8	1	3.7	0	0.0
Complications of Pneumonectomies.	GROUP I. 11 Pneumonectomies.		GROUP II. 47 Pneumonectomies.		GROUP III 69 Pneumonectomies.	
	No.	%	No.	%	No.	%
Tuberculous empyema.						
(a)With fistula	1	9.1	2	4.2	2	2.9
(b)Without "	0	0.0	1	2.1	5	7.2
Non-tuberculous empyema.						
(a)With fistula	0	0.0	0	0.0	0	0.0
(b)Without "	1	9.1	4	8.5	1	1.4
Temporary fistula	0	0.0	0	0.0	0	0.0
Permanent "	2	18.2	2	4.2	2	2.9
Ulceration of Bronchial Stump.	0	0.0	1	2.1	1	1.4

Brantigan (40) in 1945 presented 9 case reports of lobectomy for tuberculosis with no instance of pleuro-bronchial fistula or empyema. His late end results were unknown.

In 1947 Bailey (41) gave his results from lung resection on 80 patients with pulmonary tuberculosis. His total empyema incidence was 16 cases.

Carr and Harter (42) in 10 lobectomy and 4 pneumonectomy operations for pulmonary tuberculosis had 7 deaths and tuberculous empyema in 5 of the fatal cases.

Those few references serve to show that tuberculous empyema has to be reckoned with as a complication of pulmonary resection in tuberculosis.

CHAPTER II.

CASES OF EMPYEMA NOT DUE TO ARTIFICIAL PNEUMOTHORAX OR OBVIOUS SPONTANEOUS PNEUMOTHORAX.

A glance back at Table 3 (page 9) shows that 104 empyemata were not due to obvious spontaneous pneumothorax. They comprise 93 cases following artificial pneumothorax therapy and 11 cases stated to be due to causes other than artificial pneumothorax or spontaneous pneumothorax.

In this chapter I propose to analyse the causal factors in those 104 cases, starting with the 11 cases not apparently attributable to spontaneous pneumothorax or artificial pneumothorax. (Case numbers are 1B, 2D, 2G, 3A, 3F, 4A, 7E, 8F, 10F, 10K, 10X. See end of volume for individual case summaries).

Careful study of the earliest X-rays of cases 3A and 4A, before any needling of the chest was done, shows in each case a fluid level indicating the presence of air. Those cases, then, are due to silent spontaneous pneumothoraces with no clinical evidence of this event, and might properly be added on to the figures of empyemas due to spontaneous pneumothoraces.

Case 2D was an empyema following Pott's disease of the thoracic spine, one of the rarer causes of empyema.

Muir (43) mentions the possibility of this occurrence, the parietal pleurae being first involved.

Cases 10K and 10X were due to operative removal of tuberculous lobes, intentional in the latter case, but in the former it was only after operation that the tuberculous nature of the lesion was ascertained.

This leaves 6 cases not explainable on the above grounds, and incidentally their early films are not available for 3 of the cases (10F, 8F and 7E) developed empyema abroad and in the other 3 cases (1B, 2G and 3F) the X-rays cannot be traced. In case 1B the empyema followed on "advanced pulmonary disease". Case 2G had empyema slowly developing in the place of greenish pleural fluid. She had only an R.B.1 classification at the onset of empyema.

The remaining cases 3F, 7E, 8F and 10F had empyema following on acute tuberculous broncho-pneumonia.

The fact remains that of a total of 146 cases of purulent empyema in the 19 year period under review we can explain the exact nature of the onset of all but six of them, i.e., (spontaneous pneumothorax - 44 cases, artificial pneumothorax therapy - 93 cases, operative removal of diseased lung - 2 cases, and Pott's disease of the thoracic spine - 1 case).

It is significant that the clinical history of 5 of the remaining six cases is that of extensive caseous pneumonia before the empyema developed. In four of them post-mortem examinations were made and the caseating nature of the lesions in the parenchyma verified. The final question is, then, whether it is possible to have an empyema following tuberculous disease of the lung without a leak of air (spontaneous pneumothorax) or whether a spontaneous pneumothorax is present in every case of insidious empyema onset.

Muir (43), discussing tuberculosis of the pleurae, states that apart from actual perforation, pyogenic organisms occasionally invade the pleural cavity and give rise to empyema.

Taylor (44) states that empyema may succeed previous serous pleurisy. In 13 cases of this type studied, the duration of the serous-pleurisy, before empyema was diagnosed, was 11½ months; the longest period was 3 years and the shortest 4 months. Case 2G above is in this category.

Fishberg (45) on the other hand states that "whenever purulent effusions occur in tuberculous patients I am suspicious that a latent pneumothorax had existed and this is often overlooked. In most cases of empyema in phthisical subjects I have been able to discover röentgenographic evidence of an air pouch above the level of the fluid."

The point at issue is not just an academic one as the presence or otherwise of a fistula affects the prognosis. My own experience points to there being a spontaneous pneumothorax present in the vast majority of cases of empyema of insidious onset.

ANALYSIS OF DATA RELATING TO THE 104 CASES OF EMPYEMA UNDER REVIEW.

Age and sex: The average age of the patients at the onset of empyema was 26.7 years, the youngest being 16 years (case 6F) and the oldest 49 years (case 7K). The cases comprised 45 males, average age 28.2 years, and 59 females, average age 25.5 years.

Like pulmonary tuberculosis as a whole, tuberculous empyema is affecting people in the young adult age groups.

Case classification at onset.

Seventy-six cases (73%) were in the R.B.2 group and 25 cases (24%) were classified R.B.3 at the onset of the empyema. R.B.1, R.A.2 and R.A.1 cases each accounted for one case of empyema (3% total). The R.A.1 case concerned (No.3F) was one of the eleven cases previously analysed and not associated with artificial pneumothorax or spontaneous pneumothorax. It was later proved tuberculous. The R.A.2 case (No.9F) followed on an artificial pneumothorax and was later proved tuberculous.

Analysis of the case classifications of the 610 artificial pneumothoraces which did not develop empyema (Table 2, column 11) shows that on induction 73.7% were R.B.2 cases, 23.1% were R.B.3 cases, and R.A.1, R.A.2 and R.B.1 cases, together, only constituted 3.2% of the total.

Table 7 summarises the numbers of cases in the various classes (Ministry of Health (46)) calculated as a percentage of the total admissions to hospital between 1st.January/1929 and 1st.January/1948. It shows, too, the numbers of empyemas, calculated as a percentage of the total, developing in each class of case (the class being recorded at the onset of empyema) and lastly, the analysis of all artificial pneumothoraces which did not develop empyemas.

TABLE 7.

Case Classification.	RA.1,RA.2	R.B.1	RB.2	RB.3	RB.2 & RB.3.
Numbers in each class calculated as a % of 7,206 cases studied, 1929 - 48.	15.6%	2%	62%	20.4%	82.4%
Percentages of 104 empyemas in each class.	3%		73%	24%	97%
Percentages of 610 artificial pneumothoraces in each class.	3.2%		73.7%	23.1%	96.8%

The table merely proves that an advanced type of case is, in the main, being treated, and that the majority of all artificial pneumothoraces are induced on R.B.2 and R.B.3 cases. Similarly most empyemas develop in those advanced cases.

Pleural cavity involved.

Empyema was found on the (R) side in 42 cases and on the (L) side in 62 cases. 44% of all artificial pneumothoraces not developing empyema were present on the (R) side and 56% on the (L) side. When the incidence of artificial pneumothoraces in each side was analysed in relation to the sexes, an interesting fact emerged that in women the percentage of (R) artificial pneumothoraces which lasted over one month was 39% while 61% were on the (L) side. In men the

numbers of artificial pneumothoraces were approximately equal on each side.

Sex incidence of empyemas.

59 of the cases occurred in female patients and the remainder (45 cases) in males.

Refills to mean positive pressures.

As Table 5 (page 13) shows, artificial pneumothoraces that are conducted with mean positive pressures develop half as many non-purulent fluids again as artificial pneumothoraces conducted with refills to negative pressures.

Of the 93 cases of artificial pneumothorax developing empyema, 24 cases had refills to mean positive pressures. In most of the cases the mean pressure was just on the positive side of atmospheric pressure, e.g., -3 + 4, - 3 + 5, or -2 + 4 cms. of water. In many cases it is impossible to say that the refill to a positive pressure had any relationship to the empyema onset as for instance one case where the empyema came on 36 weeks from induction and 32 weeks from a refill of -2 +4 cms. H_2O. In other cases the positive pressure is beyond doubt responsible for the emoyema, e.g., case 8B where a contraselective right artificial pneumothorax ran for 4 years uneventfully. Then a single positive pressure refill of -2 +8 cms. water was followed by pus in 3 weeks and death of the patient in a further $6\frac{1}{2}$ months.

Similarly - case 10L had a left artificial pneumothorax with adhesions, running for 15 months, when a single positive pressure refill of 0 + 10 cms. water caused an empyema in 1 week.

Case 10.0 had a (R) empyema developing 1 month after a positive pressure refill of +4 cms. water, and 1 year 5 months from partial adhesion section or $2\frac{1}{4}$ years from induction.

Also in cases 3C, 3E, 3G, 3H, 3I and 1A, mean positive pressure refills appear to be closely related to the onset of empyema.

There is no doubt that refills to positive pressures are harmful and dangerous. If adhesions are present, which

they nearly always are in advanced cases, the lung is traumatised by the constant tugging at the point of insertion of adhesion to visceral pleura. Thus devitalised, caseous foci are liable to form there, and the adhesion may tear apart, causing superadded spontaneous pneumothorax. As examples, like cases 8B, 10L and 10.0 above-mentioned, prove, a contraselective artificial pneumothorax due to adhesions is always a potential danger to the patient, and even a small positive pressure refill accentuates the risk. Also in artificial pneumothorax therapy on R.B.2 cases, it is rarely possible to cut all adhesions and obtain a free artificial pneumothorax (see Table 8). A study of the cases shows that the majority of positive pressure refills were given in the early years of artificial pneumothorax therapy. We do not now refill to mean positive pressures, if possible. Nevertheless we still see cases of artificial pneumothorax sent in from clinics and other institutions where "compressive" as opposed to "relaxing" artificial pneumothorax therapy has been the aim, and the results are often disastrous. (I do not propose here to enlarge on the malpractices of conducting refill clinics without screening control or of permitting the matrons of small sanatoria to give refills in the absence of medical officers).

Blood Sedimentation Rate readings at onset of empyema.

In only 53 of the cases was the Blood Sedimentation Rate record, at the onset of the empyema, available (wide bore method). The range of values was wide, being 45 mm/hour in the highest value (case 8.0) and 10 mm./hour at the lower end of the scale (2 cases - 10L and 10X). The average Blood Sedimentation Rate at onset of empyema was 23 mms./hour. Those figures are of no diagnostic import for uncomplicated cases of pulmonary tuberculosis, when progressing, show an identical range of Blood Sedimentation Rate values. Also it is a well recognised fact that pleural inflammation in pulmonary tuberculosis is apt to be attended by a rise in Blood Sedimentation Rate whether the effusion is purulent or otherwise.

Pyrexia at Induction.

In the 93 cases where the empyema followed on the induction of an artificial pneumothorax, there was pyrexia present, as shown by persistant elevation on the temperature chart for at least a week before induction, in 34 cases. 27 cases were apyrexial on induction of the artificial pneumothorax, and in 32 cases, who were mostly admitted with empyema, there were no temperature records.

Perusal of the case records shows that the presence of pyrexia at induction does not necessarily mean an early onset of empyema in each case. However, the trend is for the onset of empyema to be earlier where there is pyrexia at induction than if the patient had no elevation of temperature. For instance, when cases with adhesion sections or positive pressure refills are removed from the 34 cases who had pyrexia at induction, 14 cases are left. In them the mean time of empyema onset from artificial pneumothorax induction was 4.4 months. When the 27 cases who had no pyrexia at induction are similarly treated, the mean time of onset of empyema from induction, in the remaining 14 cases, was 9.6 months.

Laird (25) stresses the importance of delaying the induction of artificial pneumothoraces until the acute phase of the disease has subsided. Ustvedt (9) prefers to temporize for a while in the face of high fever in the hope that rest may reduce the temperature to normal, before induction. However, he states that high fever is not an absolute contra-indication, although an endeavour should be made, as far as possible, to initiate active treatment during a favourable phase of the disease. Rafferty (8) states that an acute, pneumonic type of disease is a definite contra-indication to artificial pneumothorax.

Persistent pyrexia and elevation of Blood Sedimentation Rate above its normal limits indicate activity of the tuberculous process, and it seems more sensible to keep the pleural surfaces together in such an event rather than to

separate them by artificial pneumothorax, if it is desired to cut out the risk of fluid formation or empyema. In those cases phrenic crush and pneumoperitoneum are often of value in place of artificial pneumothorax as an initial method of active treatment.

Radiological appearances of Artificial Pneumothoraces responsible for empyemata.

Ninety-three of the empyemas in the series were due to artificial pneumothorax therapy. Of those, 26 cases had their artificial pneumothoraces induced elsewhere, and were admitted with empyema. Nevertheless it was possible to obtain X-ray records of the type of artificial pneumothorax which existed before empyema onset in 78 cases. Scrutiny of those X-rays and of the X-rays of other artificial pneumothorax cases not developing empyema, has shown me that it is possible to divide the cases according to radiological appearances into five main groups which are illustrated in Table 8. (Table 7 shows the percentages of all artificial pneumothoraces in each class of case).

Group I represents a "free artificial pneumothorax". There is concentric collapse of the lung which comes freely away from the hilar region without apical or mediastinal adhesions. Semi-aeration is present and no areas of gross opacity or visible cavitation.

Group II. Here there are adhesions visible between chest wall and lung, or apico-mediastinum and lung. Those cases have cavities, in some cases honeycomb in type. There is, however, no "ground-glass" like, milky opacity present.

Group III. Here adhesions are present between the lung and chest wall and there is a "ground-glass" opacity of a lobe or an entire lung but no visible cavities.

Group IV. has adhesions between lung and chest wall, "ground-glass" opacity of a lobe or entire lung, and cavity or cavities visible on the X-ray.

Group V is an apparently free artificial pneumothorax with "ground-glass" like upper lobe containing a cavity.

TABLE 8.

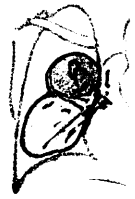
	Free artificial pneumothorax.  Group I.	Adhesions visible but no "ground-glass opacity". All these cases have cavities, some cases honey-comb in type.  Group II.	Adhesions present and "ground-glass" opacity of a lobe or entire lung but no visible cavities.  Group III.	Adhesions present and "ground-glass" opacity of a lobe or entire lung. Cavity obvious on X-ray.  Group IV.	Free artificial pneumothorax but collapsed and "ground-glass" like upper lobe containing a cavity.  Group V.
Duration of artificial pneumothorax.	Under 6 mths. / Over 6 mths.	Under 6 mths. / Over 6 mths.	Under 6 mths. / Over 6 mths.	Under 6 mths. / Over 6 mths.	Under 6 mths. / Over 6 mths.
Section A. Arrangement of 78 empyemas in groups according to the radiological appearances of their causal A.P.T.'s.	Nil / Nil	5 / 7 = 12 = 15.4%	3 / 5 = 8 = 10.3%	28 / 30 = 58 = 74.4%	Nil / Nil
Section B. Analysis of sample, i.e., 201 A.P.T.'s (not developing empyema) according to their radiological appearances.	A sample of the 703 artificial pneumothoraces which did not develop empyema is analysed below:- Nil / 6 = 6; Nil / Fluid Nil	10 / 27 = 37; Fluids / Fluids 2 / 12 = 14	2 / 15 = 17; Fluid / Fluids 1 / 5 = 6	31 / 36 = 67; Fluids / Fluids 16 / 33 = 49	Nil / 5 = 5; Nil / Fluids Nil.
TOTALS:-	Nil / 6	12 / 39	3 / 20	47 / 69	Nil / 5
Percentages of A.P.T.'s in each group.	3%	25.4%	11.4%	57.7%	2.5%

TABLE 8.

It will be at once seen (Section A, Table 8) that 74.4% of the empyemata arise from artificial pneumothoraces in Group IV. Of the 58 cases of empyemata from artificial pneumothoraces in Group IV, 28 cases arose within 6 months, and 30 cases longer than 6 months, from artificial pneumothorax induction.

Section B of Table 8 shows the results of an analysis of a sample of 201 artificial pneumothoraces in which empyema did not develop. The majority (57.7%) or 116 cases are in Group IV. 67 had no fluid copious enough to cover the hemidiaphragm or to warrant aspiration, while 49 developed non-purulent fluid. The table shows how many of the artificial pneumothoraces lasted over one month but under six months, and how many lasted over six months.

Turning again to Section A of Table 8 we see that Group II artificial pneumothoraces accounted for the next high incidence of empyema (12 cases or 15.4%). Similarly, Section B shows that 25.4% of the other artificial pneumothoraces were in this Group.

Group III accounted for 10.3% of empyemas in the sample. 11.4% of all other artificial pneumothoraces fell into this Group.

The free artificial pneumothoraces in Group I and Group V numbered together 11 cases or 5.5%, and no empyemata were found arising from this type of artificial pneumothorax. There is no necessity to elaborate this further. A glance at Table 8 reveals the position better than words.

Thus Groups II and IV accounted for the majority of all artificial pneumothoraces between them. Most empyemas followed this type of artificial pneumothorax and most of the non-purulent pleural fluids. When they are analysed they are seen to have two things in common -

(a) the presence of adhesions;

(b) the persistence of cavities;

i.e., they are contraselective, artificial pneumothoraces.

The most important group of cases, Group IV, has, in addition, that "ground-glass" like, milky, radiological

appearance round the vomica. It follows, therefore, that we must probe the pathology and significance of this radiologically milky area and the role of adhesions.

It is clearly seen by studying Table 8 that the cases with cavities and "ground-glass" appearance (Group IV) are more dangerous than those artificial pneumothoraces in Group III where there is only "ground-glass" appearance and no cavity visible. The behaviour of cavities after artificial pneumothorax induction points in many cases to a valvular factor in the draining bronchus. I therefore propose to study tracheo-bronchial tuberculosis in the next chapter and the role of adhesions in Chapter IV.

CHAPTER III.

TUBERCULOSIS OF THE TRACHEA AND BRONCHI.

In recent years there has been an ever increasing bibliography on this subject, particularly from French and American sources. There is no uniformity of opinion regarding the clinical significance and the incidence rates of tuberculous tracheo-bronchitis.

It is in collapse therapy, especially artificial pneumothorax treatment, that the condition is of most importance. Some authorities (Rafferty (8), Fernandes and Castello Branco (47) and others) go so far as to urge a bronchoscopic examination before an artificial pneumothorax is induced. In this country very few centres do this as a routine procedure. Shortage of theatre staff and of trained bronchoscopists, and long waiting lists, at present preclude this ideal in all but the most up-to-date centres. If tuberculous tracheo-bronchitis is so important in artificial pneumothorax therapy, has it then any relationship, as a causal factor, to empyema? Is it, for instance, the cause of the complete collapse of a whole lobe that one sometimes sees? Will it prevent a collapsed lung from re-expanding? Does tuberculous bronchitis help to produce that ominous ballooning of a cavity one sometimes observes after artificial

pneumothorax induction or adhesion section? Those, and many other matters, claim our interest because the above phenomena are so often associated with the formation of fluid, pus or broncho-pleural fistula.

It is proposed to mention, at this stage, the incidence rates reported by several writers. As far back as 1924 Heaf (48) noted tracheal abnormalities, varying from oedema of the mucosa to general ulceration of the whole surface, in 44% of 133 post-mortem studies.

Two years later Ophuls (49) surveyed 3,000 autopsy records and found that 10% of all active cases had infections of the bronchi or trachea from parenchymal tuberculosis. His figures are of little or no value because at some of the post-mortems no special attention had been paid to the presence or absence of such lesions. (For this very reason the old post-mortem records of Baguley Sanatorium were of no use to me in my researches into this particular subject. It is only recently, with increasingly common artificial pneumothorax therapy, that the subject is assuming great importance and people are looking for such lesions). Since I became interested in the subject I have personally removed trachea, lungs and main bronchi intact with a special view to studying the tracheo-bronchial condition.

From 1,226 autopsy studies Heinze (50) found 8% of tracheal tuberculosis. Willigk (50) and Dambromilsky (50) found tuberculous changes in the trachea in 1.6 and 25.2% of cases respectively, while Peloso (50) noted changes in the upper air passages in all cases of tuberculosis.

Clerk (51) in 1931 advocated bronchoscopy in pulmonary tuberculosis cases to diagnose unexplainable signs and symptoms but was of the opinion that it was a rare event to find a tuberculous involvement of the mucosa of the larger bronchi as a part of pulmonary tuberculosis. No less an authority than Graham (52) in 1935 stated that he had failed to find involvement of the mucosa of the larger bronchi in any of the cases that he had bronchoscoped.

Working in Mexico city, Acuna (53) made a bronchoscopic study of 279 selected patients with pulmonary tuberculosis. Tuberculous tracheo-bronchitis was found in 29.7% or 83 cases. Those were listed as granulomatous ulceration (52 cases), tuberculoma and other hyperplastic lesions (18 cases), solitary ulcer (9 cases) and fibro-stenotic lesions (4 cases). In 85.4% the bronchi were involved, in 7.2% the trachea alone, and in 2.4% the carina was affected.

Salkin, Cadden and Edson (54) found a 40% incidence of tracheo-bronchial tuberculosis in 125 consecutive autopsies of patients who died of pulmonary tuberculosis. In a series of 622 consecutive bronchoscopies 15.5% were affected.

Goldman, Brunn and Ackerman (55) at 100 post-mortem examinations found bronchi draining cavities grossly affected in every case. They also studied the problem of tracheo-bronchial tuberculosis from a bronchoscopist's viewpoint and disclosed evidence of involvement of a major bronchus in about one third of the cases. By the latter method of examination the most common positive findings were oedema and small haemorrhages, while it was less common to find ulcerations or tubercles or granulomatous masses.

McIndoe, Steele, Sampson, Anderson and Leslie (56), analysing 272 unselected cases of pulmonary tuberculosis, found the incidence of tracheal or bronchial tuberculosis to be 11%, while Rafferty (8), in his classical work, states that it is now recognised that specific disease of a major bronchus is associated with pulmonary tuberculosis in 11% of all cases and is, therefore, a common problem; finally, Fernandes and Castello Branco (47) found that 39% of the cases submitted to collapse therapy had tracheo-bronchial lesions bronchoscopically.

Those then are some of the findings re incidence. As I previously stated there is divergence of results. We can divide the lines of research into post-mortem studies and bronchoscopy of the living subject. The former procedure

deals in the main with one class of case, R.B.2 or R.B.3, and is open to the objection that post-mortem changes, in the presence of sputum in the air passages, quickly supervene and alter the appearance of the tracheo-bronchial mucosa. For this reason I have done all my post-mortems as soon after death as possible.

Bronchoscopy is an ideal method of assessing the incidence of the condition, but unfortunately it is not usually stated in articles, in what clinical class the patients belong. This, in my opinion, is most important for the case with much sputum soon develops a chronic bronchitis with patent mucous gland ducts, and lays the way open for submucosal invasion with Koch's bacillus, as opposed to the case in Group R.B.1, with little or no sputum and intact ciliae.

Because of the early post-mortem changes in the bronchial mucosa in the presence of pus the logical procedure is to make a histological examination as well as a macroscopic one. This I did when there was any doubt about the appearances of the mucosa. Chiappe (57), in June 1947, published work on those same lines, while my investigations were in progress. He studied histologically the main or large bronchi which were macroscopically free from tuberculous lesions in 20 patients who had pulmonary tuberculosis. Specific lesions were present in 5 cases and non-specific lesions in 9 cases. Later Sweany and Behm (58) made a post-mortem study of 667 cases, many of them histologically, and found an overall incidence of tracheo-bronchial tuberculosis of 72%.

Finally, the great difference between bronchoscopy and post-mortem analysis is that in the former method the bronchoscopist on the average sees as far as the origins of the pectoral bronchus and the common stem to the apical and sub-apical bronchi in the left upper lobe. In the left lower lobe he may see the origins of the anterior, middle and posterior basal bronchi and the lower lobe apical bronchus. In the (R) upper lobe he sees the origins of the pectoral,

apical and sub-apical bronchi and in the (R) lower lobe the origins of the cardiac bronchus, the 3 basal bronchi and the lower lobe apical bronchus. The origin of the (R) middle lobe bronchus is seen anterior to the latter.

With post-mortem study the distal and finer bronchi may be quickly slit open with pointed scissors and as I shall show later the incidence of bronchitis in those smaller bronchi in the affected lobes is very high. Unless a definite distinction is made of the region examined the incidence is, therefore, bound to be higher in post-mortem studies as opposed to bronchoscopic findings.

TABLE SUMMARY:	Percentage Incidence.			
Author.	No. of cases.	Post-mortem study.	Bronchoscopic study.	Remarks.
Heaf.	133	44%	-	-
Ophuls.	3,000	10%	-	Figures of little value.
Heinze.	1,226	8%	-	-
Willigk.	-	1.6%	-	-
Sweany & Behm.	667	72%	-	Histological and macroscopic study.
Dambromilsky.	-	25.2%	-	-
Peloso.	-	100%	-	-
Clerf.	-	-	Small %	General remarks.
Graham.	-	-	Negative findings	
Acuna.	279	-	29.7%	
Goldman, Brunn & Ackerman.	100 -	100% -	- 33%.	P.M. findings were of bronchi draining cavities.
McIndoe, Steele, Sampson, Anderson & Leslie.	272	-	11%	
Rafferty.	-	-	11%	General remarks.
Fernandes & Castello Branco.	-	-	39%	
Chiappe	20	-	-	25% had specific lesions histologically.
Salken, Cadden & Edson.	125 622	40% -	- 15.5%	

Types of tracheo-bronchitis and their possible bearing on empyema.

From my own experience of the condition based on post-mortem, clinical, and a limited number of bronchoscopic examinations, the following lesions occur in those bronchi within bronchoscopic vision and in the trachea. Laryngeal involvement was excluded because the structure of the larynx offers a site where sputum may lodge. (I purposely make a distinction in the size of the bronchi because tuberculous bronchitis is much more common in the smaller bronchi and, I believe, relatively more important).

A. Generalised bronchitis with here and there small specific ulcers.

B. Solitary ulcerations in the absence of gross, general bronchitis.

C. Complete fibrous stenosis of a bronchus.

D. Partial fibrous stenosis, often with intermittent atelectasis of distal lung, bronchiectasis and a large septic element due to intermittent blocking.

E. Nodules of tuberculous granulation tissue.

F. Endo-bronchial ulceration from pressure and erosion by a caseous, softening hilar lymph gland.

G. Stenosis of a bronchus from pressure from without and no endo-bronchial ulceration.

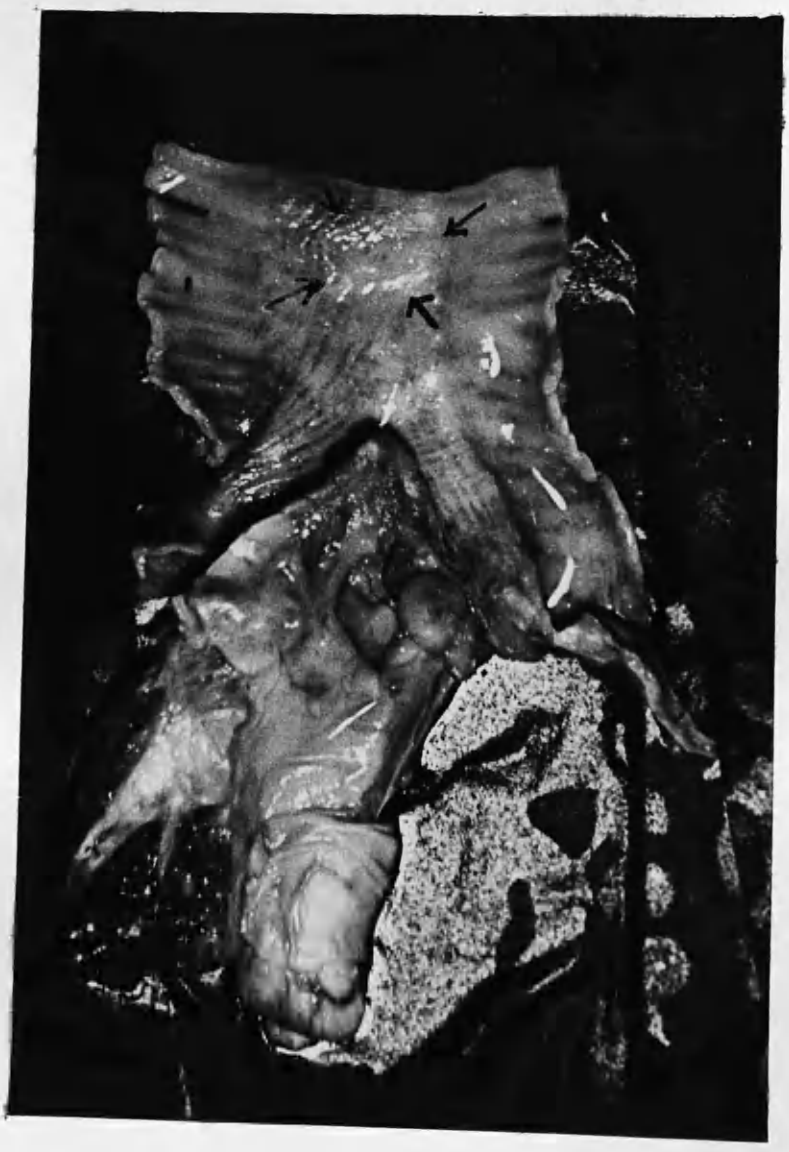
Let us discuss each of those lesions from the point of view of a possible bearing on tuberculous empyema.

A. In cases of advanced pulmonary tuberculosis chronic, non-specific bronchitis is common. Starting with acute bronchitic episodes engendered by the copious muco-purulent secretions in the bronchi, the mucous glands in the bronchial submucosa hypertrophy. The ducts become wider as is readily seen in histological sections and infection with pyogenic organisms in the first instance occurs in the glands. A peri-adenitis follows and the whole mucosa is inflamed. The muscular and elastic tissue is soon destroyed and replaced by fibrous tissue and young blood vessels. The bulk of this submucosal cellular reaction and its pressure on the ciliary

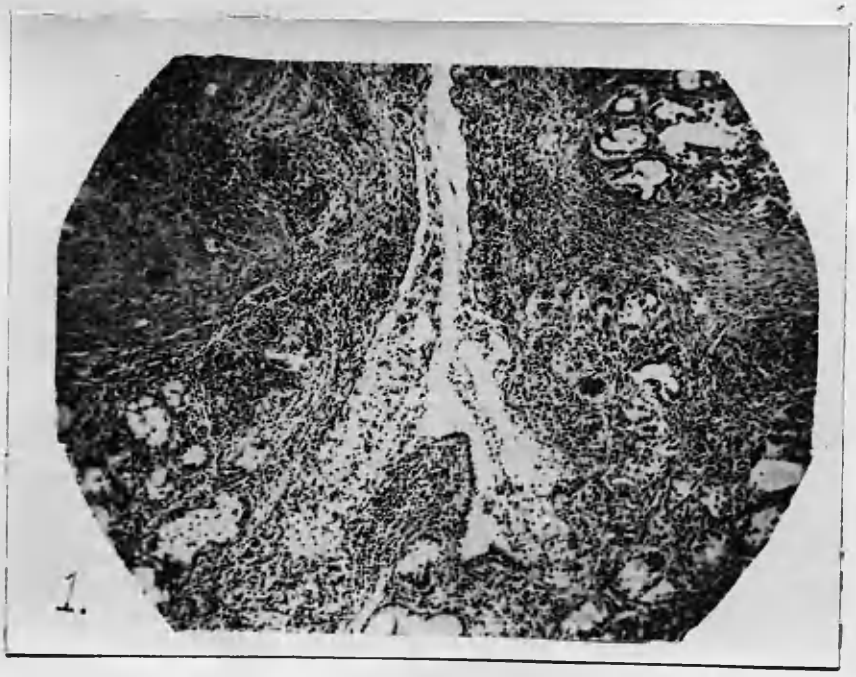
layer causes an atrophy of the latter which is finally replaced by flattened, squamous epithelium. Large areas of bronchial wall lose their ciliae and thus the onward movement of secretions is hindered. The final atrophy of the mucosa leads to gentle undulations in the course of the bronchi and secretions tend to collect in the wider parts and to stagnate and cause further cough and softening of the wall. This condition predisposes to bronchiectasis and emphysema.

This common finding is ordinary, non-specific, chronic bronchitis. It is of the utmost importance in artificial pneumothorax therapy, for the chronic and persistant coughing is one of the factors involved in the production of a tension cavity associated with artificial pneumothorax, even after complete adhesion section, and as I have stressed, such a cavity, associated with pneumothorax, is a danger signal re broncho-pleural fistula and empyema. We are discussing the bronchi within bronchoscopic vision and I have mentioned bronchiectasis which is usually in the more distal bronchi, but it will be appreciated that chronic bronchitis usually affects all the bronchial tree.

At intervals tubercle bacilli reach the submucous layers and the enlarging, resultant tubercle follicle may ulcerate into the lumen, causing small, greyish specific ulcers. The photograph from post-mortem No.4 shows this fairly common finding of chronic bronchitis with solitary tuberculous ulcers. In the section through this ulcer there is a widely patent mucous duct by means of which tubercle bacilli could easily have invaded the submucosa. I have no definite proof, however, that this is the sole means of infection of the submucosa. In this particular case there is a small lymph gland showing extensive tuberculous involvement, deep to the ulcer, in the outer coverings of the trachea and not included in this photomicrograph. Infection could just as well have passed from a lung focus via the peri-bronchial and para-tracheal lymphatics to this node and



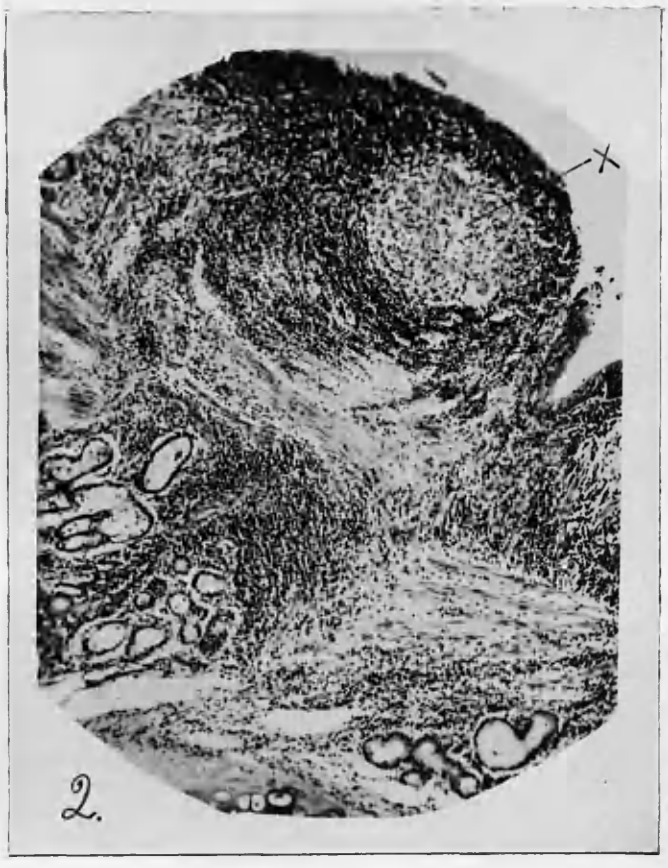
Photograph of Tracheal Ulcer. P.M. no 4.



thence to the submucosa. (Such spread is possible if we remember the spread from a peripheral Ghon's primary focus to the hilar lymph glands via peri-bronchial lymphatics in the formation of Ranke's primary complex). Indeed Reichle and Frost (59) think that infection passes from lung parenchyma or peri-bronchial alveoli via veins or lymphatics to that lobule of the mucous gland which lies external to the cartilage and related to peri-bronchial alveoli.

The photomicrograph, from a section through the base of this ulcer (No.1), shows the intense lymphocytic and polymorphonuclear invasion of the mucous gland and its surrounding tissues. The main duct is wide and contains cellular debris. A tuberculous giant cell is visible on the left side of the mucous duct. High power examination of the area shows that numerous endotheliod cells are present too.

A study of many slides through such bronchi shows constant findings. The specific, tuberculous ulcers commence with aggregations of cells deep to the ciliary layer. Those cells are lymphocytes and plasma cells (slightly larger than the former, with peripheral nuclei and often eosinophilic protoplasm) and endotheliod cells. This is the "proliferative phase" in the production of a tubercle follicle. I have repeatedly observed that the ciliae soon disappear when such a cellular aggregate is present beneath them. Further enlargement of the tubercle leads to the appearance of caseous material in the wall of the bronchi and giant cells, (photographs 2, 3, 4 and 5). The mucous glands especially are surrounded and infiltrated with plasma cells and lymphocytes. On either side of the ulcer normal ciliae are seen. There is usually a secondary septic element superimposed and the debris in the bronchial lumen contains polymorphonuclear leucocytes which are also found in the granulation tissue which now appears and breaks up the regular muscle bundles in the bronchial wall. By this time wide areas of bronchial wall lose their ciliae and the final shrinking of the submucosal granulation tissue leads to a general widening of the lumen.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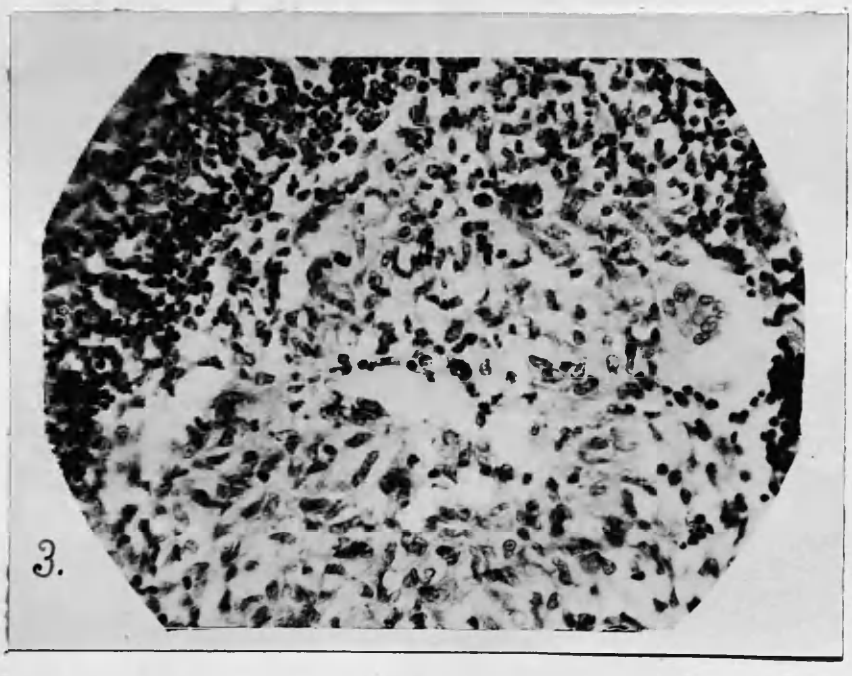
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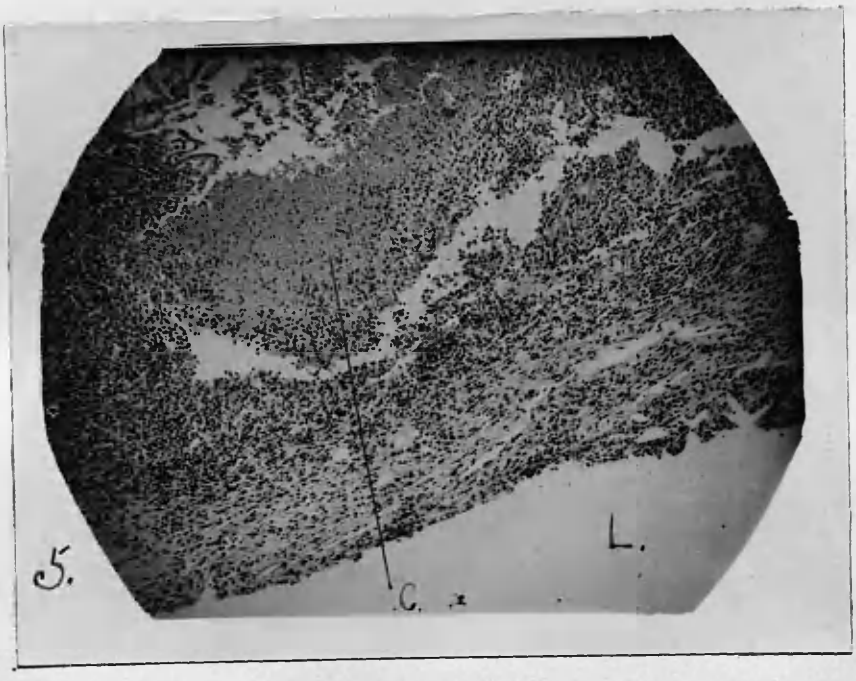


5.

Photograph No.2 (L.P.) is from a section of the draining bronchus of right lower lobe vomica and outwith the area of bronchoscopic vision in case 10X. It was removed at operation. It shows wide round cell infiltration of the submucous area, involvement of mucous glands and disruption of the muscle lines. X marks a proliferative tubercle follicle. The ciliae have vanished over this area. At the bottom of the picture a corner of a cartilage ring is evident.

Photograph No.3 (H.P.) shows the centre of area X in the preceding photograph. The peripheral zone of lymphocytes and plasma cells is evident and the endotheliod cells in the centre.

Photograph No.4 (from post-mortem No.5) is from the right apical bronchus 2nd. degree bronchus, which was macroscopically involved. An L.P. view, stained by Mallory, it shows a thinning of the bronchial wall and vascular granulation tissue in the submucous layer. There is an area in the submucosa packed with round cells, the surface of which is devoid of ciliae and ulcerated.

Photograph No.5 is an L.P. view from a section of the draining bronchus of the left apical cavity in post-mortem No.24. "L" denotes the lumen of the bronchus. The surface ciliae have gone. (High power examination of the cellular infiltration of the mucosa reveals many lymphocytes, plasma cells, endotheliod cells, and occasional giant cells." "C" depicts an area of caseation in the submucosa.

Excess of caseous material in the submucosa leads to the caseous bronchitis described by Pagel and Henke (60) or Eloesser (61) or Sweany and Behm (58). The cartilage rings become surrounded by caseous areas and tubercle follicles in some areas and a perichondritis is produced. This is a common finding and photograph No.2 shows an early stage of it. Cartilage appears to be resistant at first and one sometimes sees thickening of the perichondrium. Later the cartilage is eroded and replaced by a caseous mass.

In post-mortem No.20 (B.M., 27 years) the origin of the left upper lobe bronchus was partially stenosed by a fibrous ring (chronic perichondritis) and on longitudinal section of the bronchus the cartilage was plainly visible and could be felt by scraping with the edge of a scalpel in the centre of the fibrous ring. In other cases section of a bronchus reveals the cartilage ring, still resisting, bathed in a collar of caseation. I mention this type of lesion in detail because I believe that it would be almost impossible for a main bronchus to be so structurally weakened that it would collapse or kink when an artificial pneumothorax was induced.

In my opinion the importance of chronic bronchitis with individual tuberculous ulcers, as regards those bronchi visible by bronchoscopy, is in (a) the coughing produced and (b) the retention of secretion made possible by loss of ciliae. As I have mentioned above, chronic bronchitis plays a much more ominous role in the smaller and medium sized bronchi outwith bronchoscopic vision. We shall discuss them shortly.

B. Solitary ulcerations in the absence of gross general bronchitis.

I have seen those ulcers in the upper end of the trachea when viewing the vocal cords by laryngeal mirror. They may exist at lower levels. Histologically they resemble the tuberculous ulcers previously described. It is rare not to find some degree of chronic bronchitis in such cases. They may be a cause of persistently positive sputum and of cough, and therein lies their importance. Dumarest et al (62) describe a case of large solitary ulcer of the right lower bronchus which was the chief source of a positive sputum.

C. Complete fibrous stenosis of a bronchus.

I, personally, have come across only one example of this in a bronchus which could have been visible bronchoscopically. I have, however, had wider evidence of the condition (by bronchogram and post-mortem studies) in more distal bronchi.

Pagel and Henke (60) describe stenosis or definite closure of the lumen of the affected bronchi by retracting scar tissue in what they call "productive tuberculous bronchitis". The case I saw was in post-mortem No.1 (T.C., 55 years). This man had a fibrotic left upper lobe from old healed disease. The apex of the lung was extensively adherent to the chest wall. The entire left upper lobe bronchus was stenosed from the extreme limit of bronchoscopic vision onwards and there was a cyst-like space, perfectly dry, marking the site of an old cavity. It could not collapse owing to fibrosis around it.

Coutts (63) described a case where the left main bronchus became completely stenosed following left artificial pneumothorax therapy, and on bronchoscopy not even a scar was visible to mark the original opening. Before induction a film showed "a large area of mottling around the left root." The artificial pneumothorax, maintained for 3 years, was complicated by several attacks of atelectasis in the left lung and fluid formation.

Complete fibrous obliteration, then, of a main bronchus is rare, and the question of artificial pneumothorax therapy would hardly enter into such a case once the stenosis had developed so that associated empyema must be very unlikely. In the case described by Coutts the complete stenosis came on after the artificial pneumothorax was abandoned.

D. Partial fibrous stenosis, leading to narrowing of the lumen of a bronchus, is much more common than the preceding variety. Clinically the patient often suffers asthmatical attacks or pyrexial upsets with cough and wheezing. The latter tends to be more marked on one side than on another. Secretions may accumulate in the lobe distal to the obstruction and recurring episodes of pneumonitis occur with pyrexia, malaise and polymorphonuclear leucocytosis. As the bronchial walls become weakened with stagnant secretion, and as the cough persists, bronchiectasis forms, often aided by the pull from outside the bronchi of fibrous tissue in lung repeatedly

attacked by inflammatory episodes. It has been my experience that artificial pneumothorax therapy in the presence of bronchiectasis is always a bad risk, especially if there are tuberculous cavities distal to the diseased bronchial area.

An example of partial, fibrous stenosis of the larger bronchi was seen in post-mortem No.20 (B.M.,27 years). At the origin of the left upper lobe bronchus there was a partial narrowing. This was due to a fibrous ring in whose centre was a cartilage ring. Sections through this area showed fibrous tissue and granulation tissue in which giant cells of tuberculous type were present. This patient's history (she was admitted with left empyema) indicates a tuberculous bronchopneumonia with effusion. There was probably a small spontaneous pneumothorax then. After aspirations two discharging sinuses formed in the left chest wall but they eventually healed, although the empyema cavity was unobliterated. The explanation of this rare event was that she developed a large communication between her left upper lobe vomica and the empyema cavity, and thereafter coughed up her pus.

Another case of mine (M.G., 20 years) had a primary tuberculous complex. Enlarged right hilar glands pressed on the right lower lobe bronchus. There was collapse and fibrosis of the right lower lobe. Subsequently, after 1½ years, the X-rays showed a decrease in the size of the enlarged right hilar glands but a bronchoscopic examination revealed a narrow, fibrous stenosis of the main bronchus to the right lower lobe, and bronchograms revealed bronchiectasis beyond. She was persistently tubercle bacilli plus. At one point the wall of the right lower lobe bronchus must have been ulcerated and subsequent healing caused fibrous stenosis. Lobectomy done at Hairmyres Hospital revealed a shrunken, collapsed and posteriorly placed right lower lobe. It was adherent to the diaphragm. This patient eventually developed a broncho-pleural fistula and basal empyema for which she had to have further operative treatment. (Six months after admission a right artificial pneumothorax had been induced

but abandoned as it proved unsatisfactory).

Lemoine (64) stresses the importance of the bronchiectasis that often follows fibrous stenosis and states that when tuberculous cavitation is present distal to the stenosis, the prognosis is grave. He thinks that any form of treatment is likely to be of little avail. Andrews (65) described two cases of involvement of the main bronchi. There was tuberculous ulceration with caseation necrosis and stenosis.

In the case of partial fibrous stenosis of those bronchi visible bronchoscopically the relation they have to empyema production is as follows - (a) chronic cough and retention of sputum can set up a valve effect and produce ballooning of distal cavities, and (b) bronchiectasis or atelectasis from intermittent blocking of the region can produce serious complications in artificial pneumothorax therapy from the secondary septic element involved. Recurring pneumonitis leads to permanently collapsed and fibrotic lungs which, when an artificial pneumothorax space is present, affords a constant free pleural space - a potential danger in the face of pulmonary tuberculosis. Dumarest, Le Tacon and Varin (62) support those views. They describe 4 cases of ineffective artificial pneumothorax due to main bronchial stenosis, and conclude that this type of lesion contra-indicates artificial pneumothorax therapy, as does Jacobs (66) in his article on "The Unexpandable Lung".

Reichle and Frost (59) describe the development of fibrosis of a main bronchus; this is merely an extension of the process I have described in section A.

McConkey (67) has described an extreme case of progressive stenosis of the trachea where suffocation ensued and many writers have reported cases of partial stenosis of large bronchi.

Thus Epstein and Ornstein (68) report several cases in the main bronchi and one where the trachea was narrowed 1.5 cms. above the carina, while Farber (69) describes two

cases of partial main bronchial stenosis and unexpandable lungs following artificial pneumothorax.

Both Eloesser (61) and Coryllos (70) stress the importance of this form of stenosis in the larger bronchi, and the former points out that dyspnoea is the outstanding symptom, many of the cases being classed as "asthmatics" or "chronic bronchitics with bronchiectasis or emphysema."

E. Nodules of Tuberculous Granulation Tissue.

On several occasions I have seen this phenomenon. One case in particular was that of a girl with a recently induced, free left artificial pneumothorax. The upper lobe (which had minimal mottling only, before induction) became progressively collapsed and airless. A mass of tuberculous granulation tissue was revealed bronchoscopically, obstructing the lumen of the common stem to apical and sub-apical bronchi, and after two applications of 5% $Ag.NO_3$ the lobe became re-aerated. The danger in an atelectatic lobe is the secondary pneumonitis which often appears. With a free pleural surface fluid or pus formation readily occurs. This type of tuberculous bronchitis then, in the main bronchi, has a potential connection with empyema causation in relation to artificial pneumothorax therapy.

Examples of granulation tissue masses are reported by Epstein and Ornstein (68), Eloesser (61), Schonwald (71), Werner (72), Ballon (73) and many others. Clinically cough, persistently positive sputum if the mass is ulcerated, and dyspnoeic or asthmatic bouts are common. The pathological events in distal lung depend on whether the obstruction is incomplete, intermittent or complete. In the latter event atelectasis occurs as happened in my case described above. Incomplete obstruction may lead to emphysema in peripheral parts or to pneumonic episodes and bronchiectasis. Incidentally this type of lesion appears to respond best to local applications of $Ag.NO_3$, etc. (Since writing this section I have had admitted to hospital a girl with unexpandable right lung of long standing, following spontaneous

pneumothorax. An empyema is present and bronchoscopy reveals tuberculous granulation tissue masses in the distal part of the right main bronchus and protruding from the right upper lobe bronchi).

F. Endobronchial ulceration from pressure of a caseous, hilar lymph gland.

Tuberculous lymph glands of all shapes, sizes and degree of consistency are found in routine post-mortem examinations in the hilar and para-tracheal positions.

Pressure on bronchi with resulting atelectasis (the so-called epituberculosis) is a very common occurrence in relation to primary tuberculosis. I have seen two such cases where the glands softened and discharged their contents into the bronchus. When the pus was coughed up the lesions slowly healed. Several other instances of this phenomenon have come to my notice.

Cases too, have been described, where a calcareous mass has ulcerated into the trachea or a main bronchus and projected into the lumen (one type of false broncholithiasis). Zahn (74) in his review of 71 cases of broncholithiasis reported up to 1946 in the literature, states that the principal method of diagnosis is by bronchoscopy when the calculus is seen protruding into the lumen. He describes fully a case with calcified hilar glands which ulcerated into a main bronchus with subsequent expectoration of pieces of calcium. There was no parenchymal disease. The clinical signs and symptoms of protruding calcareous masses, which must be very rare, are similar to those of masses of granulation tissue. Zahn states that 45% show bronchial obstruction clinically or radiographically.

Aufses and Neuhof (75) in their description of chronic tuberculous mediastinitis and mediastinal lymph-adenitis also mention this formation of broncholiths and state that when the calcium mass is removed the ulcer heals and partial fibrous stenosis results. This interesting article mentions cases where a softened lymph node has discharged its contents into both bronchus and oesophagus

(with or without an associated traction diverticulum in the latter) and has produced a broncho- or tracheo-oesophageal fistula.

G. Stenosis of a main bronchus by pressure from without and no endobronchial ulceration.

The pressure to which I refer is mostly derived from the presence of enlarged hilar or paratracheal lymph glands. Common in relation to the primary tuberculosis of childhood and adolescence, it is also seen associated with advanced re-infection types of the disease.

Post-mortem No.19 (K.S., 25 years) showed a shrunken, slate-blue, left lung. The left upper lobe was extensively cavitated. The left lower lobe held a large vomica with necrotic walls and evil smelling contents. The lung between cavities was airless. There was a large firm mass of fibro-caseous lymph glands situated paratracheally. One such gland, the size of a pigeon's egg, was firmly attached to the anterior wall of the left, lower lobe bronchus just below its origin from the left main bronchus. The pressure of this gland had narrowed considerably the lumen of left lower lobe bronchus but not to the point of complete obstruction. There was no endobronchial ulceration. This was a permanent semi-occlusion and the mucosa of bronchi distal to the obstruction was plum-coloured, (possibly due to fibrous contraction around the gland obliterating peri-bronchial vessels). There was slight bronchiectasis in the left lower lobe bronchi and much secondary sepsis in the lung as evidenced by the particularly offensive contents of the lower vomica.

Rafferty (8) lists distal anaerobic infections as a common sequel of artificial pneumothorax therapy in the face of tracheo-bronchial tuberculosis. Montanini (76) mentions glandular compression as one of the extra-bronchial causes of stenosis and occlusion of the bronchi. It is probable that the gland mentioned above in Post-mortem No.19 at a previous more acute phase of its evolution had completely obstructed the left main bronchus, causing atelectasis of the left lower lobe, and that with the fibrosis and shrinking of the

gland the lumen regained part of its patency.

In post-mortem No.21 (J.E., 26 years) a similar condition was seen. Here there was a large firm gland on the under surface of the left main bronchus. It was stretched firmly, as if under tension, between under surface of the bronchus above and pericardium below, and had caused an inward bulging of the bronchial wall without any ulceration of the latter. This girl at one time had a left artificial pneumothorax which was complicated by atelectasis of the entire left upper lobe and ballooning of the left middle zone cavities. There were many adhesions which could not be divided. Clear, greenish, pleural fluid formed and the artificial pneumothorax was abandoned. There was partial re-expansion of the left lung but a shift to the left of heart and mediastinum closed the pleural cavity before empyema proper developed. The post-mortem showed a contracted, slate-blue lung as in post-mortem 19, with bronchiectasis in the lower lobes. The lung between the cavities was carnified, not, I am convinced because of atelectasis from complete obstruction of a bronchus, but because the semi-obstruction had caused pneumonic episodes and sputum retention. On carefully looking back at serial films of this girl the narrowing of the left main bronchus could be constantly noted before and after her artificial pneumothorax therapy.

I stated above that I am of the opinion that it must be almost impossible for a main bronchus to be so structurally weakened by disease in its wall that it would collapse or kink when an artificial pneumothorax is produced. I believe, however, that a case like post-mortem No.20, where the bronchial wall was intact, offers an ideal chance for a bronchus to be narrowed during artificial pneumothorax therapy. Bronchograms on semi-collapsed lungs show a crowding together towards the mediastinum of the lower and basal bronchi. In the case of the left main bronchus above, the solid gland beneath would act as a wedge or fulcrum,

and the tendency of the bronchus to descend would accentuate the bulge into the lumen and further limit the aeration of lung beyond and lead to sputum retention.

Brock (77) states that it is the right middle lobe bronchus which is most likely to be obstructed by enlarged, tuberculous, lymph glands, but he mentions the left upper lobe bronchus and lower lobe bronchi as also being liable to be compressed by the many glands which surround them at their origins. The two examples quoted above confirm the possibility.

Those, then, are the types of tracheo-bronchial tuberculosis (in areas able to be viewed with the bronchoscope) which I have seen either bronchoscopically or at post-mortem. When present they are undoubtedly important as adding to the risks of artificial pneumothorax therapy, particularly the empyema risk, by virtue of the cough they cause or their interference with aeration and drainage. I do not entirely subscribe to the views of Gramazio and Tosi (78) who believe that massive lobar atelectasis in the course of artificial pneumothorax therapy with adhesion section, has a favourable influence, by virtue of fibrosis, on the healing of tuberculous lesions. The all important point as we have seen (Table 8, Chapter II) is whether or not a cavity is present in the lobe.

Rafferty and Shields (79) studied 40 cases of bronchial tuberculosis diagnosed bronchoscopically and treated by artificial pneumothorax for the pulmonary lesion. There was a fatality rate of 42.5%. Atelectasis occurred in 17 cases (43%) soon after induction and re-expansion was usually impossible. An aerobic infection of distal lung was seen in 7 cases (17.5%), tuberculous empyema in 17 cases (43%) and unexpandable lung in 9 cases (23%).

Chamberlain and Gordon (80) had a 50% empyema incidence in cases of tuberculous bronchitis treated by artificial pneumothorax.

However, I think an equally, if not a more important, type of tuberculous bronchitis is that of the smaller and

distal bronchi and the bronchioles, which cannot be seen with the bronchoscope, as I shall endeavour to prove.

Tuberculous bronchitis in bronchi and bronchioles beyond bronchoscopic vision.

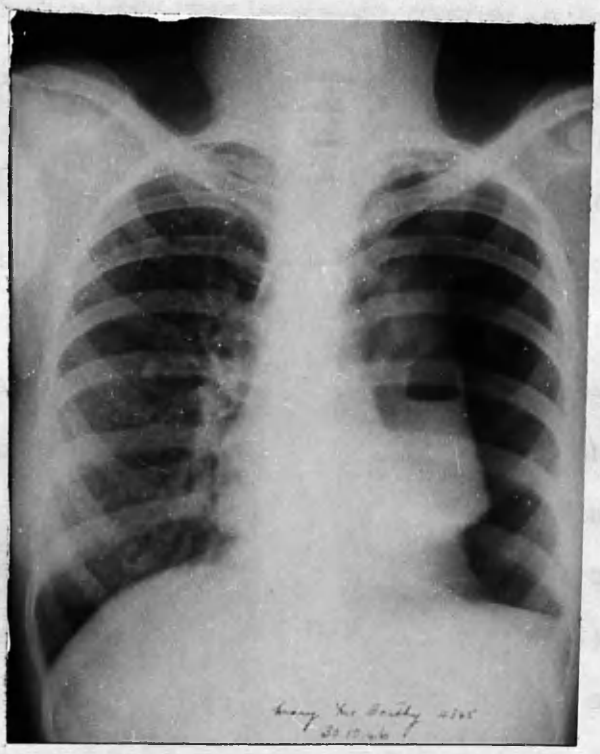
As stated previously Goldman, Brunn and Ackerman (55) found that bronchi draining cavities were affected in 100% of cases. Most writers comment on the high incidence of bronchitis in the distal bronchi without attempting to assess its significance. No less an authority than Pinner (81) states that "the significance of mucosal lesions in the smaller and smallest bronchi still needs much further study".

From personal experience and from the study of the large number of empyemata in the Baguley Sanatorium records, the commonest danger signal in artificial pneumothorax therapy is a lobe or part of a lobe, often surrounding a cavity, with "ground-glass" radiological appearances. I have repeatedly mentioned this "ground-glass" like or milky, homogeneous opacity which one often sees when an artificial pneumothorax has been induced on unsuitable cases. The same thing is seen when an artificial pneumothorax is induced, where there is obvious tuberculous broncho-pneumonia and the whole lobe or whole lung quickly assumes a milky look, often with hazy lung edges and early disaster in the form of pleural effusion which rapidly becomes purulent. But short of this gross form of pneumonia one must remember that in the vicinity of any large vomica, provided it is not too chronic, one finds small areas of the essential tuberculous pathology - aggregations of lymphocytes, plasma cells and endotheliod cells with scattered giant cells (tubercle follicles) and here and there areas of caseation in the vicinity of the vomica. I have repeatedly examined what were macroscopically healthy parts of lobes, with the microscope, and I have been impressed with the extent of the disease around a vomica.

In the subsequent discussion "atelectasis" means collapse of previously aerated lung and not "congenital airlessness" which is its literal meaning. The bronchi draining cavities are almost always affected with tuberculous

bronchitis and their pathology resembles that described previously in connection with the main bronchi which, unlike those, are visible by bronchoscope. Nearly all the bronchioles in diseased areas have pathological changes in their walls.

The question arises - "What is the underlying cause of the "ground-glass" like radiological danger signal which one often sees in unsuitable artificial pneumothorax cases?, e.g., in the attached photograph, and in cases A.T. (photograph 12), L.Q.(photograph 3), D.R. (photograph 60) and many others seen. If it is often due to peri-cavitary



atelectasis it is not so in all cases. This collapsed zone round tense vomicae is seen with true tension cavities such as are ideally chosen for Monaldi cavity drainage in the absence of artificial pneumothorax, and as continuous suction is applied to the contents of the cavity, the lumen of the latter contracts to its former volume or smaller as the peri-cavitary collapsed zone re-aerates. But we often see a milky, "ground-glass" appearance in an upper lobe when an

artificial pneumothorax is present, before an empyema develops, and yet no increase in the size of the concomitant cavity. Indeed, sometimes whole lobes are like this and no vomica visible radiologically (see Table 8). Can fibrosis cause the appearance? In unsuitable cases when an artificial pneumothorax is induced the milky appearance comes on far too quickly to be due to fibrosis, and moreover, if the physician or surgeon abandons the artificial pneumothorax quickly, in many cases the lung will re-expand showing that it had not been held in its small volume by fibrous tissue. (Fibrosis, of course, eventually supervenes, but not in the stage which we are considering).

A theory is advanced by Dufourt, Despierres and Emery (82) that atelectasis is produced in artificial pneumothorax, empyema, and after adhesion section by trauma on the pleura causing the bronchial musculature to contract reflexly. This, combined with the presence of an excess of mucoid secretion, closes bronchi which have a small or medium calibre. My subsequent histological studies show that in the bronchioles atelectasis helped by a mechanical factor and tuberculous bronchiolitis is concerned and there is no necessity to evoke the help of spasm to explain the phenomenon.

It is pertinent at this stage to consider some findings of experimental workers. Charr (83) produced artificial pneumothoraces on twelve rabbits and maintained them from 1 week to 10 months, which latter is a long period in the life of a rabbit. Gross and microscopic studies were made of both the collapsed and non-collapsed lungs. The author wanted to find if long continued artificial pneumothorax would produce pulmonary fibrosis, vascular sclerosis, bronchial occlusion or pleural thickening. Each of those headings are important in the present study of empyema. All the animals had pleural thickening. The collapsed lungs were dark red, fleshy, and smaller than normal. The important thing is that in those healthy lungs which had been collapsed alveolar collapse was most marked in the centre portions of

the lung rather than in the periphery. Also re-expansion was more complete and rapid in the peripheral zone of the lung.

Kourilsky and Anglade (84) produced atelectasis in dogs by either ligaturing a bronchus or by causing endobronchial obstruction with small pieces of rubber or laminariae. They observed that endobronchial obstruction caused immediate changes in the chest. The affected side was less mobile and the mediastinum was displaced to the obstructed side and the corresponding hemi-diaphragm elevated. If the foreign body was expelled before 20 hours there was no density in the lung parenchyma afterwards. Ligation of a bronchus was followed by the appearance on the X-rays of a diffuse, moderately dense, homogeneous shadow, partly due to a marked hyperaemia with vasodilatation. The lung could be re-aerated even after a few weeks without any sclerotic changes. In the absence of sepsis a long-standing collapse produced no marked fibrosis and the authors considered that fibrosis in long-standing atelectasis usually denotes a superimposed acute or chronic infection of the atelectatic lobe.

My histological studies of tuberculous bronchitis showed that very early in the diseased area superimposed secondary sepsis soon supervenes, and if there is any bronchiectasis present, secondary sepsis in the air tubes is always there.

When a bronchus is completely obstructed collapse, in the alveoli beyond the blockage, occurs. Coryllos (85) was the first to relate the occurrence of atelectasis to bronchial occlusion which he maintained was the main method of cavity closure. Coryllos and Birnbaum (86), Henderson and Henderson (87) and others have described fully the exact interchange of gases between the alveolar air and the blood stream during this collapse. Three gases (O_2, CO_2 and N_2) with different degrees of solubility and rates of diffusion through moist membranes, are concerned. Absorption is in

two distinct stages. Immediately after the bronchus is occluded the O_2 in the alveolar air, at a pressure of 100 mm. diffuses into venous blood where the O_2 pressure is about 40 mm. At the same time CO_2 diffuses from the blood into alveolar air, but in less amount because there is a quick equalisation of the difference of pressure of 6 mm. in the blood and in the air. Henderson and Henderson (87) state that it is a matter of seconds only in which occluded air loses O_2 to the blood stream from a pressure of 100 mm. to 40 mm. and for CO_2 to increase from 40 mm. to 46 mm. The total decrease in pressure is thus 54 mm. and the volume of gas becomes less because the lung yields to the diminished pressure within it. In yielding it brings the total gas pressure up to approximately what it was before. Pressure of air in a normal lung is 760 mm. and the pressure on the exterior of the body, transmitted through the abdominal viscera on the diaphragm, is 760 mm. Pressure of the diaphragm and of normal lung around the occluded part causes the latter to shrink until its internal pressure is 760 mm. It is usually a little less due to a small force from the other lung's elasticity. This is the end of stage 1 as described by Henderson and Henderson.

The next stage concerns the absorption of Nitrogen which is less soluble and therefore diffuses less slowly than O_2 or CO_2. Soon, as O_2 diminishes, the partial pressure of N_2 rises as it is compressed into smaller volume. It rises to 624 mm. which is reckoned to be 54 mm. higher than it was originally, and therefore 54 mm. higher than the pressure of N_2 in venous blood. (54 mm. is the sum of the falls of the partial pressures of O_2 and CO_2). Thus there exists at this stage a pressure state of 760 mm. (O_2 - 40 mm., CO_2 - 46 mm., and N_2 - 624 mm. + water vapour 50 mm.) In the blood the N_2 pressure is only 570 mm. This difference in N_2 pressures causes the N_2 to diffuse slowly into the blood and be carried off. As the total pressure in occluded air is always nearly 760 mm., some O_2 and CO_2 are also absorbed

from occluded lung to venous blood because as N_2 is absorbed the pressures of O_2 and CO_2 are left a little above that in venous blood. At the same time there is a continued absorption of O_2 from the blood in the tissues and the pressure continues to be only 706 mm. in venous blood passing through occluded lung. Therefore the process goes on and finally no gases are left for O_2 and CO_2 are always slightly and the N_2 considerably above the pressure of each in the venous blood. The mediastinum moves over and the diaphragm up, to press on the occluded lung, owing to the atmospheric pressure through the healthy lung and the abdomen. Henderson and Henderson consider that this is a rapid process as the alveolar surface and the blood flow are both large.

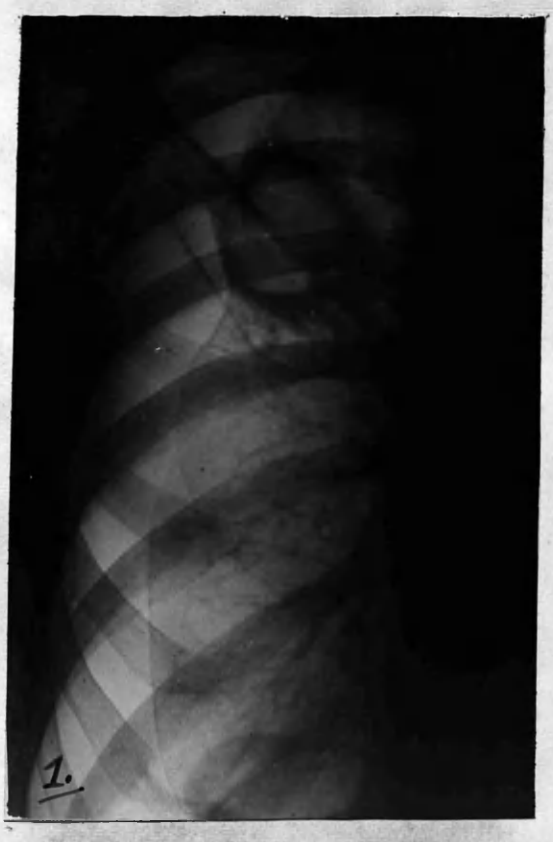
Kaunitz (88) in a general way describes those changes and for convenience divides them into (1) an early or dry, (2) a wet, and (3) a fibrotic stage. His article is on the radiographic shadows of the dry and wet stages of obstructive atelectasis. The "wet stage" he describes as being due to a transudation and accumulation of bronchial secretion in the alveoli. I mention this work because if it does occur it will aid pneumonic processes in tuberculous lung.

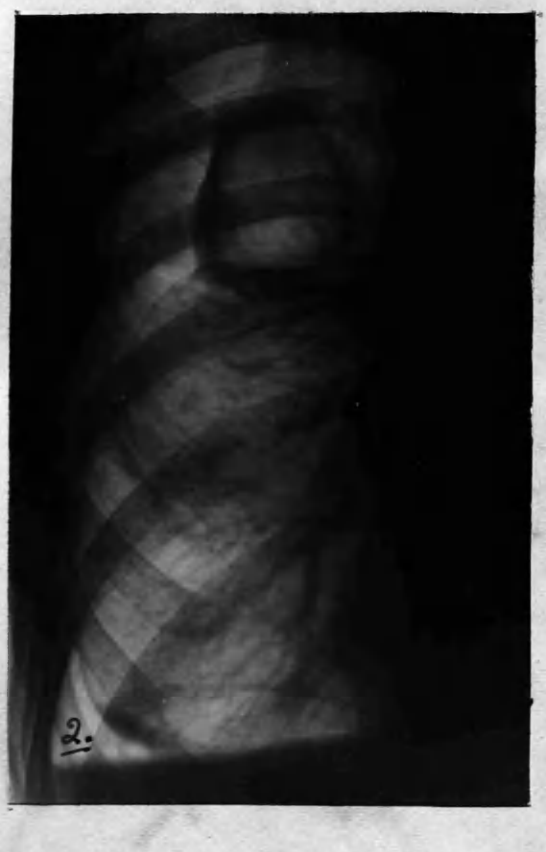
It would be wrong to omit the work of Rappaport (89) at this stage. Discussing "pulmonary atelectasis and respiratory failure" he states that the bronchial plug theory of massive collapse is so untenable that it is not even worth discussion. The author states that the most acute and fatal cases do not show any trace of a bronchial plug and are so abrupt that there can be no time for absorption of air, yet atelectasis is complete. Rappaport believes that all atelectasis depends on the production of excessive pulmonary plethora and is found to a greater or less extent in tuberculosis, asthma, emphysema, etc. The author points out that there is a good blood supply in atelectatic lung and the capillaries are engorged and that the alveoli still have some air which is mixed as froth with the pulmonary secretions. This article is not convincing and lacks the scientific

explanation of Henderson and Henderson (87) and Coryllos and Birnbaum (86). In other experiments by Coryllos and Birnbaum (90) they used intrajugular injections of iodized oil for the arterial circulation and Ringer's solution and India ink for the capillary circulation and showed that in the compressed, atelectatic and consolidated lung the circulation is progressively impaired. The degree of collapse of alveoli causes and regulates the impairment of circulation and the authors state that impairment of circulation is not due to capillary thrombosis or capillary compression by alveolar exudate as had been believed previously. They observed that the capillary circulation alone was involved but the impairment was not complete. The pulmonary arterial tree's circulation was unaffected.

I mention all those works in detail because I believe that the "ground-glass", radiological appearance - the danger signal one so often sees - is due to atelectasis. I shall now try to prove that the tuberculous bronchitis in bronchi beyond bronchoscopic vision is all important (Cuthbert and Nagley (165)). I have repeatedly seen this collapsed, milky appearance during artificial pneumothorax therapy and yet on bronchoscopy no visible cause of atelectasis.

The enclosed photograph shows a right artificial pneumothorax induced in 8.7.47, with large apical vomica and adhesions. Immediately after adhesion section (2.2.48) the cavity grew bigger (photograph 2). The ominous atelectatic area around it increased. The artificial pneumothorax was abandoned at once and air was withdrawn. Pleural gas analysis gave figures of CO_2 4.2% and O_2 1.8%, showing that there was no leak into the pleural cavity yet. It was a race against time to get the lung to re-expand before a broncho-pleural communication occurred. In this case the lung re-expanded and no fluid formed. Bronchoscopy after adhesion section showed no tuberculous bronchitis in the area within vision. Yet I have observed at post-mortem





that in all such advanced cases the draining bronchus has reddened, swollen, oedematous and irregular mucosa. At post-mortem examinations I have repeatedly exposed such draining bronchi, and by gentle pressure on the upper lobes with the flat of my hand have shown how by simulating the collapse of an artificial pneumothorax it is easy to occlude such inflamed bronchi, but that the main bronchi are much more difficult to kink, being wider and more stoutly constructed and splinted, as it were, by other hilar structures. Once the draining bronchus kinks, a tension type of cavity is apt to be formed, air entering and building up under pressure with every cough or deep breath of the patient. The surrounding lung tissue is in turn compressed and

quickly becomes atelectatic. In the case here mentioned there was a valvular effect in the draining bronchus before adhesiotomy but it was increased after that operation; air entered the cavity with coughing, etc., became trapped and was unable to escape. This is another example of a large tension cavity after artificial pneumothorax due to intermittent kinking of the draining bronchus. The latter is clearly seen in the photograph and its irregular lumen is narrowed at the site of the arrows.

This photograph from post-mortem No.6 (B.K.) shows a trachea (T) free from disease. The black arrows (B) depict cavity walls and the arrow (A) points to the draining bronchus which in this case is the sub-apical bronchus of the right upper lobe.

It is clearly seen how the mucosa is irregular and swollen. Just proximal to the cavity the draining bronchus has a partial fibrous structure 'S' which was very marked when the bronchus was slit open with fine scissors.

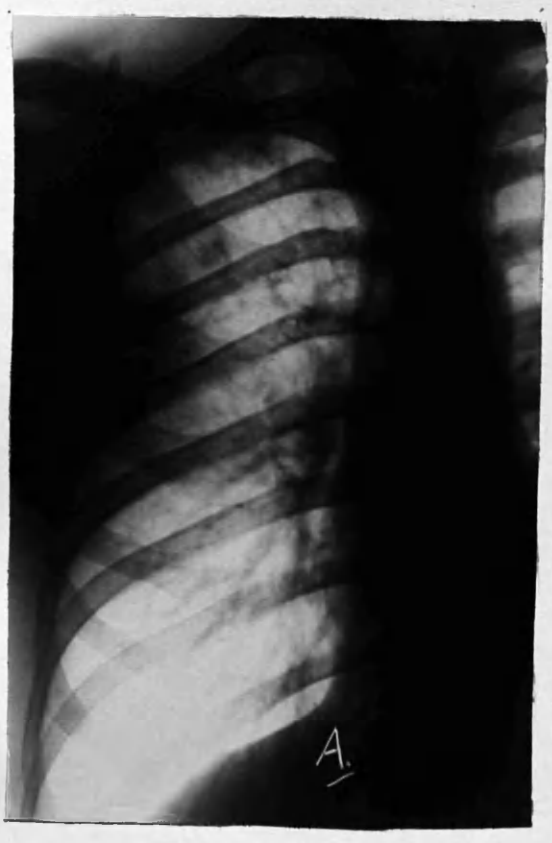
This case is reproduced to illustrate the points I have been discussing with regard to the ease with which a tension type of cavity forms when an artificial pneumothorax is induced in unsuitable cases.

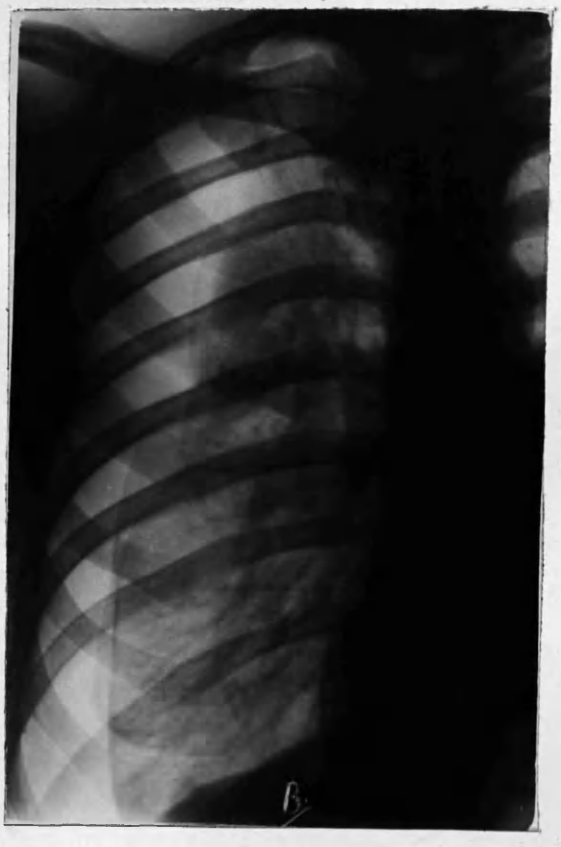
In healthy lung peri-cavitary atelectasis would probably be of little import and the lung would remain strong. As I have mentioned above the tuberculous process invades the tissues widely around all such cavities and the small bronchi and bronchioles in the vicinity are all more or less involved and easily occluded by any abnormal pressure. Those bronchioles and bronchi must be distinguished from the cavity draining bronchus which is also affected. Even if there is no tension cavity with peri-cavitary atelectasis a whole lobe can go milky and opaque by virtue of its having scattered foci of caseous change and a large number of small bronchi and bronchioles affected. The nature of the tuberculous involvement in those small air passages makes them very susceptible to obstruction by means of pressure on the lung surface which will disturb their normal alignment and bend them in any way.

I have for convenience described below three changes in the closure of the smallest air passages, for I believe that this is the fundamental factor directly or indirectly in causing disasters in most of the bad artificial pneumothoraces in the R.B.2 class of case with which we are chiefly concerned.

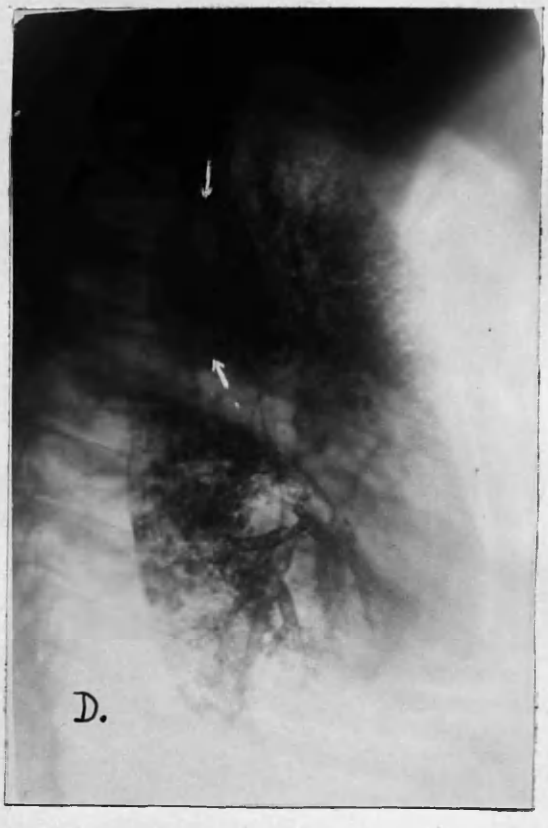
Here is another example of the role of the smallest bronchi in causing atelectasis.

C.H. on 4.12.47 had an X-ray appearance as in figure 'A'. The left lung field was clear. The right upper lobe had several small areas of mottling but no definite cavitation. An artificial pneumothorax was induced and a film (B) shows a semi-opaque right upper lobe adherent apico-mediastinally.


A.


B.


C.

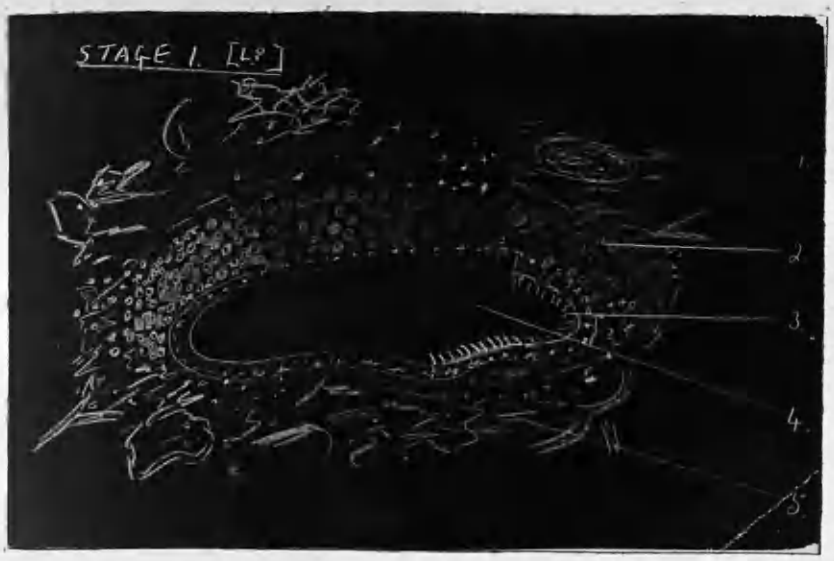

D.

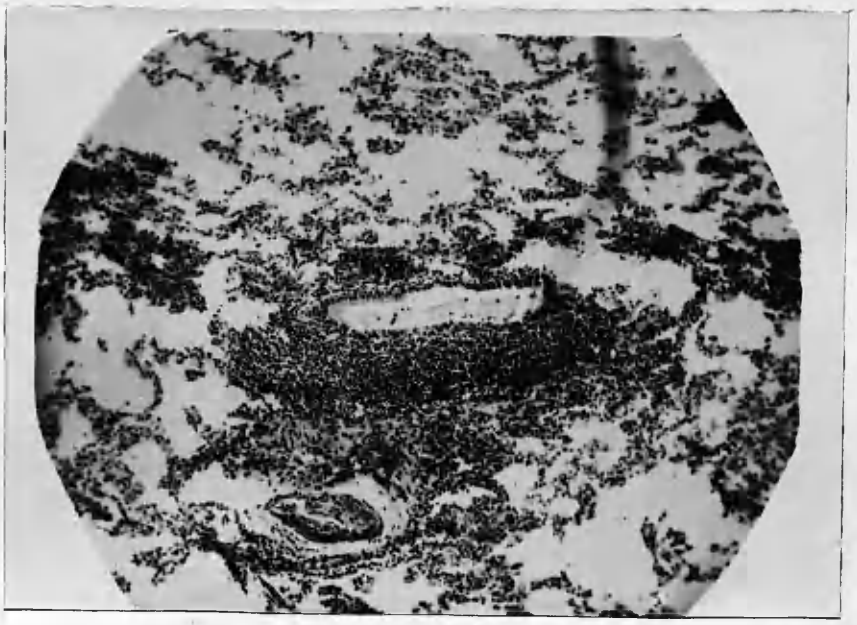
The right lower lobe is free and aerated. At this time bronchoscopic examination was made and all bronchi within vision appeared perfectly normal. On 6.4.48 a bronchogram was obtained and the P.A. view (C) shows filling of the right lower lobe bronchi and a large number of right upper lobe alveoli filled. A lateral view, however, (figure D) shows beyond doubt that the opaque area is a posterior segment supplied by the sub-apical bronchus whose finer branches fail to fill with the lipiodol (which was warmed). The arrows point to the obstructed, finer bronchi. I consider that failure of alveoli to fill with warmed lipiodol is significant and I have obtained similar results on other like cases with opaque "ground-glass" lobes during artificial pneumothorax therapy.

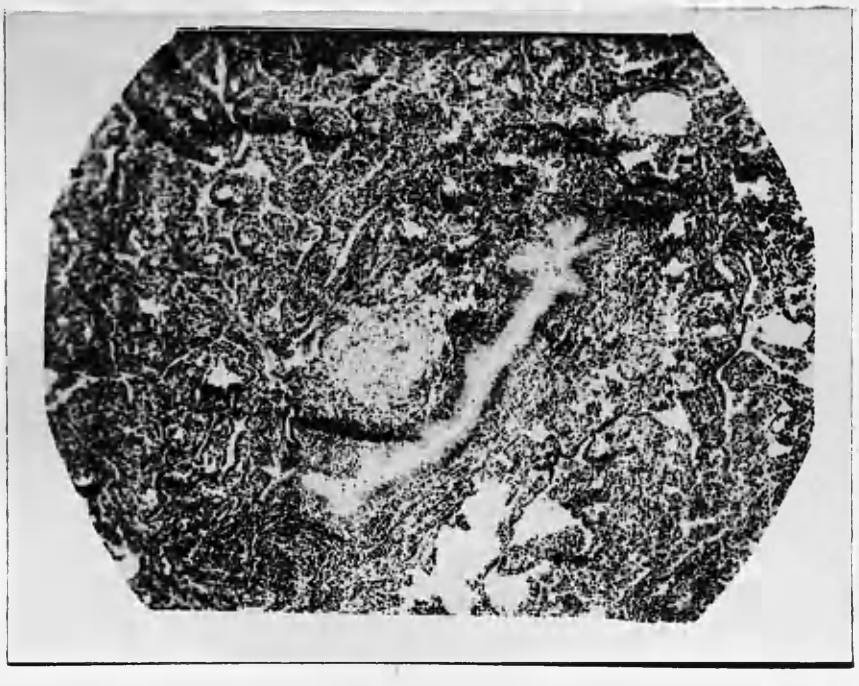
By studying X-ray films taken just before death and then examining the areas corresponding to the mottling, in the less involved part of the lung, on the post-mortem specimen, I have come to the conclusion that areas such as are shown in the right upper lobe of the above case (figure A) are patches of caseous broncho-pneumonia. In and around them and in the vicinity of the caseous wall of a vomica one observes, in histological specimens, that the walls of the bronchioles and smaller bronchi (4th degree) have a tuberculous infiltration which I have classified into 3 stages according to the changes seen.

Stage 1. (See photograph diagram). The submucosal tissues become infiltrated with round cells, lymphocytes, plasma cells, occasional polymorphonuclear leucocytes and endotheliod cells. This cell mass is liable to collect as a button-like projection or a "cuff" at one area (No.2 in diagram). The ciliae over the cell aggregate soon vanish but remain present where the submucous tissues stay normal (No.3). The lumen of the fine bronchus or bronchiole retains its normal contour at first, being only slightly flattened from the pressure of the round cell mass. It contains debris and pus cells indicating the presence of secondary infection.

65A1.







Examined Lungs (P.M. 24).

The artery which runs alongside the bronchiole shows early endarteritis (No.1) in the face of this cellular infiltration which is presumably the reaction of the tissues to the spread of tubercle bacilli in a periphero-mesial direction along the lymphatics which run with the fine bronchi or bronchioles. This endarteritis will help to devitalise the parts beyond as well as mitigating against "staining" should the area soften and the vessel be eroded.

No 5 represents an area where there are a few round cells but no gross gathering as yet. (This photograph is of a bronchiole as there is no cartilage evident).

Stage 2. Here the contour of the fine bronchus is altered by cellular pressure (No.1) and all ciliae have gone. Endarteritis is present (No.2); the lumen of the bronchus has more debris and pus cells which, with loss of ciliary action, contribute to the obstruction. No.4 depicts an increase of round cells and endotheliod cells in the alveolar walls and collapse of alveoli, with fibrin in many of them. (The diagram was drawn from a bronchiole with stage 2 while the photograph is from another slide of a fine bronchus with stage 2. Cartilage is present).

Stage 3. is that of final closure. The bronchiole or fine bronchus is flattened and the alveoli around collapsed, the whole area being invaded with round and endotheliod cells to a greater extent than in the previous 2 stages. The approximation of alveolar walls makes the spread of a caseous process through them easy and as secondary sepsis is at hand, the area softens and the outlines of collapsed bronchi are lost and merge into a part, densely packed with round cells and pus cells. Obliteration of the accompanying artery by endarteritis is prominent in this photograph.

I am convinced from a close clinical and post-mortem study that the "ground-glass" appearance which I have so often mentioned is due to the above changes. The all important point is that in so many of the cases the diseased bronchi are outwith bronchoscopic vision.

I agree with Pinner's views that uncomplicated, reversible atelectasis, does not occur in cases of pulmonary tuberculosis which are seen at autopsy. The above changes occur in the areas be they large or small.

However, the pericavitary "ground-glass" radiological appearance which develops rapidly round a tension vomica in artificial pneumothorax therapy may, as I have seen, just as quickly resolve, leaving no radiological traces, if steps are taken to abandon the artificial pneumothorax at once and to withdraw air. The explanation is probably that the bronchioles or finer bronchi concerned merely exhibit stage 1 of the above classification. When kinked they collapse, but release of the pressure on them, when the artificial pneumothorax quickly resolves, allows them to become patent again. Early pneumonitis in the collapsed area, short of caseation, will resolve, leaving no gross, permanent effects. So much may be said for brief compression of alveoli.

Longer compression produces a chronic inflammation in the collapsed lung with organisation or carnification. Post-mortems No.24 and 21 are excellent examples of this, especially the former where the right lung was one firm fleshy mass. In some cases this is good from the point of view of healing of the tuberculous lesion (although I am not so sure if possible tuberculous bronchiectasis, which often supervenes a few years later, is to be desired).

The photograph (an L.P. view) of carnified lung from post-mortem No.24, on separate sheet,* shows collapsed alveoli with caseous areas "C" at intervals. (High power examination reveals areas of granulation tissue with new capillaries and leashes of fibroblasts spreading into the collapsed alveoli.) The upper part of this photograph shows fibrous tissue and an area of pigment is evident.

If in the collapsed area a caseous focus is present this, in many cases, readily spreads, the part softening and, helped by a tension vomica, the area may burst into the artificial pneumothorax space, causing empyema

and broncho-pleural fistula. This is especially liable to happen after devitalisation of tissues by a cautery in adhesion section. I have seen several clinical cases where the point of rupture could be seen on the lung edge in an X-ray film.

Of course, in the presence of an artificial pneumothorax, a caseous area in a collapsed lobe is dangerous even without a cavity present. A tension cavity in addition, however, increases the risk of empyema enormously. (See figures in analysis of 78 empyema cases - page 34). Unfortunately when we look at the ominous "ground-glass" appearance round a tension cavity when an artificial pneumothorax is present, we cannot, from X-ray appearances, say whether the picture is due to collapse and pneumonitis or collapse, pneumonitis and softening caseous areas. My experience is that it is better to think in terms of the latter eventuality, and, if speedy thoracoscopy and adhesion section fail to get rid of the cavity, the artificial pneumothorax should be abandoned at once and the lung encouraged to expand by withdrawal of air.

When we know the pathology, therefore, it is easy to visualise the chain of events in a diseased lobe on induction of an artificial pneumothorax.

Cavity with surrounding small patches of tuberculous broncho-pneumonia.

↓

Artificial pneumothorax induced.

↓

Diseased draining bronchus, structurally weakened, kinks. (It blocks easily on the least alteration of alignment because ciliary loss causes retention of secretions and its walls are already swollen and oedematous. Adhesion section often helps gravity to kink the draining bronchi).

↓

Tension cavity liable to develop.

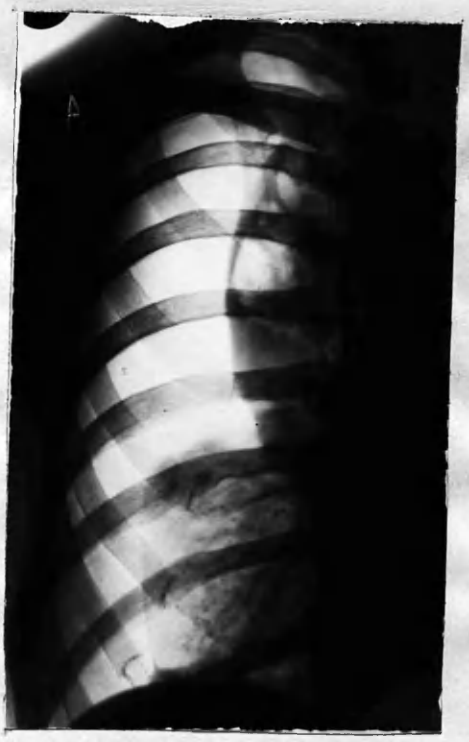
↓

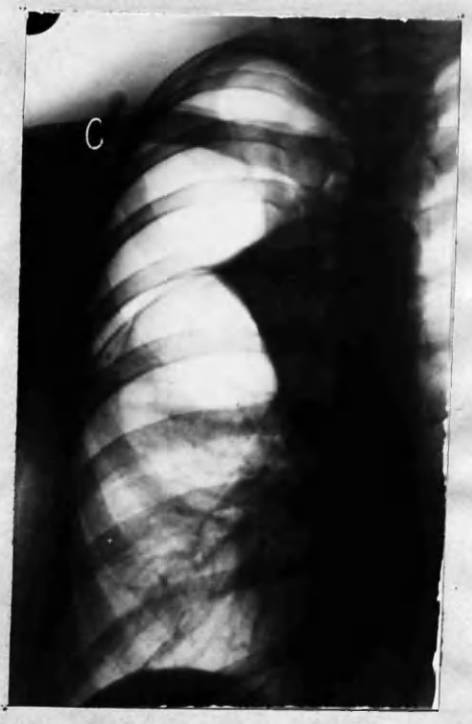
Alteration of alignment of diseased bronchioles and 4th degree bronchi in the vicinity of patches of tuberculous broncho-pneumonia. ⟶

→Early collapse and infection of adjacent alveoli producing "ground-glass" appearance in the whole lobe or segment of lobe on the skiagram, with its resultant dangers as regards empyema formation as described above.

Thus we see that the diseased bronchioles and small bronchi run two chances of being obstructed by alteration of alignment - (a) when the artificial pneumothorax is induced or when supporting adhesions are cut; if they escape at this stage, (b) when pressed upon from another direction when a "tension cavity" forms. This explains the "ground-glass" appearance which forms in a lobe where no cavity is visible as well as that following the ballooning of a vomica.

As mentioned previously, I have found the prognosis to be much worse, as regards empyema formation, where there is a milky looking atelectatic lobe containing a vomica than where such a lobe has no visible cavity. Occasionally a large tension cavity in a milky lobe may quickly disappear. This is probably because the intermittent, valvular mechanism in the draining bronchus ceases to work and a complete kinking or permanent block ensues with subsequent absorption of the air in the cavity. The prognosis is then increased for the better, as in this case, e.g. (B.W.) When a collapsed right upper lobe with cavity existed fluid formed (Group IV, Table 8, page 34)(A). Shortly afterwards something happened to the draining bronchus and the cavity closed (Group III, Table 8). (B) The artificial pneumothorax then lasted 4 years in spite of apical adhesions and when the lung was finally allowed to re-expand (C) the lower and middle lobes filled the pleural space and pushed the airless upper lobe against the mediastinum - a phenomenon occasionally seen in those cases. Unfortunately there is no way of telling which case will take this course, and I believe that there is no justification for hanging on in the slender hope that the rare event of spontaneous closure of the cavity will occur. The previous figures show the small risk of success and the grave risk of fluid formation and empyema.





Post-mortem Findings:

All post-mortem examinations were done as soon after death as possible to prevent the secretions in the bronchi softening the mucosa.

The chest was opened and sternum removed with the medial third of each clavicle. This enabled the apices of the lungs to be freed from chest wall. Where adhesions were of any extent and not friable, the lungs were freed in the endothoracic fascial plane and removed with parietal pleurae. The separation was made down to the diaphragm which was cut around at its attachment to the chest wall as advocated by Miller (91). The trachea was divided immediately below the larynx and lungs, pleurae, heart and trachea removed in toto by dissection from the vertebral column. This removal made for better study of bronchi and was superior to the usual method of individual lung removal where each hilus is severed. The trachea and main bronchi were then opened with scissors under a running stream of cold water. The secondary and distal bronchi were explored by sharp pointed, long-bladed, thin scissors used for that purpose only.

Bronchi Beyond Bronchoscopic Vision: (28 post-mortem examinations).

TABLE 9.	Numbers of cases.	Percentage of Total.
Inflamed, irregular and swollen mucosa.	22	78.6%
Partial stenosis:	1	3.6%
Complete stenosis:	-	-

Trachea and Bronchi Within Bronchoscopic Vision:

TABLE 10.	Numbers of cases.	Percentage of Total.
Generalised bronchitis:	5	17.9%
Bronchitis with Ulceration:	9	32.1%
Partial fibrous stenosis:	1	3.6%
Complete fibrous stenosis:	1	3.6%
Masses of tuberculous granulation tissue:	-	-
Endobronchial ulceration from caseous gland outside:	-	-
Partial stenosis of bronchus from pressure from outside:	2	7.2%

Cases where tuberculous bronchitis was present at the furthest limits of bronchoscopy only - 5, 7, 11, 16.

For reference purposes the findings of Sweany and Behm (58), relating to tuberculosis of the trachea and major bronchi, in a post-mortem study of 667 cases, are appended.

Nature of involvement:-

Diffuse superficial infiltration:	12 cases	(3.2%).
Diffuse and deep infiltration and infiltration and ulceration:	85 "	(22.8%).
Circumscribed ulceration:	265 "	(70.3%).
Fibroid stenosis:	5 "	(1.3%).
Perforation:	6 "	(1.6%).
Caseous bronchitis:	4 "	(1.0%).
TOTAL:-	377.	

Microscopic study of the remaining 290 grossly negative cases gave an estimated figure of 37.5% positive, thus increasing positive findings to 72.0%. It must be noted

that cases of laryngeal involvement are counted. I have not included those in my series.

Percentage of positive findings by microscopic methods in those main bronchi which are apparently normal.

Fourteen cases had, on macroscopic inspection, apparently normal tracheae and main bronchi (i.e., post-mortems 1, 2, 3, 8, 9, 10, 11, 12, 13, 14, 16, 23, 24 and 26). When histological examination of the main bronchi was made it was seen that in 5 cases there was tuberculous involvement of the mucosa (i.e., cases 3, 9, 11, 16 and 23). This raises the percentage of cases having tuberculosis of the trachea or main bronchi to approximately 68%. The matter is important where operative removal of lungs or lobes is being contemplated, as it indicates with what frequency diseased areas may be cut across with correspondingly increased risk of bronchial stump ulceration and subsequent broncho-pleural fistulae.

It was noted that many of the main bronchi, while they did not exhibit signs of tuberculous bronchitis, nevertheless showed evidence of non-specific, chronic bronchitis.

Treatment:

At the conclusion of this chapter I will briefly indicate some of the recent methods of treatment advocated for tracheo-bronchial tuberculosis.

Vinson and Habein (92) in 1928 report a good result from deep X-ray therapy of the area following the removal of ulcerating tissue.

Ballon (93) and Werner (94) in 1935 both have removed granulation tissue and dilated the bronchi at intervals. The former has applied 2% to 5% $AgNO_3$ locally after removing granulation tissue.

Epstein and Ornstein (68) advocate the use of a cautery for large obstructing masses of granulation tissue. Small superficial ulcers, they think, should be left alone as they tend to heal when the parenchymal lesion is controlled.

For the stenotic type of tuberculous bronchitis they advocate thoracoplasty and they prescribe no active treatment for 'caseous necrosis' cases.

Rafferty (8) in 1945 stresses the importance of general, as well as local treatment for the condition. General treatment such as humid atmospheres, steam inhalations and expectorants lessens the violent coughing and decreases the risk of bronchogenic spread of tuberculosis. Locally he mentions the application of 30% $AgNO_3$ to the granulations. This decreases the amount of stenosis, associated with healing, he states. Frequent bronchoscopic aspirations, to get rid of secretions, are advised.

Again in 1945 Acuna (53) advises the application of silver nitrate solution to lesions and the dilatation of strictures.

In 1946 Secretan and Zuidema (95) described their treatment of tracheal lesions with 10 - 30% of $AgNO_3$ and of bronchial lesions by diathermy. They say that it helps long-standing bronchial stenosis to dilate it.

In 1947 several articles appeared among which is one by Dufourt, Mounier-Kuhn and Despierres (96) who gave 200 mg. of vitamin D2 with calcium gluconate over a period of three months in 9 cases of bronchial tuberculosis. Rapid healing of half the lesions occurred while the rest were not affected.

Lemoine (97), dealing with the bronchiectasis secondary to stenosing bronchial tuberculosis, advocated bronchial dilation by bougies and applications of Adrenalin. Intramuscular penicillin was given too. Failure to dilate the strictures was an indication for surgical resection of the lobe. This last statement is important as it is the opposite of the treatment advised by Epstein and Ornstein, above, who say that no treatment is necessary for stenosis of a single lobe bronchus. I have seen one case of bronchiectasis, with persistently tubercle bacilli positive sputum, following partial stenosis of right middle lobe bronchus, where lobectomy was done. If left alone there is invariably

a bronchogenic spread to the same or opposite lung as the pathological changes in true bronchiectasis are of a chronic nature.

Halle (98) states that local treatment of tuberculous tracheal and bronchial lesions should be combined with general treatment. Locally he believes in the bronchoscopic aspiration of secretions, cauterization of ulcers and granulations with diathermy or $AgNO_3$, and the application of Adrenalin to swollen mucous membranes. Fourteen of his twenty-one cases reported have had intra-tracheal instillation of penicillin several times a week. This, he believes, lessens secondary infection and thus the bronchial secretions, and he has observed by the bronchoscope superior results with this than with $AgNO_3$ alone.

Peremans and Mottard (99) use locally a solution of 1/1,000 Adrenalin, 1% $AgNO_3$ or 30% Gomenol for simple ulceration or stenosing lesions. True fibrous stenosis they dilate with bougies. (The use of Adrenalin locally may be justified if there is any truth in the statements of Oatway, Gale and Mowry (100) that the most serious bronchial lesions occur predominantly in women patients with clinical evidences of allergy, and that there is an apparent direct relationship between a personal history of allergy and the presence of tuberculous tracheo-bronchitis.).

Finally, Brewer and Bogen (101), summarising the work done to November 1947 on Streptomycin in tuberculous tracheo-bronchitis, state that there are conflicting reports as to its efficacy. The best results appear to be achieved by large intramuscular doses and aerosol inhalations.

This excursion into the realms of tracheo-bronchial tuberculosis has revealed the importance of the condition in artificial pneumothorax therapy. Disease in the bronchioles and finer bronchi in moderately advanced cases is very frequent, and has hitherto been somewhat neglected in favour of the lesions in the main bronchi.

The disease in the smallest bronchi and cavity draining bronchus is often the starting factor in a chain of events which culminates in the ominous "ground-glass" like, radiological opacity which one commonly sees around cavities in artificial pneumothorax therapy before the onset of fluid or empyema.

To study the finest bronchi, histological examination is necessary as there is a limit to the extent to which one can follow the course of a bronchus with fine scissors.

CHAPTER IV.

THE ROLE OF PLEURAL ADHESIONS IN EMPYEMA ONSET.

Historical: James Carson of Liverpool is quoted by Keers and Rigden (102) as having been the first to suggest the introduction of air into the pleural cavity to overcome the lung's elasticity and collapse pleural cavities. Two attempts by him to produce an artificial pneumothorax failed. The same authors state that Carlo Forlanini of Milan induced the first artificial pneumothorax in 1888. Alexander (103) states that Forlanini first proposed the introduction of artificial pneumothorax in 1882 and published his first reports in 1894. I think it is a fair statement to say that the incidence of tuberculous empyema rose from then onwards. Forlanini at the very start, in 1894, is credited with saying that pleural adhesions limit the volume of the artificial pneumothorax and therefore its curative value.

Bayliss (36) states that Cayley, in 1885, reported the case of a tuberculous patient in whom he had induced artificial pneumothorax by an open incision of the chest wall to control haemoptysis.

Potain in 1888 published reports of air replacement of fluids in spontaneous pneumothoraces due to pulmonary tuberculosis. This worker appears to be the first to have used a monometer to measure intrapleural pressures.

Bayliss further records that J.B. Murphy of Chicago first urged the use of radiology to control the degree of pulmonary collapse in 1898 and Saugman in 1904 made routine use of the manometer in artificial pneumothorax therapy.

Newton (104) quotes Freidrick of Marburg as having devised the operation of open intrapleural pneumolysis in 1908 but it fell from favour owing to its high incidence of complications.

Ascoli in 1912 showed that the hitherto complete collapse of the lung was not necessary in order to secure a satisfactory result, and the following year (1913) Hans Jacoboeus of Stockholm devised the operation of closed intrapleural pneumonolysis.

Those workers paved the way for modern artificial pneumothorax therapy.

Formation of adhesions:

As Fishberg (45) has stated, tuberculosis of the pleura is almost as common as tuberculosis of the lungs. Tuberculosis spreads by direct extension from pulmonary parenchyma to visceral pleura by blood or by lymph channels. The blood supply of the pleura is derived from two sources. Parietal pleura is supplied from intercostal, phrenic, internal mammary, mediastinal and bronchial arteries. The visceral pleura derives its supply from a capillary network which lies beneath it and is supplied by branches of the pulmonary and bronchial arteries.

Miller (105) has described the lymphatic systems in the pleurae. Small lymph glands and lymphatics are richest on the membrane covering the interlobar fissures, although present on the entire visceral surface.

A. Lymphatics from parietal pleura pass to small intercostal glands situated near the heads of the ribs and indirectly to the axillary glands through their connection with the lymphatics of the 4th and 5th intercostal spaces. (Lately, during 4 thoracoplasty operations, I have obtained samples of the small lymph glands one finds in the inter-

costal spaces. In two cases histological examination revealed tuberculous involvement. Those glands were from the anterior and posterior ends of the 2nd. intercostal space, respectively. The glands in the other two cases were not apparently involved. Each of those cases had firmly adherent pleural spaces and presumably the infection had travelled by the routes mentioned above. The importance of not crushing such foci will be realised. Similarly in post-mortem No.17, where the left pleural cavity was adherent in all aspects, there was a small gland at the anterior end of the 2nd. left interspace which showed active, proliferating caseous tuberculosis).

B. There is also an anastomosis between the lymph vessels of the pleura and those of the peritoneum. In the thorax the diaphragmatic lymphatics drain into the sternal glands in front and those of the posterior mediastinum behind. As Coope (106) points out, the extension of inflammation along the trunks which connect pleurae and abdomen is almost invariably in the direction of abdomen to thorax. (This is often observed in the case of subdiaphragmatic abscesses following "leaking" peptic ulcers or appendicitis or amoebic hepatitis say, where, if unspotted, a pleural effusion or lung abscess may occur. On the other hand, I have seen tuberculous empyemas, secondarily infected, perched on the summits of paralysed hemi-diaphragms which had been elevated by a pneumoperitoneum, and yet no signs of abdominal effusion or abscess following in the free air space under the diaphragm. (See case A.W. No.20,525). This, to my mind, is one of the best examples of the flow of lymph from abdomen to pleura and not in the opposite direction. However, with very advanced pulmonary tuberculosis it is possible to get some flow of bacilli from pleurae to under surface of diaphragm, e.g., in post-mortem No.17 the spleen was adherent to the liver, and in the tissues and adhesions between the lower surface of the left diaphragm and the left lobe of the liver were several pea-size chalky nodules. No other abdominal lymphatic involvement was seen.

In post-mortem No.24, where there was a right tuberculous and mixed infection empyema, there was a spread of tuberculous nodules to the peritoneum and ligaments on the under surface of the right hemi-diaphragm).

C. Lymphatics in pulmonary parenchyma drain into the bronchial lymph glands. (Examples of this are commonly seen in connection with the formation of the primary pulmonary complex). Miller (105) states that visceral pleura has a set of lymphatics entirely distinct from that of the lung substance proper, which are stated to appear in the injected specimen as a network of large lymphatic vessels in the meshes of which are numerous smaller vessels. This pleural system has an independent drainage of its own directly to hilar glands. The diaphragmatic pleura of the lower lobes, however, drains via lymph vessels in the pulmonary ligament into pre-aortic nodes. Lymph flows from pleura to hilum and is prevented from going in the reverse direction by valves which point towards the hilum.

Short lymphatic trunks make a connection between the pulmonary parenchyma and pleural lymph systems but the valves in those trunks all point towards the pleura, thus ensuring that the direction of lymph flow is from lung tissue towards the pleura. It is, therefore, evident how tubercle bacilli can reach the visceral pleura from pulmonary parenchyma.

When treating patients with the reinfection type of pulmonary tuberculosis one is constantly seeing cases who have pain on deep breathing at one area of the chest wall, slight rises of Blood Sedimentation Rate, pyrexia, and a pleural rub audible with the stethoscope over the site of pain. This dry pleurisy represents the reaction of the pleura to the spread towards it of underlying parenchymal disease. The inflammatory episode causes thickening of the visceral pleura and the formation of plastic lymph which glues the visceral to parietal pleura. Later the bridge organises with fibrous tissue. Occasionally the formation of a pleural

effusion separates the pleural surfaces and relieves the pain. In most cases slight effusions absorb if the patient takes sufficient rest. Those episodes leave their mark in the form of radiological evidences of obliteration of the costo-phrenic angle, peaking of the diaphragm or prominence of the interlobar septum say. In gross cases the extensive pleural fibrosis pulls the heart and mediastinum over and may even cause spinal deformity and tiling of the ribs. The pleurisy and subsequent adhesion of pleural layers are nature's attempts to prevent caseous material from invading the pleural space and causing spontaneous pneumothorax with its dire sequelae of tuberculous empyema, broncho-pleural fistula, etc. Naturally, adhesions will be most commonly found where the underlying pulmonary disease is most prevalent. Thus, apico-mediastinal adhesions are commonest, especially in the paravertebral gutter, and then in frequency come the interlobar surfaces and the diaphragmatic surfaces. Those latter from their dependent position may be caused by former fluid over the diaphragm (i.e., gravity cause).

Brock (77) discussing lung abscesses, points out that the two commonest sites for their formation are the axillary and posterior sub-apical parts of the upper lobes and the apical parts of the lower lobes and he believes that position, when lying down, favours the occurrence of abscess in those areas. A patient on his side will inhale material readily into the axillary branches of the upper lobe bronchi, on his back the apical branch to lower lobe is the first bronchus encountered by material flowing along the posterior wall.

Lipiodol has been trickled into the noses of men in a drunken sleep and X-rays subsequently taken confirmed the above views. Some such postural factor in the bronchogenic spread of tuberculosis may likewise confirm the preponderance of tuberculous apical lesions, especially in the axillary and posterior aspects of the upper lobes and the consequent common finding of pleural adhesions there.

The practical importance of adhesions is in artificial pneumothorax therapy. In some cases induction is impossible and all degress of adhesions are found down to a space completely devoid of any adhesion. As a rule the extent of adhesions is directly proportional to the extent of underlying parenchymal disease.

Analysis of post-mortem records in Baguley Sanatorium reveals the numbers of cases with adhesions. Those post-mortems were carefully done.

1. Period 19.2.28 to 24.9.35.

159 post-mortems done on cases of pulmonary tuberculosis.

A. Adhesions of degree of severity from apical bands to obliterative pleuritis,

right sided - present in 116 cases.

left " - " " 113 "

B. Cases with serous fluid with or without pleural adhesions - 25 cases. Those included 3 cases of generalised dropsy with both sides of chest involved, 1 case of Ayerza's syndrome and tuberculosis, and 1 case with an artificial pneumothorax. Therefore, 16 pleural effusions occurred, apart from generalised dropsy, artificial pneumothorax, and Ayerza's syndrome.

C. Spontaneous pneumothoraces - total 11.
Of those 7 had associated empyema and 4 had no empyema (1 of them had clear fluid).

D. Empyemas, apart from spontaneous pneumothoraces, were 6. Two of them were associated with artificial pneumothorax and 4 were not associated with artificial pneumothorax or known spontaneous pneumothorax. It is very significant that those four lungs concerned had caseating lesions in them, i.e.,

(a) Scattered caseating tubercles in the lung. A caseating tubercle ruptured at the base into the pleural cavity (Post-mortem No.18).

(b) Scattered caseating tubercles in the lung up to the size of an almond (Post-mortem No.50).

(c) Right lung - disseminated caseating tubercles (No.105).

(d) Left lung oedematous. Caseation in middle zone. Cavitation at apex and dense adhesions over the cavity area (No.149).

Next, 54 post-mortem findings, done between 19.1.36 and 6.7.46, were analysed to see if an artificial pneumothorax could have been induced. In the 108 lungs examined the adhesions were classed as apical or localised (and therefore artificial pneumothorax could have been induced) or generalised (where from the post-mortem findings no artificial pneumothorax would have been possible.

	Localised adhesions (artificial pneumothorax possible).	Generalised adhesions.	TOTAL.
Right side.	18	21	39.
Left "	15	21	36.

One of the cases of right-sided generalised adhesions was complicated by a dense calcified plaque on the pleural surface. Non-purulent effusions of varying amount were present in 17 cases. (In 3 of those cases miliary tubercles or nodules were noted on the pleural surface.) There was one fatal spontaneous pneumothorax with no fluid.

In this series there were 6 empyemas, four of which were associated with artificial pneumothorax therapy (post-mortem Nos.39, 47, 60 and 63). One was an old, chronic empyema with broncho-pleural fistula and metastatic bone formation in the empyema wall (post-mortem No.22). One empyema (cause not known) had associated liver and kidney amyloid disease (post-mortem No.41).

Gloyne (107) found, in a series of cases, pleural sac adhesions at autopsy in the following sites in order of frequency:-

(a) upper lobe, (b) interlobar fissure, (c) pericardium, (d) lower lobe, (e) diaphragm, and (f) mediastinal pleura.

He studied the formation of adhesions, in guinea pigs, histologically, and described the following stages in their formation,

(a) pleural exudation with endothelial proliferation,

(b) the formation of fibrin,

(c) granulation tissue production,

(d) the development of new blood capillaries,

(e) the formation of connective tissue.

Three factors characteristic of a tuberculous lesion were added to these:-

(f) the presence of tubercle bacilli, chiefly in granulation tissue areas,

(g) the formation of giant cells,

(h) the production of caseation.

Gloyne found elastic tissue in adhesions but this was not constant in occurrence. He thought elastic fibres would be derived from subpleural elastic layers.

My own small series of post-mortem examinations recorded here was done with two objects in view, both bearing on the production of empyema, i.e.,

(a) the study of tuberculous bronchitis,

(b) the condition of the pleural surfaces, endo-thoracic fascia, sub-pleural parenchyma and adhesions, etc.

Those autopsies were done with great care and many histological examinations made. I have made many more autopsies on tuberculous cases but did not pay attention to detail as in this series.

Adhesions were predominantly apical and, as a rule, in the posterior or apico-mediastinal positions.

Table II shows the numbers of cases where an artificial pneumothorax could have been induced (31 cases). It would have been impossible to induce an artificial pneumothorax due to dense or total adhesions in 17 cases. In one case (post-mortem 9) the right lung was absent following pneumonectomy; in another case (post-mortem 24) an empyema was present following adhesion section.

TABLE 11.	Right side.	Left side.
Possible artificial pneumothoraces.	15	11
No artificial pneumothorax possible.	7	10
Lung completely free.	1	4
Empyema following adhesion section.	1	-
Empyema following pneumonectomy.	1	-
TOTALS:-	25	25

Alexander (103) states that diffuse adhesions prevent induction in about 20% of attempts at artificial pneumothorax and that in approximately 42% to 50% of cases, adhesions allow only partial artificial pneumothorax, insufficient to allow complete healing of the lesion.

When an artificial pneumothorax is induced any lateral adhesions become elongated owing to the sliding movement of the lung on the lateral chest wall in breathing. The material drawn into the adhesion when it elongates is derived from the endothoracic fascia and parietal pleura rather than from lung tissue or visceral pleura. I have made histological studies of this endothoracic fascia. When healthy, it is a delicate layer of loose areolar tissue. This is the layer through which I removed the lungs and both pleural coverings at autopsy. This layer is also utilised in Semb's apicolysis at thoracoplasty operations and in the operation of extrapleural pneumothorax. In the face of extensive pleural adhesions or inflammation, this endothoracic fascia becomes inflamed and organised with fibrous tissue, and removal of lungs even through it may be difficult at autopsy, especially at the apices. As mentioned previously the lymphatics here communicate with the intercostal glands near the heads of the ribs and indirectly to axillary glands through their connection with the 4th and 5th intercostal spaces.

My researches into this region have shown me that it is possible in the face of fairly advanced phthisis to have tuberculous nodules in the endothoracic fascia as well as fibrous tissue.

This photograph from post-mortem No.17 is a section through a shotty, yellowish calcareous looking nodule which was one of several in the left extra-pleural fascia. It shows the structure of an organising caseous tubercle. This same case had a small lymph gland sectioned, from the anterior end of second left interspace, and this revealed active, proliferating, caseous tuberculosis.

The evidence, then, points to a spread of tuberculosis in some cases with adhesions from periphery of lung to endothoracic fascia. In my opinion this is vitally important because if a partial, artificial pneumothorax is induced and adhesions stretch, even to enucleate them is not safe as it may be quite possible to devitalise a focus like the one photographed with resultant empyema following. The presence of tuberculous foci in the extrapleural space also explains the tuberculous infection of extrapleural pneumothoraces and the extrapleural space infections containing tubercle bacilli after Semb's apicolysis in thoracoplasty operations. I have seen a case of intrapleural empyema following such an

extrapleural infection. The pleura had been torn slightly near the neck of a rib at operation.

The apical and apico-mediastinal adhesions do not elongate so readily and large apical cavities often have their outer walls formed of fused pleurae and organised endothoracic fasciae. They usually derive much of the blood supply of cavity wall through this fusion. Often the adhesion presents one free edge and tapers back to a symphisis. If partially divided, the blood supply of part of lung surface or cavity wall may suffer and subsequent sloughing and empyema formation, result.

Again in some cases tent-like projections of lung tissue or even cavity prolongations into adhesions have been described. To cut across them is to court disaster and enucleation is necessary.

If a simple looking adhesion is left uncut it may hold open a cavity and, as we have seen, the chances of fluid or pus forming are considerable (table 8). Even when no cavity is visible, adhesions, by their constant tugging on the lung surface, can devitalize it and small caseous areas are liable to form in lung parenchyma where the adhesion is inserted. It takes only a slight incident like a refill to positive pressures (as I have shown), or exertion, or a large cough, to cause a rupture through this area, i.e., a super-added spontaneous pneumothorax, and possible empyema.

Thus it is evident that artificial pneumothorax therapy is a dangerous procedure. I firmly believe that it should be reserved for an earlier type of case than is being admitted to most of our sanatoria at the present time. Unless all adhesions are divided and the lung made concentrically free, there is a great possibility that the tuberculous lesion remains uncontrolled in spite of absence of sputum and apparent wellbeing on the patient's part. Only too often I have seen relapse or bronchogenic spread of disease to the other lung when the artificial pneumothorax was abandoned after several years. As Brock (20) states "the

patient dates his recovery from the date of effective or complete collapse and not from induction."

I have shown (Cuthbert (108)) that about one third of those so-called "quiescent" cases can still produce viable tubercle bacilli when delicate means of detection like laryngeal swab or pulmonary lavage are used.

The ideal aim, then, is to induce an artificial pneumothorax on a case with slight or minimal disease (R.B.1 or the slighter degrees of R.B.2) No refills to positive pressures are given and it should rarely be necessary to give a refill of over 500 c.c. air. If possible, the patient should be afebrile before induction, have a Blood Sedimentation Rate of under 20 m.m. in the first hour, and should have had a spell in hospital of 4 or 6 weeks to allow him to settle. A preliminary phrenic crush and pneumoperitoneum often helps to attain those objectives.

After induction, no matter how free the artificial pneumothorax looks on the X-ray, a thoracoscopy should be done and all possible adhesions divided in about one month from induction. If it is not possible to divide them all they can be tackled at another stage two weeks later. It is bad policy, in my opinion, to half sever an adhesion and then leave it.

The sooner all this programme is done the better; for recovery starts when the artificial pneumothorax is effective. I have in hospital now a youth on whom I induced an artificial pneumothorax in May 1947 for right upper lobe disease of fairly slight extent. Several adhesions were divided soon after induction but 2 thin strings in the region of the upper paravertebral gutter were left as they seemed harmless. On the X-ray it appeared that there was a free artificial pneumothorax. Sputum vanished, general condition was excellent, and the patient rose to a 12 hours grade by March/1948 when he had a sudden spontaneous pneumothorax not associated with a refill, while "donkeying" the floor of a ward. One of the harmless looking adhesions had

ruptured. Non-purulent fluid developed and resolved with rest; fortunately he recovered, and the artificial pneumothorax was continued. This is only one of many examples of the dangers of uncut adhesions. Air travel in cases of pneumothorax likewise is dangerous if any adhesions are present, as the pleural gas expansion with increasing height causes positive intrapleural pressures and the liability of rupture of cavity walls.

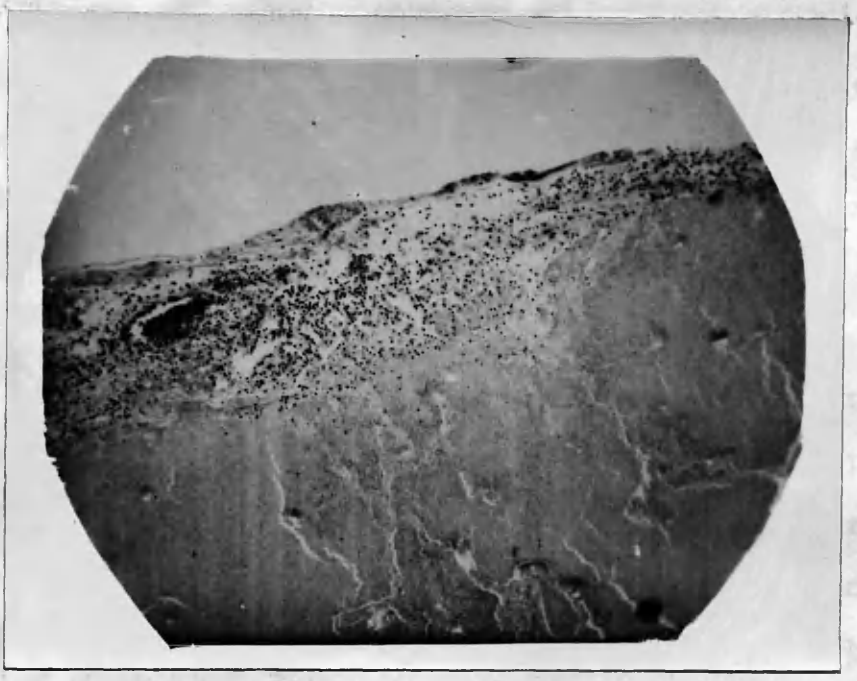
It is obvious that with an earlier type of case being treated there will be fewer adhesions, more chance of a larger percentage of free artificial pneumothoraces, and therefore fewer empyemata. Artificial pneumothorax therapy in advanced R.B.2 and R.B.3 cases is difficult. Repeated screening and radiological control is necessary, and if the artificial pneumothorax is contraselective and adhesions indivisible, it should be abandoned at once. Most empyemata are avoidable if clinical judgment is used.

Laes (109), Rafferty (8), Brock (20) and many others testify to the folly of maintaining a contraselective artificial pneumothorax. I mention the practice of stretching and rupturing adhesions purposely by large air refills, only to condemn it. It resembles the conditions found in flying to high altitudes.

SUB-PLEURAL CASEOUS FOCI WITH MINIMAL PLEURAL REACTION.

We have seen, then, how the pleura reacts to the approach of parenchymal disease to the surface of the lung by the formation of adhesions, and the role that they play in artificial pneumothorax therapy. I have been impressed in my researches by a type of lesion where there is little, if any, pleural reaction. In those cases a zone of firm caseation forms below the pleural surface which is little thickened and macroscopically shows no visible reaction. Post-mortem No.18 is a good example of this. Here there was a free pleural cavity apart from a few thin apico-mediastinal adhesions, and an artificial pneumothorax could easily have been induced. Over the axillary surface of the lung there

were subpleural, firm, caseous areas with a marbled appearance up to 2 square inches in area.



This L.P. photograph shows the proximity of the caseous material to the visceral pleura which is very little affected. The touch of a sharp artificial pneumothorax needle, especially of the Morland variety, which, incidentally, would be introduced in this area, could easily liberate caseous material into the pleural space. It is common to feel the lung surface impinge on the point of the needle during refills where the greatest care has been exercised, and it has even been stated by Tchertkoff (110) that in an induction the initial space is only found after the tip of the needle has traumatised lung and permitted a bubble of air to escape and separate the pleurae. (Occasionally I have seen patients cough slightly owing to a little of the local anaesthetic given before an artificial pneumothorax induction entering parenchyma by mistake, i.e., this is further proof that lung may be punctured accidentally at induction).

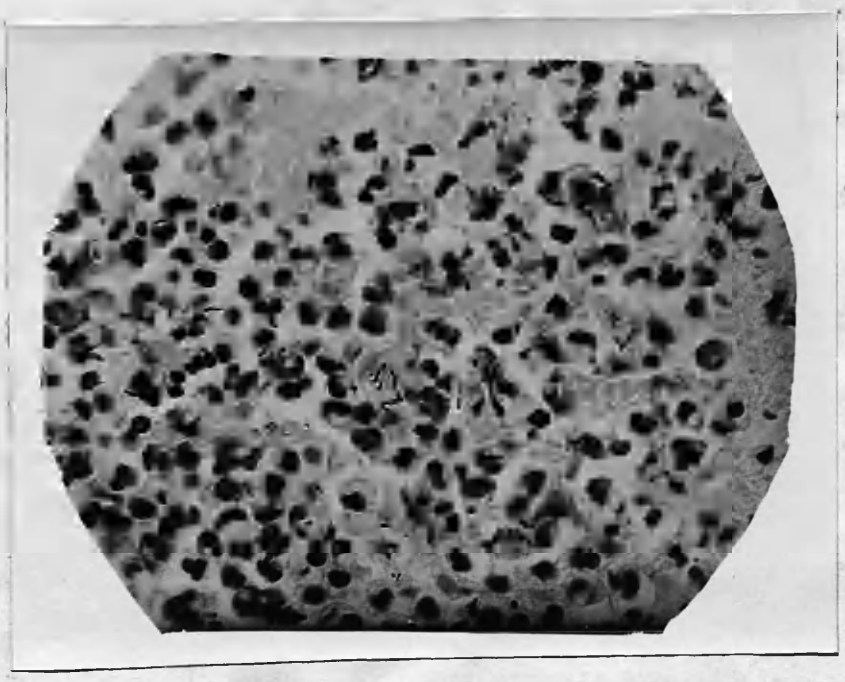
In post-mortem No.20 there was a right-sided clear, copious pleural effusion which developed within two days of death. The right pleural space was free apart from a few, friable, apical adhesions. On the lateral aspect of the

right lower lobe was a caseous mass beneath the pleura. Its area was that of a penny and its centre was soft and fluctuant. The overlying pleura was unthickened and like tissue paper. This cavity was of recent onset and was obviously the place where tubercle bacilli had invaded the pleural cavity. The thin pleura was on the point of rupturing and spilling its underlying soft contents into the pleural cavity too.

Those subpleural caseous areas, without much pleural reaction over them, explain the mechanism of spontaneous pneumothorax formation. Why there should be little pleural reaction may be due to the fact that endarteritis is present in the vessels which are running along diseased bronchioles. Obstruction of the latter causes collapse of the lobule of lung beyond in a cone-like zone (as is seen in pulmonary infarct) and a caseo-pneumonic change results - a "cold" inflammation with little vascular reaction.

Again, there is the condition of small, subpleural tubercles as opposed to those larger caseous areas. Small tubercles are often blood or lymph borne. Examples are sometimes seen at thoracoscopy when an artificial pneumothorax has been induced in the face of "haematogenous disease" as it is described. Here there has been local vascular invasion by tubercle bacilli and the resulting tubercles are scattered uniformly in a segment of lung, and reach the pleurae. The heat from a cautery at adhesion section may devitalise them and cause empyema. I have often seen examples of those fine tubercles, and in post-mortem No.10 they were numerous on both lungs. Joannides (111) demonstrated by photo-micrographic studies the frequent presence of tubercles on the lung surface, and Grancher and Hutinel (6) described the caseation of subpleural tuberculous lesions as being the forerunners of spontaneous pneumothoraces and broncho-pleural fistulae. Anderson and Alexander (112) observed tubercles on the pleural surface, through the thoracoscope, in "at least seven" of 111 patients.

Finally, this study of a subpleural cavity wall from post-mortem No.5 shows the very narrow margin of safety between parenchymal disease and pleural surface. In this case a silent spontaneous pneumothorax had occurred. The right lung had a large apical vomica, part of whose wall was adherent over the apex and upper chest wall. The lower pleural space was the seat of a pyo-pneumothorax and cavity wall partly roofed this space. A specimen was taken from this non-adherent area and sectioned.



The L.P. view (stained H.& E.) shows the pleural cavity and surface of visceral pleura (S). This is slightly thickened, covered with debris containing groups of polymorphs and lymphocytes, and fibrosed in its inner aspect. The

cellular reaction around the cavity wall is evident. At the extreme periphery of the cellular zone (area X) an oil immersion photograph was taken from the adjacent section which had been stained by Z.N. for tissues. This photograph shows numerous tubercle bacilli and proves that the bacilli are very near to the surface and that artificial pneumothoraces with surface cavities are dangerous.

It must be remembered in discussing the formation of spontaneous pneumothoraces that compensatory emphysema of unaffected lung is often found in the healthy lobes or even in the diseased lobe between lesions. There is nothing to prevent an emphysematous bulla rupturing as it does in lungs where there is no tuberculosis. Such instances might explain the fact that a certain proportion of spontaneous pneumothoraces escape without tuberculous pleural involvement.

It remains now to give some figures of the results of adhesion section. In chapter I, I gave the results of some other workers as regards the incidence of empyema. If the empyema onset is within three months of adhesion section, it is attributable to the latter operation.

There were available records of 90 of the 93 artificial pneumothoraces which were followed, in my series under review, by empyema.

27 cases of empyema followed adhesion section but only 18 of those were attributable to the adhesion section. 63 cases of empyema were due to artificial pneumothorax without adhesion section.

A. The overall average time of onset from induction, with or without adhesion section, was 10.4 months.
Case 10N, which developed empyema 6 years after induction, raises this average figure considerably.

B. The average time of onset from induction, where no adhesion section was done, was 8.4 months.

C. The overall average time of onset after all adhesion sections was 6.7 months.

D. The average time of onset in the 9 cases not attributable to adhesion section (over 3 months) was 17.6 months.

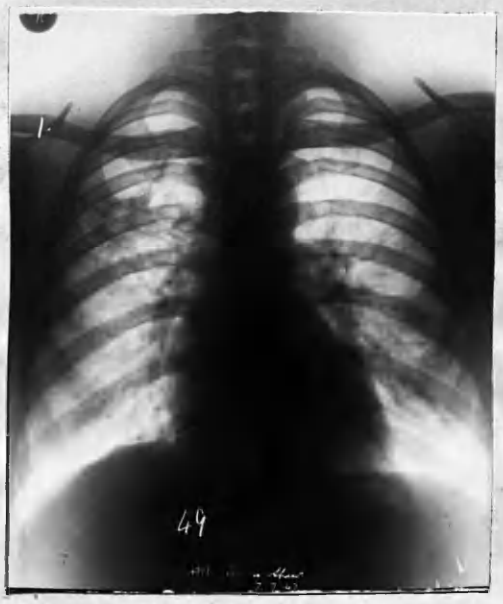
E. The average time of onset in the 18 cases attributable to adhesion section (under 3 months) was 1.21 months.

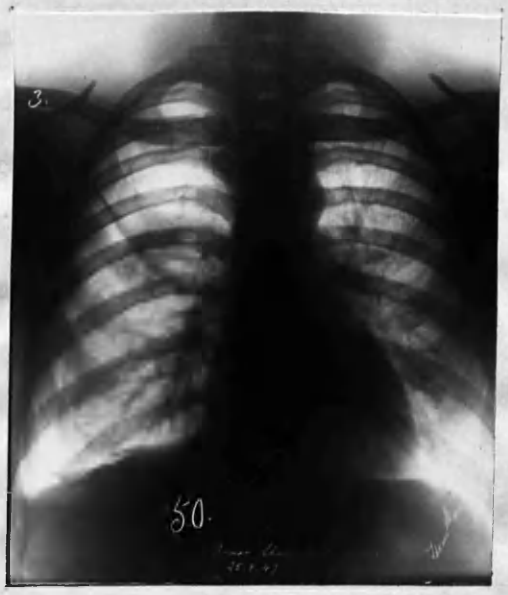
It is to be noted that this refers to the onset of pus and not non-purulent fluid which appeared much earlier than the pus. Comparison of E and D shows that if adhesion section is going to be of significance it does so early on.

Brock (20) states that if adhesion section is not done many more cases develop fluid and progress to an empyema than do develop it with adhesion section. Edwards and Lynn (113) say that with pneumonolysis complete collapse can be obtained in over 30% of all cases of artificial pneumothorax and Bayliss (36) believes that the more serious complications of artificial pneumothorax, such as empyema, are much more common in those patients in whom the pneumothorax is complicated by adhesions than when a technically satisfactory collapse is present. Drash (11) and Goorwitch (22) and many others also believe that large serous effusions and purulent fluids are less frequent following pneumonolysis than in unoperated artificial pneumothoraces restricted by adhesions. I believe, and have seen in Ayrshire, that this is so when fairly early or minimal cases are being treated by artificial pneumothorax and total adhesiotomy is possible. With R.B.2 and R.B.3 cases, however, complete division of adhesions is rarely possible and very few "free artificial pneumothoraces" are obtainable (see table 8). This explains the fact that in the Baguley series of cases the incidence of tuberculous empyema in artificial pneumothoraces where no adhesion section was done was 9.5% as compared with 18.3% in those artificial pneumothoraces having adhesion section. It suggests that it is better to leave those bad artificial pneumothoraces alone rather than to perform a partial adhesion section only.

Drash (11) states that "apparently the only valid contra-indication to adhesiotomy is the existence of an acute pleurisy with fluid, either serous or purulent", while

Brock (20) on the other hand, states that he has certainly seen fluid disappear completely after a satisfactory freeing of the lung. I have observed this on several occasions and even a case of empyema cleared up after aspiration and division of all adhesions, and the artificial pneumothorax is maintained at present and dry, a year after adhesion section. The following X-ray reproductions illustrate this point.

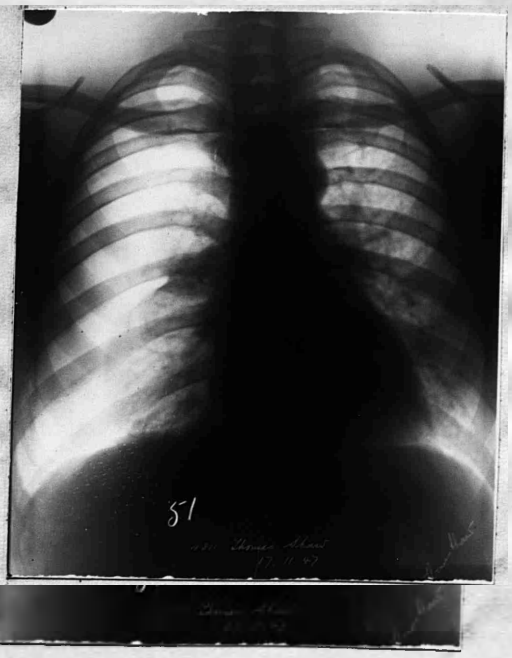




T.S. No. In the right lung, below clavicle, an area of clearing, with definite mottliration is present. The cavita lung fields appear clear. left l

No. 50. 25.8.47.

T.S. No. A right artificial pneumothorax is present with a layer of fluid at the base (taken after aspiration). The vomica in right upper lobe is patent and several string-like adhesions are visible stretching fanwise from the right upper lobe to chest wall. Left lung field remains clear.



T.S. No.51. 17.11.47.

This was taken after complete adhesion section and

T.S.No.51 (continued):-

aspiration. A free right artificial pneumothorax is present with rounded upper lobe down to lower border of anterior end of first rib. No evidence of vomica in right upper lobe. No signs of any adhesions and no fluid in right costo-phrenic angle. Left lung field remains clear. (The patient's general condition is excellent).

CHAPTER V.

THE CLINICAL COURSE OF EMPYEMA.

We have previously (Tables 1 and 6) mentioned the numbers of artificial pneumothoraces which developed non-purulent fluid. Weisman (114) found effusions in 84% of 150 consecutive artificial pneumothoraces, but from collected cases he reports clear effusion as occurring in 17.1% to 90% of artificial pneumothoraces, while Coryllos (3) states that straw-coloured, non-purulent exudates occur in 50% to 70% of all artificial pneumothoraces. In some cases, with careful aspirations and adequate rest, the fluid will absorb and the artificial pneumothorax may be continued. Occasionally an obliterative pleuritis starts and the lung slowly re-expands. In other cases the fluid persists and after a variable period, even after a year, say, gradually changes its character, until finally it becomes opalescent, turbid and finally purulent. Other empyemas start abruptly with non-purulent fluid present for a very short time, measured in days or weeks, before the onset of pus. The purulent fluid may contain tubercle bacilli alone or secondary pyogenic organisms when it is a "mixed infection empyema". The prognosis is very much worse in the latter.

Coryllos (3) states that about 18% of all effusions become purulent. Hayes (115) reports 21% and Dumarest and Brette (15) 16% of effusions as becoming purulent.

It is stated in text books that sepsis introduced from the outside at aspiration is a common means of determining the onset of mixed infection empyema. This may be so but

where there has been no chest intubation or sinus formation I think that most secondary infection is from the bronchial tree. Most, if not all, empyemata are associated with broncho-pleural fistulae of varying sizes. The small fistula possibly communicates with alveoli which are free of secondary organisms. Larger fistulae possibly communicate with septic cavities (as I have seen) and the larger bronchi, and in those secondary infection easily supervenes in the pleural space.

A study of pleural fluids from cases of pulmonary tuberculosis in Baguley Sanatorium and Baguley Military Wing between January/1944 and December/1947 was interesting. Those fluids were sent for routine analysis after aspiration, and were from 209 separate patients (Table 12). Clear fluids contain many white cells under the microscope. Only macroscopic evidence of pus is counted as "empyema".

TABLE 12.	T.B.Minus Clear fluids always.	T.B.+	Pure Tub. Empyema.	Pure T.E. progressing to mixed infection.	Mixed infection from the outset.	TOTAL CASES.
Jan.-June 1944.	10	1	3	-	3	17
June-Dec. 1944.	10	2	1	1	-	14
Jan.-Dec. 1945.	37	15	2	3	6	63
Jan.-Dec. 1946.	48	7	7	-	4	66
Jan.-Dec. 1947.	24	1	11	1	12	49
TOTALS:	129	26	24	5	25	209.

Of the 155 patients who had clear fluids throughout, 26 were tubercle bacilli plus after centrifuging the deposit and staining by Z.N. No guinea-pig or culture technique was done as a routine. This latter procedure raises the yield of positive fluids as I have often found. Those fluids were non-purulent.

Twenty-four cases of empyema remained as pure tuberculous empyemata throughout their illness. In only 5 of the cases was there evidence of a pure tuberculous empyema of several months standing having progressed to a "mixed" infection empyema. Twenty-five cases had "mixed" infection

empyemata from the start or from an early period measured in days or weeks from the onset. This rather suggests a wide communication at the outset between pleura and large or medium sized bronchi.

The following table (No.13) indicates the relative frequency with which the various organisms causing secondary infection occurred. Many cases had several types of organisms present.

TABLE 13.

Type of organism.	Frequency of occurrence.
Coagulase positive Staphylococcus Aureus.	15
Diphtheroid organisms.	10
Staphylococcus Albus.	7
Coliform organisms and B. Proteus.	7
Non-haemolytic Streptococci.	3
H. Influenzae; B. Pyocyaneus; Staph.Citreus; Haemolytic Streptococci; Vincent's Fusiform Bacilli and Spirochaetes; Non-haemolytic Anaerobic Streptococci.	1 each.

It will be seen that a large proportion of the organisms are penicillin or sulphonamide sensitive, but many are not.

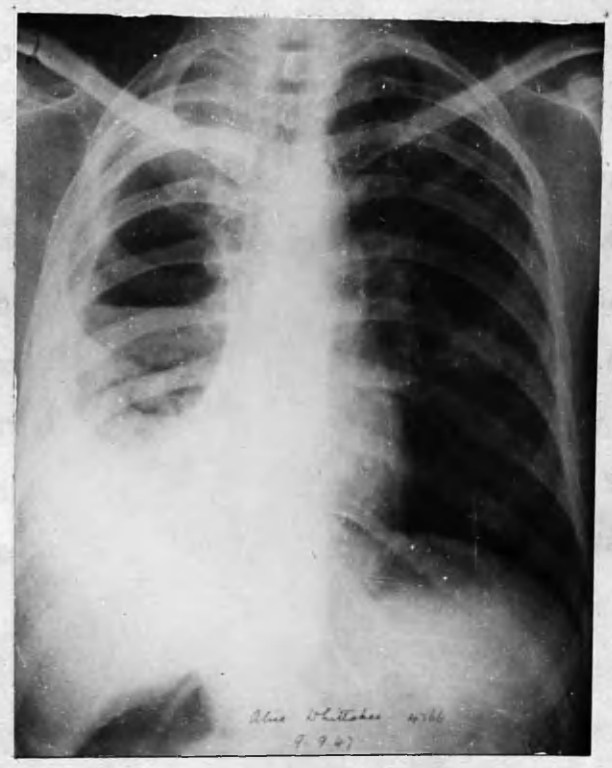
The finding of Vincent's organisms is interesting. They are common in putrid lung abscesses and are normal inhabitants of the mouth and throat, assuming epidemic virulence in times of war. The above case had extensive pulmonary tuberculosis. Jump and Sperling (116) cite a case where there was a combined Vincent's infection of the pleura and vagina. Bronchograms suggested a bronchiectasis in the lung. Those authors failed to find a record of any case of empyema due to Vincent's organisms without an underlying pulmonary lesion. (In another case with abdominal and pulmonary tuberculosis (post-mortem No.10) the sputum had numerous fuso-spirillory organisms before death. There was no empyema here - the tuberculous lung lesions were early and the cavities small).

The case in which haemolytic streptococci was recovered was No.9J in our series. Following a right partial adhesion section 9½ months from artificial pneumothorax induction, an empyema was present within 7 days. The patient at the same time developed a fulminating haemolytic streptococcal septicaemia and died in 4 days. Presumably this was an exogeneous infection.

Anaerobic cultures of pleural fluids are not done as a routine so that the true incidence of anaerobic organisms cannot be stated. It might repay study.

It is impossible to forecast the course of an empyema. No two cases are alike, as the extent of the underlying Parenchymal disease and the condition of the contralateral lung varies.

The attached photograph is included, as it represents a group of cases often mistaken for empyemata if earlier films are not available. The apparent right empyema is in reality a giant pulmonary cavity. The whole lung is excavated. Even at post-mortem, in such cases it may be hard to detect the remnants of the peripheral lung tissue which are glued firmly to the chest wall. True empyemata may take several courses, depending largely, I think, on the size of the broncho-pleural fistula.



In some cases a clear or turbid, non-purulent pleural effusion, associated with an artificial pneumothorax, changes its character and becomes slowly purulent after several

months. In other cases, not infrequently, the onset is more dramatic, when a spontaneous pneumothorax is superadded to an artificial pneumothorax. The series of cases under review offers many examples of this. The patient complains of dyspnoea which may come on after exercise or a coughing bout or may occur while he is resting in bed. In some minor cases the discomfort passes in a few hours; in other episodes the intrapleural pressures rise and the breathlessness with it. Shock may be present. Screening reveals that the lung is more collapsed than usual and the mediastinum may be displaced to the opposite side. That evening the temperature will be elevated. Hutchinson and Blair (117) were among the first writers to illustrate how a steep initial rise of temperature is coincident with the first symptoms of superadded lung rupture in artificial pneumothorax therapy. There is usually a gradual staircase fall to normal in about a week's time. When the lung rupture has been severe, however, as when part of a cavity wall, devitalised by the heat of a cautery, sloughs, then there may be no return of the temperature to its previous level but instead the rapid onset of empyema. Left to itself toxaemia usually progresses and the temperature remains elevated with marked deterioration in the patient's general condition and death results. The mediastinum, if not fixed, is pushed to the opposite side and the lung on the affected side becomes progressively collapsed and airless. Oedema of the lower chest wall may be seen and "empyema necessitatis". In empyemata of older standing the pus may gain access to an intercostal space and track along the line of intercostal vessels to present at the border of the sternum anteriorly, or posteriorly near the outer border of the erector spinae muscles, or in the posterior axillary line. In rare cases the pus ruptures into the oesophagus.

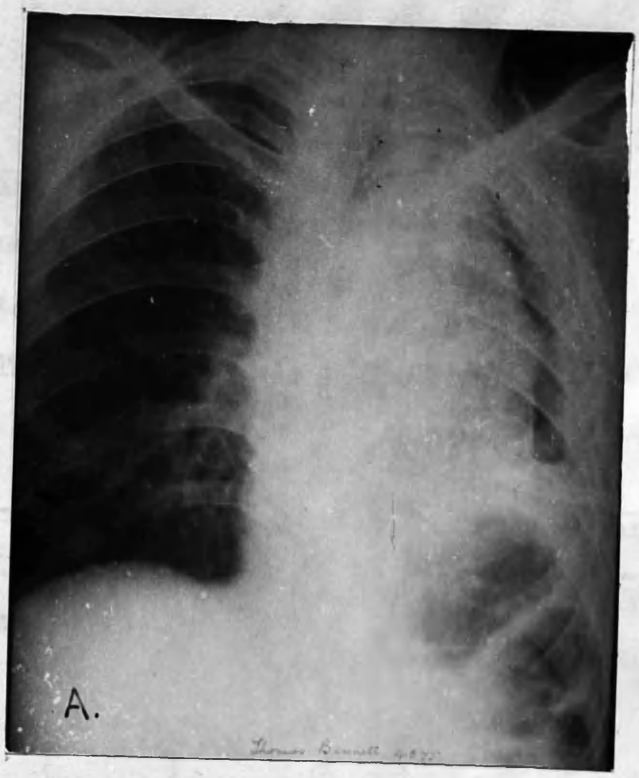
If the pus becomes encysted and the pleurae greatly thickened, toxaemia may be less marked and the patient will in some cases be able to walk about and even do a certain amount of work. I have seen such cases and Chandler (118)

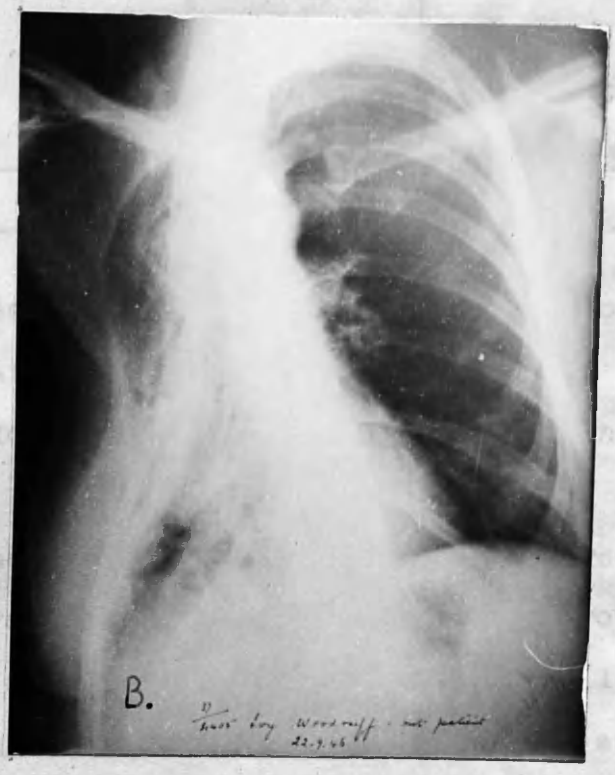
quotes cases where such encysted empyemata remained for twenty years, the patient remaining in perfect health. I consider that such cases, however, are merely marking time and the condition may be likened to a time-bomb, liable to go off unexpectedly when the patient alters the even tenour of his ways and lowers his general condition. In one of my cases the pus became so inspissated that a calcium plaque was formed eventually and had lasted for many years. Finally, the underlying parenchymal lesion deteriorated. At exploratory aspiration of the pleural space the needle had to be bored through chalky material. The aspiration yielded a few c.cs. of turbid fluid containing viable Tubercle Bacilli.

With frequent needling of empyemata there are several dangers of which the commonest is sinus formation in the chest wall. Occasionally secondary infection is introduced from the aspiration and this almost always follows once a sinus forms. Finally, tuberculous meningitis is often associated with empyemata. I have seen three such cases in the past year and can only think that the needle point has carried infected material into a blood vessel during the manipulations.

As in bronchiectasis so in cases of empyema, brain abscess has been described. Slowly, as the cachexia continues, there is a thickening of visceral and parietal pleurae and the mediastinum becomes fixed. The extrapleural fascia becomes organised and deformity ensues.

The ribs crowd together, become triangular in section, and are adherent to their periosteum as is seen at operation. The spinal curve is concave towards the side of the lesion. Photograph A represents the deformity in an old-standing empyema. (Photograph B shows that after thoracoplasty where deformity has been uncorrected the spinal curve is in the opposite direction, being concave to the healthy side, and the remaining ribs on the thoracoplasty side are splayed apart, while those on the healthy side are crowded together. This photograph is included to compare the deformities in empyema and as a result of uncorrected thoracoplasty).





Amyloid disease is often found associated with sinus and secondary infection of the empyema cavity. Finally, I mention it last because it so infrequently occurs, the pus may be gradually absorbed or may be kept in check with frequent aspirations, until the underlying lung re-expands and the empyema ceases to exist.

The above clinical picture gives an idea of the severity of the condition which we are discussing. What is the mortality rate?

The Mortality of Tuberculous Empyema.

This analysis includes the 104 cases under review, plus case 9H due to spontaneous pneumothorax which was treated by operations. Table 14 shows the overall figures irrespective of the type of treatment adopted.

TABLE 14.	Number.	Percentage of total.	Special case numbers.
Death under 5 years from empyema onset.	71	67.6) 74.3	
Death over 5 years from empyema onset.	7	6.7)	
Recovery from empyema.	20	19.05	
Improvement. (Sinus remains on 3.4.1948).	2	1.9	6B; 8D.
Lost sight of and deteriorating.	1	.95	4A.
Empyema persisting on 3.4.1948.	4	3.8	10G; 10M; 9H; 10K.
TOTAL:-	105	100.0%	

In all, 43 cases were discharged or took their own discharge following treatment for empyema, and had to be followed up through various Tuberculosis Officers. One case was lost sight of.

Only twenty cases (19.05%) of the total recovered completely from the empyema. In two cases there was improvement in the patients' clinical condition but a chest wall sinus remained on 3.4.48, necessitating daily dressings and discomfort. The case who could not be traced (No.4A) was deteriorating rapidly when she took her discharge and the prognosis then was extremely poor. Seventy-one cases (67.6%) died within five years of the empyema onset. Six cases

persisted on 3.4.48. Their condition is summarised as follows:-

Case No.	Duration of empyema to 3.4.1948.	Prognosis, etc.
8D.	9 years.	Fairly good. Patient is well with a chest wall sinus.
6B.	13 years.	Patient fairly well and does house-work, but is of poor colour. Sinus persists.
9H.	6.1/12 yrs.	Poor prognosis. Chest wall sinus. Oedema of ankles and albuminuria.
10G.	2½ years.	Poor prognosis. General condition is deteriorating.
10K.	5 years.	Poor prognosis. Deteriorating general condition.
10M.	2 yrs. 8 mths.	Rapid deterioration. Bad prognosis.

On the whole, then, the picture of tuberculous empyema is a gloomy one.

The next chapter will be devoted to the treatment and management of the condition.

CHAPTER VI.

THE TREATMENT OF TUBERCULOUS EMPYEMA.

In May 1947 the wards of one of the large Manchester Municipal Hospitals, which housed advanced cases of pulmonary tuberculosis, closed. The patients were discharged to Baguley Sanatorium and included a large number of cases of tuberculous empyema. A study of them confirmed the previous views regarding the etiology of the condition. Valuable experience as regards treatment was gained.

In broad lines it may be stated that there are four main approaches to the treatment of tuberculous empyema.

(a) To leave them alone, only aspirating when the fluid by its pressure and volume is causing dyspnoea or cardiac embarrassment.

(b) The use of regular or frequent aspirations. Varieties of this are aspirations using high, terminal, negative intrapleural pressures; the use of continuous negative suction, or the use of chemotherapeutic

(b) continued:-

agents as intrapleural washouts or by systemic routes.

((a) and (b) may be called "conservative methods" of treatment).

(c) The use of surgical methods, extensive thoracoplasty and plastic operations, after conservative methods have had an extended trial, usually after many months or even years.

(d) The early use of surgical methods, i.e., within a matter of weeks from the onset of the empyema.

Rosenblatt (119) quotes the results in 51 patients with tuberculous empyema treated conservatively, and concludes that it is a mistake to say that the patient is doomed if he does not submit to surgery. Chandler (118), too, and others, advocate conservative measures. Chandler states "my own view, based on a long experience, is that they should be left alone unless there is some very definite reason for intervention; that they often do more good than harm; that they can persist for 10 and 20 years or more, the patient keeping in perfect health."

On the other hand, Brock (120), Hoyle (121), Coryllos (3) and many others, advocate surgical measures of treatment in many cases, and Klassen, Miller and Curtis (122) state that in their experience aspiration and irrigation alone have been of no apparent value in the treatment of empyemata.

Thus the stage is set for a discussion on the subject.

Conservative Methods of Treatment:

In the series under review 90 cases of tuberculous empyema were treated by conservative methods. Table 15 shows the results.

TABLE 15.

	Nos.	Percentage.	Remarks.
A. Death under 5 years.	66	73.3 } 77.7%	Average duration of empyema 20 months.
B. Death over 5 years.	4	4.4 }	
C. Full recovery.	16	17.8%	
D. Improvement and alive on 3.4.48 with sinus.	1	1.1%	No.6B. Duration 13 years to date. Fair general condition.
E. Lost sight of.	1	1.1%	No.4A - deteriorating.
F. Empyema persists. Deteriorating 3.4.48.	2	2.3%	10G; 10M.

Counting groups E and F the total deaths or impending deaths with conservative treatment amount to 81.1%. No one can deny that this shows a poor prognosis with conservative treatment alone.

Only 16 cases (17.8%) had complete recovery from the lesion (4B, 4H, 7B, 7C, 8A, 8C, 8H, 8J, 9D, 9E, 9G, 10D, 10E, 10.I, 10L and 10T). The lists on page 177 show the durations and exact methods of treatment.

It is vitally important in comparing the results of other workers in this field that the definition of empyema should be standardised. Thus Gibbons (1), whose definition of empyema is "any collection of fluid in the pleural space, containing bacteria", reports that he continues pneumothorax for as long as possible, and that many of his cases show a spontaneous resorption of the empyema without adverse effect upon the pneumothorax or the underlying lung. This has not been my experience with purulent empyemata and it is not the outcome in the early cases of this series where air replacement of pus was attempted in 21 cases with a fatal outcome in 18 cases.

Coryllos (3), discussing turbid and purulent exudates only, states that most authors show 15 to 25% of good results with conservative treatment alone.

Woodruff's (2) definition of a tuberculous empyema does not come up to our standard. He defines tuberculous empyema as "any turbid pleural fluid in the presence of pulmonary tuberculosis or any fluid containing tubercle bacilli on direct smear." Such empyemata without secondary

infection are placed in Group A, while Group B contains empyemas with secondary infection. In spite of his lower standard of empyema his results with conservative treatment are bad, e.g.,

Cases with no treatment.	No.	Dead.	Resting.	Well and working.
	6	2	2	2
Cases treated by aspiration only.				
GROUP A.	38	15	8	15 (39.5%).
GROUP B.	6	5	1	0
Uncertain.	2	1	0	1
TOTAL:-	46	21	9	16 (35%).

Woodruff states that of the 16 who are well and working the lungs of 9 are presumably re-expanded. This, then does not satisfy my criterion of cure, for I consider that a space containing fluid, no matter how well the patient may feel, is a constant threat to future health. His results with pleural irrigations of saline or the common dyes are similar.

Ornstein and Herman (123) obtained complete re-expansion of the lung in 48 of the 98 cases they treated by conservative methods but their results are not comparable with our series, as a study of their complicated classification reveals that their definition of empyema includes (a) non-purulent, (b) sero-purulent and (c) purulent fluids. I have found that the former two types of fluid do not offer the same problems in treatment as purulent fluids.

Smyth (124), in an excellent opening paper at the inaugural meeting of the Irish Society of Thoracic Surgeons, shows how only a few cases "reach the shore" safely with conservative treatment and that the majority die.

In the era before sulphonamides or penicillin were used, the diversity of drugs employed to treat the common erysipelas showed that no one method was specific and that the results were often a matter of chance. So, in the treatment of tuberculous empyemas, the number of remedies advocated is legion. I shall mention some of them here to indicate the

position of affairs with regard to the conservative treatment of the condition. The great numbers of the remedies sought show that the condition is a troublesome one.

Coryllos (3) lists as pleural irrigation substances that have been used - normal saline, colloids of gold, copper or silver (argyrol, argochrome), formalin or iodoform in glycerin, lipiodol, ethyl morrhuate, allochrysin, methylene blue, gentian violet, Lugol's solution. myrol, solganol-B, insufflations of the vapour of azote-gomenol, potassium permanganate, acriflavine, trypaflavine, Dakin's solution, vuzin, rivanol, simple oil or oil with gomenol, vaccines and bacteriophages. Fishberg (45) adds to this list lysol and quinine. To those may be added heparin as described by MacMillan (125) who claims that it has no effect on the patient's blood-clotting time but that it facilitates aspiration by dissolving fibrinous material.

Paquette (126) advises repeated pleural lavage with dilute solutions of mercurochrome. Bruce and others(127), mention the use of diasone and other sulpha-drugs in mixed infection empyemata, but penicillin in large doses after pleural lavage was the best treatment in their hands when no open drainage was employed.

Donaldson and Samuel (128) used aspiration, lavage with distilled water, and the instillation of 10 to 50 mg. of tyrothrisin with no effects upon the character or the amount of purulent exudate in 8 patients suffering from tuberculous empyemata.

Ornstein and Herman (123) advocate lavage of the pleural space with oxygen. They also wash out the pleural space after aspiration with azochloramide 1 : 2,000, with triethanolamine or sodium tetradecyl sulphate 1 : 800, and later they use a mercurial antiseptic and sodium tetradecyl sulphate.

Raab (129) treated 6 tuberculous empyemas, 5 of which were purulent, by weekly injections of 5 to 8 c.cs. of sterilised Vit.A and D concentrate. Although the author

considers that this form of treatment merits further investigation, analysis of his results shows that he had no true cures and that fluid was still present in each case.

Coello (130) has injected patients' whole blood and penicillin into the empyema cavity after aspiration. O'Brien, Brown and Pearse (131) instill, twice daily, after irrigating the pleural cavities "a new bacteriotoxic solution, glycerite of hydrogen peroxide (1.5%) containing 0.1% of 8-hydroxy-quinoline". They give 9 case reports and a careful analysis of those does not tally with the authors' claims that the sterilization of the empyema cavities occurred in 7 out of 9 patients with cutaneous fistulae.

Andosca and Foley (132) used Calcium Ribonate and Vitamin C in six cases who had tuberculous empyemata. Aspirations were done, on an average, every two weeks. After three months therapy there was definite improvement in three cases as shown by a less viscid and less copious fluid. There was no change in two cases and one case died. This is an unscientific article with no mention of the presence or otherwise of fistulae, secondary infection or the duration of the empyemas.

Streptomycin has been used too, in tuberculous empyema. I, personally, have had no experience with it. Hinshaw, Feldman and Pfuetze (133) found improvement in only one out of seven cases treated by intrapleural or intramuscular (or both) streptomycin. Their dosage was 1 to 3 g. per 24 hours divided into 4 to 6 doses. The solution consisted of 100 to 250 mg. of streptomycin in 1.0 c.c. sterile water. Hinshaw, Pyle and Feldman (170) reiterate those views. Freedlander and French (134) report that streptomycin is ineffective in empyemata which are usually acid in reaction. They point out that the drug is alkaline, insoluble in lipids and highly ionized. Similarly, the Committee on therapy and its sub-committee on Streptomycin (American Trudeau Society) (135), in November/1947, point out that streptomycin is recommended for the treatment of

tuberculous, draining, cutaneous sinuses and sppears to be highly effective in a large majority of cases, regardless of the underlying disease, except that sinuses associated with tuberculous empyemata are less likely to respond to treatment. They do not recommend streptomycin for the treatment of chronic empyema of tuberculous origin because in studies reported to date it had apparently been ineffective. A great drawback with streptomycin is that many strains of tubercle bacilli appear to become drug resistant.

Recently para-amino-salicylic acid has been described by Dempsey and Logg (136), Lehmann (137), Vallentin (138) and others. We will discuss results with it, later.

At the end of the subsequent case reports a table (page 177) summarises the individual results and methods of treatment of the 90 cases of tuberculous empyema in our series, treated by conservative means.

Discussion:

It is now necessary to consider why conservative methods of treatment usually fail. At the outset it may be stated that no two cases of tuberculous empyema or indeed of uncomplicated pulmonary tuberculosis are alike and that fact does not simplify the question.

The fluid may be pure tuberculous pus or contaminated with secondary organisms. There may or may not be chest wall sinuses present. The disease in the underlying lung may be quiescent or cavitated. The opposite lung may be involved by a tuberculous process. Patients' general conditions differ widely. Finally, the size of the bronchopleural fistula present, must be considered.

Provided the empyema is uncontaminated with secondary organisms I am convinced from personal experience that a most firm grip of affairs must be taken from the outset by the physician. It is a serious condition with bad prognosis and aspirations must be thorough and at regular intervals. It is no use allowing the pus to gather and further compress the lung with longish intervals between aspirations.

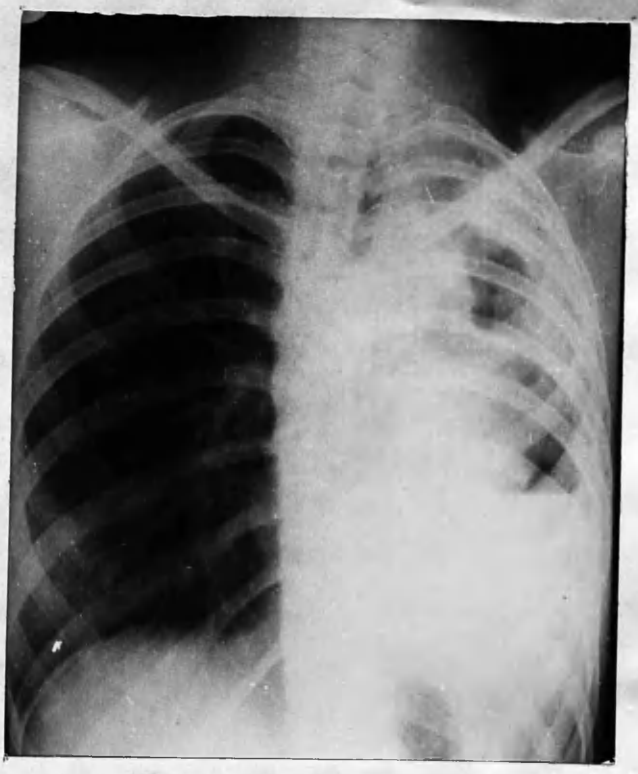
In spite of the argument that too many needlings lead to fistula formation I consider the chest should be kept as dry as possible, and this usually necessitates aspiration every 2 or 3 days from the outset. In the ideal course of events, where a negative intrapleural pressure can be attained, all the pus should be removed and then excess of air until the patient experiences a tight feeling in the chest. In the absence of gross underlying disease I have recently had success with such cases where, in addition, the patient was made to blow up a rubber air-ring every 2 hours.

The points about aspirations are that the technique should observe all the rules of asepsis.

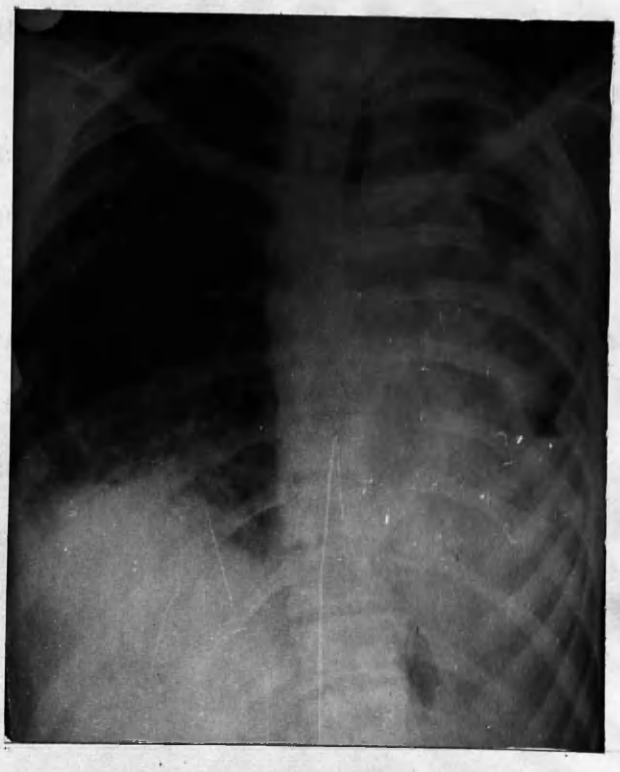
Pus is often easier to withdraw using a 10 c.c. syringe than, for instance, a 50 c.c. syringe. I, personally, always use a 10 c.c. syringe with a two-way tap which transmits the pus through a foot of rubber tubing to a receiver. The bore of the needle selected will depend on the character of the pus. It is sometimes advisable to have a short piece (1 inch say) of rubber tube between the needle and the syringe. This allows for jars and movements of the syringe not being transmitted to the needle which keeps its place more easily in the chest wall.

Aspirations should be done using the minimum of local anaesthetic (no more than 2 c.c.) as excessive local anaesthetic may devitalise the intercostal tissues, when it is repeated often, and prepare a suitable soil for a chest wall sinus. The site of election for aspiration is high in the anterior axilla - "anterior" to be away from the field of any future operation - high in position so that the needle track will not be bathed in the residual pus after the aspiration. In order to get the last traces of pus the needle and the patient can be tilted into suitable positions. It is a good plan at the terminal stages of the procedure to have the patient exhale deeply and hold his breath out. This elevates the pus with the hemi-diaphragm on to the needle point. After aspiration the patient will recline for an hour

on the opposite side to allow the needle track to seal off.



Unless there is much fibrin I prefer not to irrigate. If the latter is done I think that saline is as good an agent as any and certainly non-irritant, and my results and those in the series under review, quoted previously confirm this. Indiscriminate irrigation is even dangerous. The attached photographs show a sudden bronchogenic spread of pus through an undetected large left-sided broncho-pleural fistula to the right lower lobe. This occurred after the first irrigation of Azochloramide and was the 'straw that broke the camel's back' in this case, the patient deteriorating rapidly thereafter.



Where the aspirations are performed religiously, in favourable cases, the lung will re-expand and the parietal and visceral pleurae stick together and organise. In spontaneous pneumothoraces it is a good practice to introduce a Monaldi type of tube and apply continuous negative suction from an electric pump to attain this end.

All too frequently, and with the best attention, the lung re-expands slowly and then remains stationary, often with a narrow, peripheral slit of pleural space left. At

this point aspiration must be thorough. If the fluid is permitted to re-accumulate the lung will collapse again. Even if the air and pus are all removed and the parietal and visceral pleurae come in contact, they will not necessarily adhere, and in a few days screening will show that the lung is away from the chest wall again and a few more ounces of pus can be obtained. The longer the lung takes to come out the less chance has it of finally, completely re-expanding. Histological study was made of numerous cases of empyemata. Some of the material was obtained from parietal pleura at operation, other sections were from post-mortem material. The explanation of this difficulty in making parietal and visceral pleurae adhere is that the longer the pus persists the thicker is the deposit of fibrinous material and curd on the pleural surfaces. Sections show that new capillaries and granulation tissue only invade this fibrin in the deepest parts. The periphery is avascular. When an ordinary wound granulates and the two granulating edges are approximated, they usually stick readily, and fibroblastic and capillary bridges soon unite the surfaces firmly. Not so with the pleurae. For them to stick together entails several weeks of close apposition to allow the avascular part of the fibrin bridge to be invaded with granulation tissue. After an empyema has lasted several months, it is almost impossible to attain those conditions by aspiration alone as breathing, coughing, and the effect of gravity, all pull the elastic lung away from the chest wall.

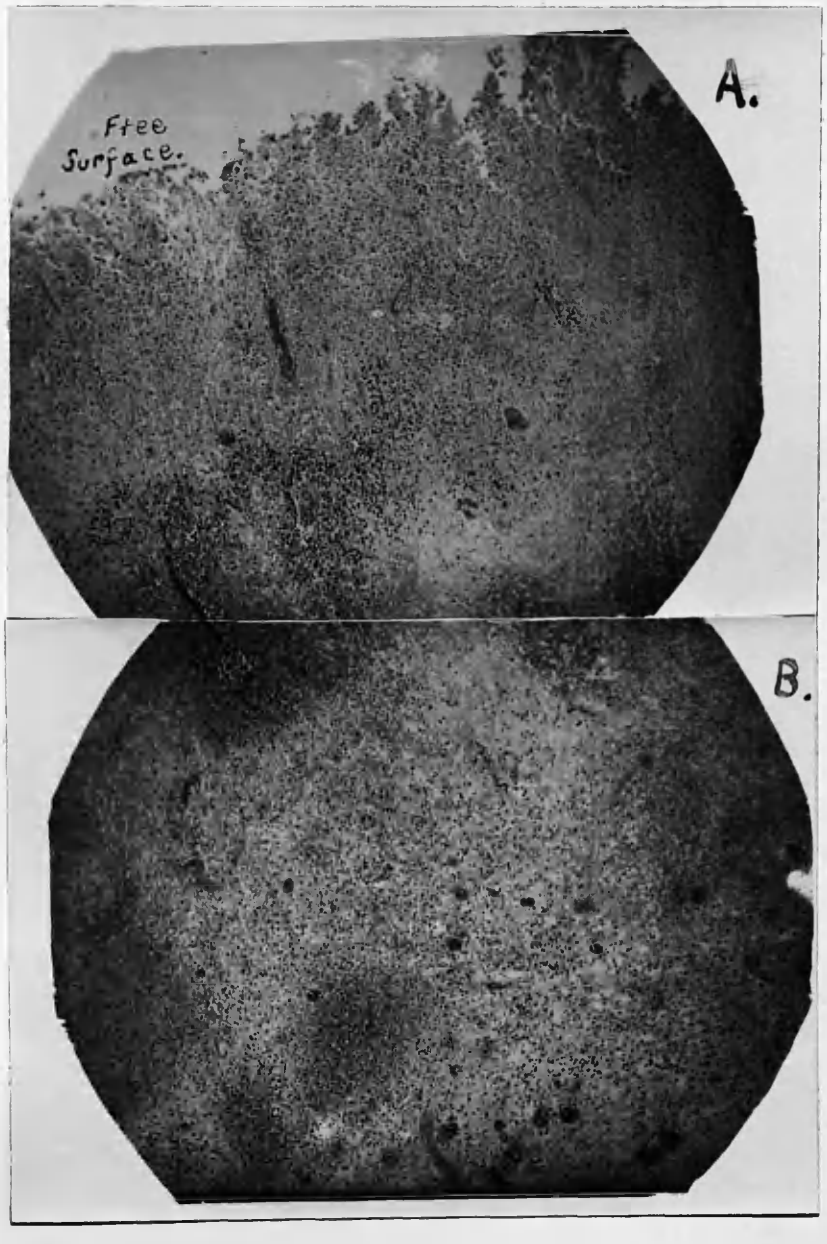
Why not intubate with difficult cases? As a rule uncontaminated pure tuberculous empyemata soon develop a secondary infection when intubated. (Secondarily infected empyemata, of course, usually have to be intubated to combat the pyrexia and rapid toxaemia from the start).

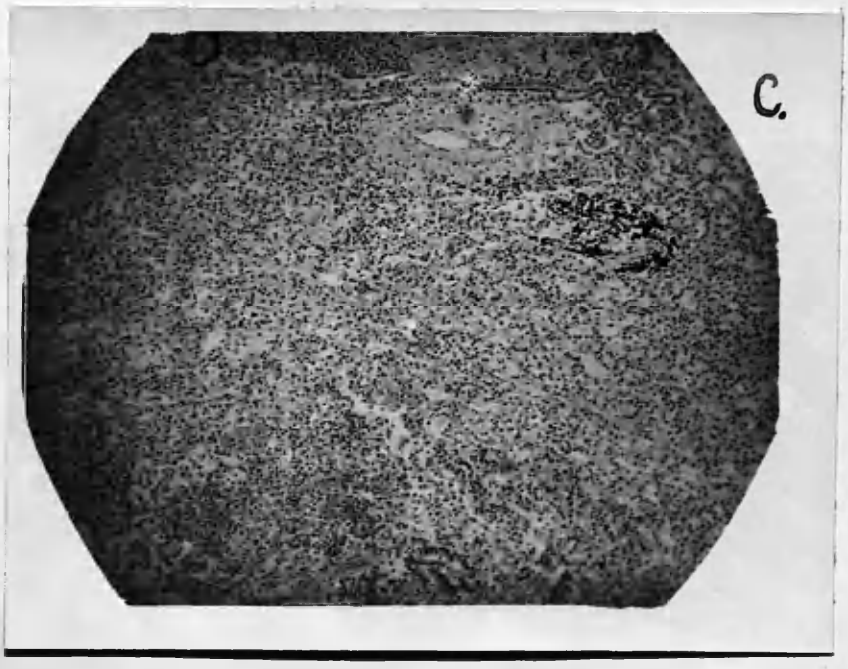
In any case, a sinus forms and adds to the difficulty. My experience has been that intubation of simple tuberculous empyemata usually sets the patient on the downward path unless surgery is contemplated. I have not found that the lungs in

such cases re-expand faster, even if continuous suction with an electric pump is used. It is hard to keep up the negative pressure, and collodion, vaselined gauze or perspex in chloroform, have all been tried to seal off the entrance of the tube to the chest wall. I think it is bad policy to resect a piece of bone at intubation in every case. Long-standing sinuses develop a regeneration ring of bone round the tube with the sinus in the centre. I have a fine specimen, removed at operation, in my possession. Such sinuses could hardly heal by themselves. However, old-standing empyemata have the ribs so "tiled" and close together that there may be no other alternative to resection of a segment of rib.

All the classical causes whereby sinuses do not heal are usually seen in a case of tuberculous empyema. Mobility of the part (ribs and lung) is present. Inadequate drainage is common as the costo-phrenic angle usually forms a sump where pus lies. There is often a broncho-pleural fistula present too, and the thickened epithelial and fibrinous lining of the pleurae maintain purulent secretions. Finally, there are tuberculous tissue and tubercle bacilli concerned, nearly always with secondary septic invaders and a poor general condition of the patient.

The accompanying diagram shows the state of affairs with regard to visceral pleura obtained from the case described in post-mortem No.13. This empyema had lasted for 5½ months. The peripheral zones 1 and 2 have no vascular, granulation tissue. There are collections of lymphocytes and occasional polymorphonuclear leucocytes in layer 2.


Free
Surface.
A.
B.


C.

(Photograph 'A' from the actual section). It is only in the deepest layer of the fibrin curd that young blood vessels are seen. (Layer 3 or photograph 'B' from the actual section). Layers 4 and 5 represent thickened visceral pleura and a thin layer of collapsed and organised lung respectively. This latter zone in many sections contains pigment (photograph 'C'). Histiocytes laden with pigment can be seen in deeper lung tissues but whether they are going or coming from hilum to periphery is not known. The lung beneath this area is aerated (area 6 in diagram) in this case.

When intubation is indicated I prefer to connect the tube to a continuous suction apparatus. A recent type of rubber tube on the market is radio-opaque and this is useful at screening to determine that the tube is in the optimal position for drainage. I think that drainage under a water seal at the bedside without suction serves, as a rule, very little useful purpose. As it is usually adopted the weeks or months slip past with daily irrigations and progressive pleural thickening as shown above. Quite apart from the condition in the underlying lung it is this progressive pleural thickening that spells disaster to the majority of programmes of treatment where conservative methods are used no matter what new remedy is tried in the pleural space.

At operation or post-mortem examination on an empyema of over 4 months' standing, it is usually found that the thickened visceral pleura is like an orange rind round the lung, and expansion of the organ, apart from its own disease, is physically impossible.

I wholeheartedly agree, from personal experience, with Fishberg (45) who says "I have never seen a lung, that had been in contact with pus in the pleural cavity for more than 3 months, re-expand under any treatment." Fishberg, of course, was not reckoning with decortication, but otherwise I believe his view is sound. Self cures of empyema, where the lung has been collapsed for several months, are due, as a rule, not to re-expansion of the organ so much as to a shift or pull of the mediastinum over to the diseased

side (especially on the left).

We see then the necessity for vigorous and thorough aspirations at an early stage in treatment; every week's delay in attaining lung re-expansion means more pleural thickening and less chance of permanent cure. Thus the treatment of empyemata by leaving them alone and only aspirating when there are pressure symptoms, will yield poor results.

There are other factors which determine a lung's non-expansion:-

(A) The extent of disease in the lung tissue itself.

(B) The size of the bronchopleural fistula.

A. Some lungs when they collapse in the face of rising intrapleural pressure and later pus formation are capable, after long periods, of re-expansion. (Post-mortem No.26 is a case in point). The only disease was a small caseous area at the apex of the right lower lobe through which the spontaneous pneumothorax presumably occurred. No bronchopleural fistula of moment now remained. Had it not been for the rind of thickened visceral pleura this lung was physically capable of expansion after the empyema had lasted for $4\frac{1}{2}$ months. Post-mortem No.24 is a different story. The lung had been collapsed by empyema for only three months, yet on section it was carnified and firm and quite incapable of re-expansion, apart from its rind of thickened pleura. The photograph from the case under discussion follows page 65A$_1$. and shows in its upper part an area of extensive fibrosis. Carbon pigment can be seen and areas of caseation (C). The point is that before the spontaneous pneumothorax was superadded to his right artificial pneumothorax he had cavitation of the right upper lobe and areas of mottling around. In the section on tracheo-bronchitis (Chapter III) I have already shown that the bronchioles and finer bronchi around such areas are usually diseased. The least alteration of alignment, as with a superadded spontaneous pneumothorax, blocks them, and the area of the collapsed alveoli dependent on

those bronchi becomes pneumonic. This is the process, previously described, which leads to the radiological "ground-glass" appearance in an artificial pneumothorax. Caseation can spread and areas of it are common. The above-mentioned photograph illustrates the final condition well. When the lung collapse is maintained the pneumonic lung organises. With an L.P. view the alveolar outlines persist for a long time, but an H.P. view reveals the numerous patches of new capillaries and leashes of fibroblasts growing in all directions. Thus the extent of the original pulmonary disease determines the lung's future chances of re-expansion too.

B. Broncho-pleural fistula.

The significance of large broncho-pleural fistulae is that aspiration leaving negative intrapleural pressures to encourage the lung to expand is not possible, and therefore this complication cuts down the chances of recovery with conservative treatment. The other important factor is that large broncho-pleural fistulae usually communicate with medium sized bronchi or with cavities, and the chance of there being secondary infection from the outset, with its increasingly bad prognosis, is great.

It is stated by some that in every case of empyema a broncho-pleural fistula is present (Fishberg (45), Coryllos (3), and others). The question is not just an academic one but is of the utmost importance. The smallest fistulae which communicate with alveoli may be sealed off or heal quickly, and the lung be capable of re-expanding as a negative intrapleural pressure develops or is helped to occur by aspirations of air and fluid. Such fistulae, as a rule, do not allow secondary infection from the bronchial tree.

There are several tests for broncho-pleural fistula in common use -

1. After aspiration of pus and withdrawal of air manometric intrapleural pressures are recorded. After a lapse of half an hour, say, the pressures are again taken. With fistula present high negative pressures are not maintained.

2. In some cases of large fistula, as where a cavity wall sloughs after adhesion section, the insertion of an aspirating needle through chest wall to empyema cavity causes a whistling sound from the fistula as the lung moves with respiration. In such cases the empyema contents may be expectorated with change of posture.
3. Methyline blue may be injected into the pleural cavity and will colour the sputum later. Sometimes it is decolourised, in which case the sputum will have to be shaken with H_2O_2 to regain the colour.
4. Ether vapour will likewise be detected in the nose if ether is introduced into the empyema cavity. I have observed that P.A.S. functions in a similar manner, in some cases, by its taste on the tongue.
5. A variety of chemical test which I have used is described by Rosen (139) and depends on the fact that sulphanilamides give an immediate and stable yellow reaction with Ehrlich's reagent. I have used 40 c.c. of soluble M. & B.693 into the pleural cavity and had the patient expectorate into a glass jar containing dilute Ehrlich's reagent. It is necessary to keep a control jar as the reagent itself is straw-coloured. The above tests (apart from No.1) are crude and only demonstrate fairly large fistulae. Moreover, they fail if a fluid level covers the opening.
6. Similarly, retrograde bronchograms, when lipiodol is introduced to the pleural space to outline the cavity, only form in some cases.
7. The most scientific means of determining the size and existence of a broncho-pleural fistula is by gas-analysis of the pleural air. This can only be done where the chest has not been intubated.

Davy (140) first published his "observations on air found in the pleura in a case of pneumothorax; with experiments on the absorption of different kinds of air introduced into the pleura" in 1823.

As previously described in the chapter on tracheo-bronchial tuberculosis, Coryllos and Birnbaum (86) and later Henderson and Henderson (87) described the mechanism of gas absorption from alveoli where there was bronchial obstruction, and the absorption of gas from any closed space within the body, respectively.

In 1932 Coryllos, Konterwitz and Levine (141) described for the first time a clinical application of gas analysis of pleural air. They point out that whatever the gas or mixture of gases introduced into the pleural cavity, provided they are non-irritant, after a short while, not exceeding 2 hours, O_2 and CO_2 are present in it in amounts depending on the partial pressures of the same gases in the blood circulating under the pleural serosa. In human artificial pneumothorax for tuberculosis the O_2 values were around 1 to 2% or less, while the percentages of CO_2 were around 8.0 to 10.0% or even 14% or more, especially if fluid was present. In the case of small broncho-pleural fistulae, the pleural gas will communicate with alveolar air (O_2.15%, CO_2.5% and N_2 80% + water vapours) and will approach more nearly to it. Closure of the fistula for a short time by fluid, etc., will be revealed by the changes occurring in the composition of the pleural air.

They give elaborate formulae for the determination of the amount of air in the pleural cavity and for calculating the absorption index of the pleura. Those calculations, as shown in a more scientific article by Matsuzawa (170), entail the reduction of all figures to a standard temperature and pressure, and involve considerable skilled experience, and are quite outwith the scope of a busy hospital where much routine work is done.

The third calculation possible, the diagnosis of spontaneous pneumothorax and its course, merely entails an analysis of the pleural gas O_2 and CO_2 calculated as a percentage and is easily done. Moreover, it is the procedure which is of interest to us in our study of empyemata.

Pleural gas analysis appears to have become a routine procedure in some American hospitals and Ulmar (10) in 1937 states that it is the surest way to determine the existence of a broncho-pleural fistula, but that a knowledge of pre-existing gas values in the particular case being studied, is necessary. Ornstein, Herman and Friedman (142) also noted that, although there were wide variations in the O_2 and CO_2 content of the pleural gases in different cases, the values were usually constant for the same person.

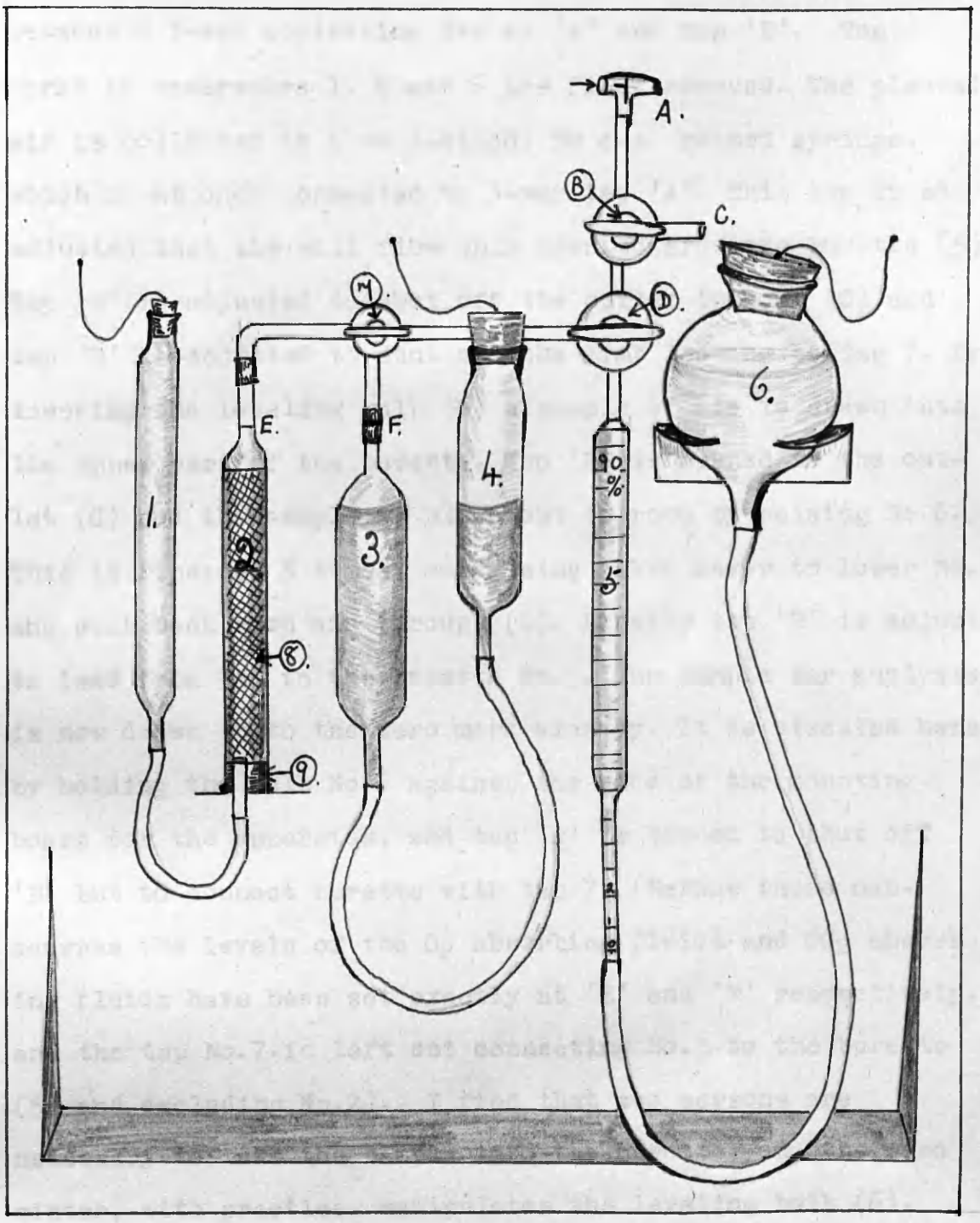
Finally, Coryllos (3) in 1937 gave his conception of tuberculous empyema. He states "in pulmonary tuberculosis the production of purulent exudate in the pleural cavity, or the change of existing clear effusions to purulent ones, is always due to the development of pleuropulmonary fistulae". He classifies fistulae into 3 sizes according to data obtained from gas analysis procedure -

1. Punctiform (under 0.5 mm.)
2. Small (less than 1 mm.)
3. Large (1 to 10 mm., or more).

The idea behind Coryllos' work is that the O_2 and CO_2 values will run constant in the air in an artificial pneumothorax until a leak into the alveoli or bronchial tree occurs. Then the percentages will alter, depending on the size of the leak. The larger the fistula the more nearly will pleural gas values approximate to atmospheric air, the O_2 percentage rising and the CO_2 percentage falling. It is only in the punctiform fistulae that there is any justification for conservative measures. To quote Coryllos - "Radical measures are not urgent in punctiform fistulae which may close, the fluid absorb, and the lung re-expand."

Unfortunately Coryllos' theory is all right as far as it goes but is wrong in one important respect - the condition of the pulmonary parenchyma - as I shall now show.

Some personal observations using pleural gas analysis:



Parts of apparatus:

1. Oxygen absorption reservoir containing concentrated ammonium hydroxide, ammonium chloride, and D.W. (a blue-coloured solution).
2. Oxygen absorption tube with the same solution as in 1 and, in addition, a roll of copper gauze.
3. CO_2 absorption tube containing potash solution.
4. CO_2 absorption reservoir.
5. Burette having upper (B) and lower (D) 3-way taps.
6. Leveling bulb.
7. 3-way tap.
8. Copper gauze (40 mesh) in O_2 absorption chamber.
9. Rubber cork.

The apparatus, designed to sample air in an oxygen tent, is modified by having the shortest possible distance between a 3-way aspirating tap at 'A' and tap 'B'. The corks in reservoirs 1, 4 and 6 are first removed. The pleural air is collected in a well-oiled, 50 c.c. record syringe, which is at once connected to 3-way tap 'A'. This tap is so adjusted that air will flow only down towards the burette (5). Tap 'B' is adjusted to shut off the outlet to room (C) and tap 'D' is adjusted to shut off the limb leading to tap 7. By lowering the leveling bulb (6) a sample of air is drawn into the upper part of the burette. Tap 'B' is turned to the outlet (C) and the sample is blown out to room by raising No.6. This is repeated 3 times, care being taken never to lower No.6 and suck back room air through (C). Finally tap 'B' is adjusted to lead from 'A' to the burette No.5. The sample for analysis is now drawn in to the zero mark exactly. It is steadied here by holding the bulb No.6 against the side of the mounting board for the apparatus, and tap 'D' is turned to shut off 'B' but to connect burette with tap 7. (Before those manoeuvres the levels of the O_2 absorbing fluids and CO_2 absorbing fluids have been set exactly at 'E' and 'F' respectively, and the tap No.7 is left set connecting No.3 to the burette (5) and excluding No.2). I find that two persons are necessary to take the sample into the burette, and the ward sister, with practice, manipulates the leveling bulb (6). Now the sample of air (10 c.c.) is connected to the CO_2 absorption reservoir No.3 and is passed back and forwards over the potash solution by alternately raising and lowering the leveling bulb (6). After about 10 movements the level of potash is brought exactly to the mark 'F' where it was at first, and holding the leveling bulb steady the percentage of CO_2 in the sample is read on the burette scale (5). Then, with the level of potash at 'F', the tap No.7 is turned to connect burette No.5 to reservoir No.2, and the process is repeated. The final O_2 percentage is got by subtracting from the final burette reading the figure for CO_2 percentage.

Before proceeding to test another sample the air in the tubes between tap 'D' and reservoirs 3 and 2 is exhausted of CO_2 and O_2 and the inert N_2 left there.

Several artificial pneumothoraces were observed. Refills were given one forenoon each week and pleural gas analyses were made 24 hours later, e.g.,

Case 1. H.C. (L) artificial pneumothorax of a month's duration.

30.1.48. CO_2 3.3%; O_2 1.5%. Then thoracoscopy and adhesion section. No fluid followed.

7.2.48. CO_2 3.6%; O_2 1.9%.

and so on until

6.3.48. CO_2 3.3%; O_2 2.0%.

This left artificial pneumothorax continues on 28.5.48 uneventfully and free from fluid.

Case 2. M.K. Right artificial pneumothorax of a month's duration.

25.1.48. CO_2 5%; O_2 1%. Adhesion section was then done.

31.1.48. CO_2 4.6%; O_2 1.4%. No fluid.

7.2.48. CO_2 4.1%; O_2 .9%.

14.2.48. CO_2 5.0%; O_2 1%. Merest trace of fluid in costo-phrenic angle.

27.2.48. Refill given and slight "staining" afterwards.

6.3.48. CO_2 4.9%; O_2 2.6%.

The artificial pneumothorax has been free from fluid and is proceeding uneventfully on 28.5.48.

Case 3. A.C. Left artificial pneumothorax of 2 weeks' duration.

31.1.48. CO_2 5.5%; O_2 1.5%.

7.2.48. CO_2 4.0%; O_2 1.5%.

14.2.48. CO_2 4.2%; O_2 1.8%. Then adhesion section on 16.2.48.

21.2.48. CO_2 4.4%; O_2 1.6%.

24.2.48. CO_2 4%; O_2 2.5%. Blood-stained effusion formed and aspirated.

5.3.48. Honey-like fluid removed. "Staining" afterwards.

6.3.48. CO_2 4.3%; O_2 5.2% (i.e., a small fistula present). The fluid ceased to form and the artificial pneumothorax was continued uneventfully.

15.5.48. CO_2 4.5%; O_2 1.9%. The fistula has healed.

Those, and other cases tested, run corresponding O_2 and CO_2 values from week to week. Any event out of the ordinary, such as the "staining" in case No.3, is followed by a corresponding rise in the O_2 value depending on the size of the fistula, and there is a return to previous values as the fistula heals. I confirmed that in the presence of purulent fluids the CO_2 values rise up to 8% or 10% or more, and I am of the opinion that in those cases it is the O_2 reading that is of value.

For the tests to be of much use it is necessary to know the general run of the O_2 and CO_2 values in the individual case concerned, especially before adhesion section.

Case 4. M.S. An Empyema.

This girl had a well established right artificial pneumothorax which suddenly developed a turbid fluid. At this stage, a few days later, a gas analysis revealed CO_2 6%; O_2 1%. She had no previous analysis figures for comparison but it was obvious from the low O_2 value that the fistula was insignificant and the prognosis was good. The fluid in a week was thin pus, but steady and careful aspiration cleared up the empyema and the lung re-expanded.

Case 5. K.D. An Empyema where the lung re-expanded.

A female case, admitted 12.6.47, R.A.2 classification.

Aged 29 years.

On discharge from army in January/1946 started to have bronchitis.

23.7.47 pulmonary lavage was positive and 2.8.47 laryngeal swab was positive. All other tests negative for tubercle bacilli.

Clinically scanty, scattered râles audible at apices. General condition good. Blood Sedimentation Rate 11 mms./hour. Trace of muco-purulent sputum each morning. Left artificial pneumothorax induced 16.9.47 and thoracoscopy 27.10.47 but no adhesions were divided. A trace of fluid appeared at the left base.

29.10.47 four ounces of straw-coloured fluid were removed

and the pleural fluid became purulent in the next few days. No tubercle bacilli seen on concentration. The artificial pneumothorax was abandoned.

Gas analysis 7.11.47 O_2 1%, CO_2 9%, i.e., no gross fistula present. Vigorous aspirations, leaving an intrapleural negative pressure, caused left lung to re-expand by 20.11.47. The gas analysis proof of a punctiform leak in the pleura was supported by intrapleural pressure readings, i.e., on 11.11.47 pressures after aspiration were -18 -8 cms.water and after 13 minutes -12-7 cms. water.

Bronchograms later revealed bilateral apical cystic bronchiectasis.

No.42. 9.9.47. Both lungs show an apical mottling. In the original film there are thin walled cavities suggesting the "soap-bubble" appearance of cystic disease. One such cavity is easily seen below and behind the left clavicle. Lower and mid.zones clear in each lung.

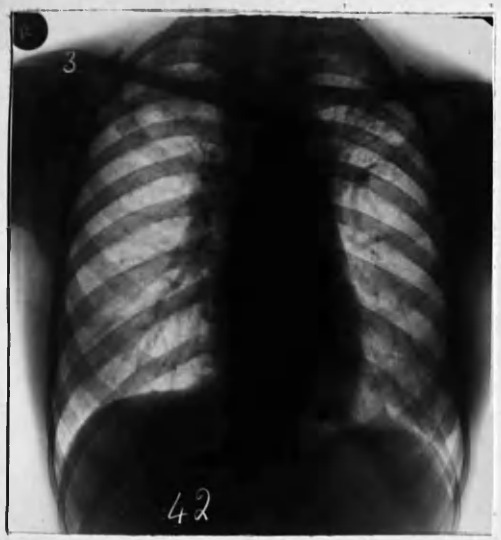
No.43. 4.11.47. The appearances are unchanged at the right apex. On the left side an artificial pneumothorax is present with a layer of fluid at the base and extensive apical adhesions. The cystic appearance at the left apex is accentuated and there is a fluid level at the upper limit of the artificial pneumothorax space.

No.44. 19.11.47. A left oblique view and an upper lobe bronchogram. This shows extensive saccular dilatation in the left upper lobe bronchi.

No.45. 19.11.47. This is a P.A. view and left upper lobe bronchogram. Mid.zone and lower lobe bronchi show no dilatation like the left upper lobe bronchi. The left lung has fully re-expanded.

No.46. 19.11.47. Left lateral upper lobe bronchogram. The dilatations appear to be in left apical bronchus and in the left sub-apical bronchus.

No.47. 26.11.47. A right P.A. vertical bronchogram to show the apex. There are saccular dilatations in the upper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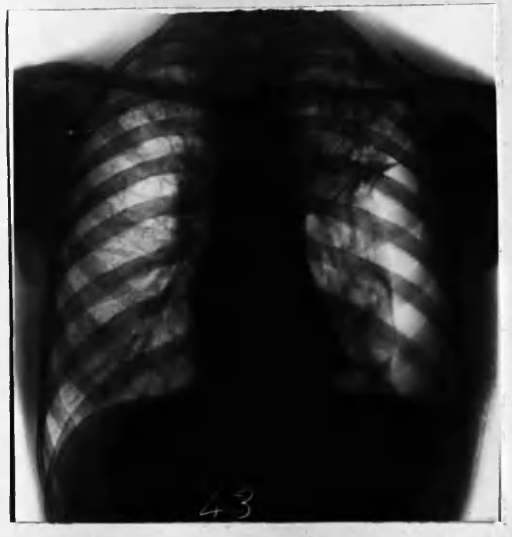
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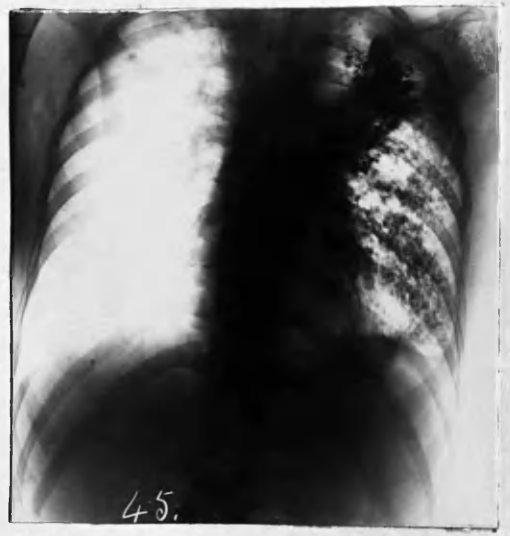
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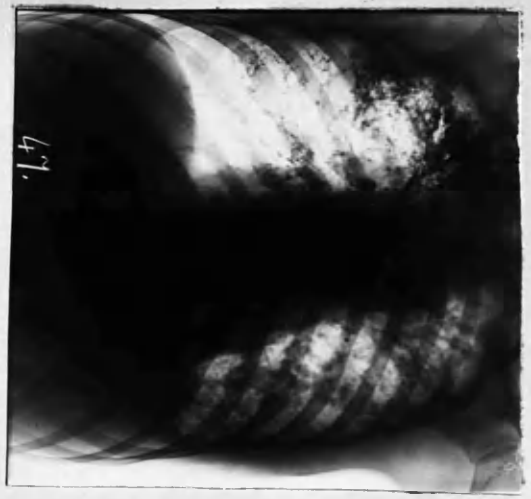
47.


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No.47 (continued):-

lobe bronchi. Note the evidence of old lipiodol mottling in left mid. and lower zones.

No.48. 26.11.47. A right lateral, upper lobe bronchogram. The saccular dilatations appear to be confined to right apical bronchus. Right sub-apical bronchus is possibly dilated at its origin but it has not filled well.

This case is interesting (a) the nature of the lesion is not common; (b) it is one of several I have seen where an artificial pneumothorax for bronchiectasis was followed by empyema and (c) the use of gas analysis revealed a punctiform fistula, and with vigorous aspiration the lung re-expanded in one month.

Case 6. V.C. An Empyema with healed fistula where the lung has not re-expanded.

This girl was admitted in mid-February/1948 having had a left spontaneous pneumothorax on 1.1.48. The left lung was flattened against the mediastinum which was displaced to the right. Much fluid was present on the left side. The right upper lobe had some active looking disease.

Left pleural gas analysis -

15.2.48. O_2 3%; CO_2 9%, two hours after aspiration.

21.2.48. (before aspiration) O_2 1%; CO_2 8%.

5.3.48. O_2 1.5%; CO_2 8%.

Those readings indicate that a small fistula has healed. This is confirmed by high negative intrapleural pressures being maintained between aspirations. The lung steadily re-expanded from the first aspiration but stopped short of complete re-expansion, and the pleura is very thick now. To date (20.7.48) about one ounce of thin pus forms each day; vigorous aspirations are done every third day, and 10 c.cs. of P.A.S. have been injected intrapleurally at each aspiration from the start. The lung shows no signs of re-expanding fully and unless aspirations are continued regularly tends to be further compressed by pus formation.

This case shows that even with a healed fistula the lung will not necessarily re-expand completely even with thorough aspiration to negative pressures. The pleura over the lung had thickened and bound the lung down before re-expansion was possible. X-rays show that there is minimal disease in this left lung. The thin pus obtained is teeming with tubercle bacilli on direct smear in spite of all her P.A.S.

Case 7. L.B. An Empyema with proved healed fistula and failure to expand.

This is the case described on page 114 (No.9) and post-mortem No.26. On admission the right pleural gas analysis was CO_2 14%; O_2 5.0%. There was pus present. Two weeks later aspirations to negative pressures were done and the negative pressure was maintained, confirming that there was no gross leak. The lung failed to re-expand and post-mortem revealed that there was no visible fistula but re-expansion was impossible owing to a rind of thickened pleura.

The conclusions from the above and many other cases are that pleural gas analysis is a valuable means of detecting a broncho-pleural fistula. I do not think it is a sufficiently delicate procedure to allow of the classification of fistulae into 3 grades as claimed by Coryllos. It merely shows small and large fistulae. The method, combined with the testing of intrapleural pressures after aspiration, indicates when small fistulae have healed, but this is no guarantee that the lung will then re-expand. Cases 4, 5, 6 and 7 just mentioned all had empyemata and small fistulae which had apparently healed. Yet in two of the cases the lung did not expand owing to the thickening of the visceral pleura progressing faster than the lung's outward movement. All the cases had comparable, thorough and regular aspirations.

Then again in cases such as No.8 (A.P., post-mortem 24) described later, and previously on page 114, who have much caseous pneumonic disease in the lung before it became atelectatic, carnification is apt to set in. In this case the post-mortem showed no evidence of any fistula, yet

the lung could never have re-expanded. The statement then of Coryllos that "radical measures are not urgent in punctiform fistulae which may close, the fluid absorb and the lung re-expand" is only partly true. If such cases show no expansion in two months at the most, in my opinion surgical measures should be considered at once. It is only those cases with punctiform fistulae capable of healing which will come into the small group of 17.8% of empyemata which heal by conservative means. Thus at the outset, in treating a case of empyema, a thorough investigation should be made of the size of fistula present, the type and bacteriology of the pus, A.P. and L. X-rays to determine the size of the empyema space, the amount of fluid and the mediastinal displacement, and the extent and type of tuberculous disease in the same and the contralateral lung.

Only in cases with the smallest fistulae is vigorous conservative treatment by frequent aspirations and perhaps saline irrigations justified, and then only for two months say. If the lung has not re-expanded by then the pleura will be progressively thickening to bind down the lung and its chances of coming out become steadily poorer. Only if the opposite lung's condition contra-indicates surgery on the empyema side will it be justified, in my opinion, to go on aspirating for months. Indeed in case 6 just reviewed, there is no alternative short of intubation, and the ultimate outlook, in spite of the patient's present good general condition, is grim.

The occasional good results obtained by A.J. Coello (130) with his auto-haemo-pleuro-therapy are probably explainable on the basis that the blood injected helps to seal off punctiform fistulae. In addition, Dr. Coello is keen on the method and therefore aspirates regularly. I am convinced that aspiration should be thorough, frequent and regular at the outset, and those personal views are confirmed by Howlett and Ehrenkrantz (143) who, dealing with pure tuberculous exudates in artificial pneumothoraces, by regular

aspirations twice or thrice weekly, obtained absence of fluid or pleural space obliteration in all but 3 of 34 cases. With haphazard aspirations good results were obtained in only 18 of 33 cases. Those, of course, were not purulent empyemata, but the principle is the same.

It only remains now to complete the discussion on the conservative treatment of tuberculous empyema by mentioning methods like oleothorax, and the instillation of P.A.S. and recent antibiotics.

Oleothorax:

H. de R. Woodcock (144) in 1913 first injected paraffin into the pleural space of cases of pulmonary tuberculosis to prevent pleural adhesions, but it was Bernou (145) who, in 1921 took up the method. Since then its uses have been mainly threefold. Oil has been used in the face of an obliterative pleuritis during artificial pneumothorax therapy in an attempt to prevent the lung from expanding. Again oil has been substituted for air from the start as soon as a satisfactory artificial pneumothorax has been obtained. This use of oil calls for fewer refills than with air and thus helps where the patient lives in a remote locality where refills are not available; or it may be useful where the patient is apprehensive over refills. The third use of oil is in the treatment of empyemata. Here 2% or 5% Gomenol is usually added to a sterile vegetable oil, such as olive oil or recently cottonseed oil. Gomenol is an essential oil from a species of myrtle, Melaleuca Viridiflora, and is an antiseptic.

Even in the healthy pleura much irritation is sometimes caused. Often a pyrexial upset attends the introduction of oil to the pleural cavity, and the method is especially dangerous in the face of string adhesions as the hydrostatic pressure of the oil alters its position with the patient's movements, with the danger of adhesion rupture, lung puncture and broncho-pleural fistula. The method, in this country, is

not much in vogue now and the concensus of opinion is that it is of no great use.

Keers and Rigden (102) say that they have never found oleothorax treatment of any benefit in pyopneumothorax, although it is sometimes alleged to diminish pus formation and reduce toxic symptoms.

Woodruff (2), whose definition of an empyema is 'any fluid containing tubercle bacilli on direct smear or any turbid fluid in the presence of pulmonary tuberculosis', found that oleothorax was of no value where there was secondary infection. He stated that it was of no value in empyemata without secondary infection where 11 out of 22 patients were well without further treatment. Of those, 3 had completely obliterated the space. Of the remainder there was still a possibility of perforation ahead. If those results are Woodruff's idea of an agent of value, it makes the case for surgical treatment of empyema stronger.

In 1946 Klassen, Miller and Curtis (122) found oleothorax the most favourable form of treatment of tuberculous empyema when compared with thoracoplasty or aspiration and irrigation. Their definition of tuberculous empyema is a "thick and turbid fluid which contains tubercle bacilli as shown by direct smear, guinea-pig inoculation or culture". 65% of patients so treated were well but there is no exact indication of the potential dangers in store for the cases and of the numbers of pleural spaces that really obliterated. In addition, the authors realise that the oleothorax cases were more favourable for treatment than the others.

Again in 1946 Browning, Ray and Rotenberg (146) give a follow-up of 75 cases which in 1941 were regarded as satisfactory after oleothorax. Those cases were all that had been satisfactory out of 48 cases treated for persistent tuberculous empyema and 53 cases treated for obliterative pleurisy. Of the 75 cases now under review six had a recurrence of empyema and 8 had developed broncho-pleural fistula. In 43 cases the oil was removed, replaced by air, and

attempts made to re-expand the lung with success in 16 cases only.

Meyer (147) in 1947 thought that there was a definite place for oleothorax in the treatment of empyema but had only treated 4 cases thus. The fluids were stated to have contained tubercle bacilli but it was not stated whether definite pus was present. In 2 cases the empyema cleared up while in the other two a thoracoplasty had to be done. The results of Moyer (148) also show that the method of oleothorax is no better than other conservative means of treatment. 84 cases of tuberculous empyema were treated and 43 showed a satisfactory result in a 5 years or longer observation period. There were 26 deaths. The presence of oil was directly responsible for complications in 11 cases, i.e., bronchopleural and pleuro-cutaneous fistulae.

The few cases of oleothorax that I have seen for the treatment of tuberculous empyema had thickened pleura which, with mediastinal fixation, I believe is the greatest bugbear in preventing final obliteration of the pleural space when conservative treatment is used.

Streptomycin has been discussed previously (page 109). I have had no experience with streptomycin in tuberculous empyema.

The Treatment of Tuberculous Empyema by 'NISIN'.

In November 1947 we were approached by the firm of Benger's Ltd., with a view to making clinical trials in pulmonary tuberculosis with the substance "Nisin" which they were manufacturing.

Nisin is a new antibiotic produced by the growth of streptoccus lactis (Group N. Lancefield). It originally was found to inhibit the growth of the "starters" used in cheesemaking. When the cause of this inhibitory action on "starters" was discovered, it was tested against various other organisms among which was the tubercle bacillus. In vitro a dilution of 80,000 to 400,000 was stated to kill bacilli tuberculosis and other acid-fast organisms. It is

active against many other organisms, e.g., some types of pneumonococcus, corynebacteria, erysipelotrix and actinomycis.

Lactobacillus Bulgaricus was shown by Rogers (149) to be inhibited by streptococcus lactis in 1928.

Whitehead and Riddet (150) then described the slow development of acidity in cheese manufacture. After Whitehead and Riddet's publication, Whitehead (151) partially isolated a substance which inhibited cultures of streptococcus cremoris in the form of cheese starter. He described the substance as a protein or polypetide.

Working on the substance 'Nisin', Mattick and Hirsch (152) in 1944 found it to be active in vitro against most of the groups of pathogenic streptococci, clostridia, and most of the groups of the lactobacilli. Staphylococci, in the presence of serum or blood, were very much less susceptible, and the authors found no gram negative organisms so far tested to be susceptible.

That same year (1944) Oxford (153) was working independently and described a substance called Diplococcin which was isolated from lactic streptococci. Diplococcin was active against a haemolytic streptococcus and other strains of lactic streptococci. There was some effect too, on staphylococci.

In 1946 Mattick and Hirsch (154) showed that tubercle bacilli in milk were killed by the concomitant growth of an inhibitory strain of streptococcus lactis, and they thought that this was a situation which might arise not infrequently in practice.

As originally delivered to us Nisin was a white powder in sealed glass ampoules. We were informed that Hirsch had ascertained that in a concentration of 1/250 Nisin had no effect on leucocytes. He also found that it limited the spread of experimental tuberculosis in guinea pigs. It remains stable for an indefinite time and can be boiled for one hour at P.H.2 for intramuscular use. The solution remains stable for months but should be stored in a refrigerator. For local

application it is dissolved in isotonic saline (1 ampoule in 1 c.c. being a strong solution). Each ampoule had 100,000 units. The units were arbitrary. Recently the Nisin has been supplied ready dissolved in solution in ampoules. It was suggested that for local administration a start should be made with 250,000 units, and this increased up to 1,000,000 for tuberculous laryngitis. The Benger's Company's medical officer had seen Nisin used locally on skin lesions and he stated that it benefitted impetigo.

Several laryngeal cases were sprayed three times a day with Nisin at Baguley Sanatorium as a start. At first 2,000,000 units in 20 c.c. saline were used and later 3,000,000 units in 20 c.c. were tried. In no case was there any undue discomfort felt by the patient. Careful observation showed that there was no noticeable effect on the laryngeal condition for better or for worse. Then a member of the staff had an intramuscular injection of 1,000,000 units and apart from slight pain there were no ill effects. The next step was to try Nisin intrapleurally and at first the contents of 1 ampoule were given daily (1,000,000 units). Careful watch on the patient's red and white cell count, Blood Sedimentation Rate, and urine showed that Nisin is well tolerated in the chest.

In one case a female patient showed some intolerance in the form of local pain, rise of temperature for 12 hours, and vomiting after her intrapleural injections, and the drug was discontinued after 3 applications. It is conceivable that those ill effects may have been due to traces of hydrolysed yeast from the culture medium used in the growth of the streptococcus lactis, and which were in the earlier samples given to us. The general method was to aspirate as much pus as possible from the pleural space daily and to leave in 1,000,000 units of Nisin. In each case the factor of frequent aspirations and negative pressures was also working towards cure.

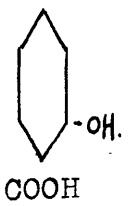
Dr. L. Parker of Baguley Sanatorium is doing the preliminary work with Nisin. His results on six cases of

tuberculous empyema show that it offers no advantages whatever over any of the other conservative methods of treatment used. It does not clear tubercle bacilli from the pleural cavity. In this connection it must be remembered that when a new remedy is being used aspirations tend to be more frequent and thorough than usual. This was the method adopted. Frequent aspirations were made, every day at first, and then 100,000 units of Nisin were instilled and later 1,000,000 units. The lung if it re-expanded did so no faster than in any other cases and pleural thickening was just as marked. In case No.8 (A.P.) Post-mortem 24, Nisin failed to prevent the development of large, evil-looking pleuro-cutaneous sinuses, and it did not help in their healing.

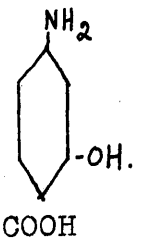
Para-Aminosalicylic Acid.

Lehmann (137) in 1946 reported that he began his work by concentrating on the observation of the American biochemist, F. Bernheim, that benzoic acid and salicylic acid specifically stimulate the respiration of the tubercle bacillus. Lehmann introduced various chemical groupings to the benzoic and salicylic acid molecules to inhibit this metabolic process which might in turn retard the proliferation of the tubercle bacillus.

One such compound was para-amino-salicylic acid (P.A.S.) where an amine group was introduced into position 4 in the salicylic acid, e.g.,



Salicylic acid.



Para-amino-salicylic acid (P.A.S).

Then laboratory investigations were made concerning the effect of P.A.S. on the growth of tubercle bacilli and Lehmann concluded that P.A.S. did not act by killing the bacteria but merely by inhibiting their proliferation. The actual destruction of the bacteria was left to the organism's own defences. Salicylic acid was claimed to stimulate the oxygen consumption of pathogenic tubercle bacilli only and not of

apathogenic. Continuous daily injections, intravenous and intramuscular, were well tolerated by rabbits but it was observed that P.A.S. when injected intramuscularly was very quickly excreted or destroyed. Clinical tests in humans followed and in mice and rats. No toxic effects were observed.

The drug was, however, seen to be a poison to the guinea-pig but it was thought that, even so, it displayed a protective effect against tuberculosis.

Again in 1946 Vallentin (138) published the results of human clinical trials with P.A.S. in pulmonary tuberculosis. Those experiments had commenced in 1944 at the suggestion of Professor Lehmann. Vallentin soon discovered that the drug is absolutely harmless. He observed no toxic effects on the blood or internal organs. In isolated cases only, was there slight and transient renal irritation in the form of mild albuminuria and occasional red blood cells and casts in the urine. Vallentin fixed an oral dosage of 14 grammes per day in 4 doses of 5, 3, 3, and 3 grammes for 3 or 4 weeks at a time with a week between the periods. For local use a 5 or 10% solution was used. The usual blood concentration by standard oral administration was 5 mg.%. Vallentin very properly observed that as P.A.S. is bacteriostatic in vitro it cannot, therefore, kill the bacilli in the body. The pathological changes in chronic phthisis are such that the drug cannot be expected to reach all the bacillary foci. Therefore one can never expect such a preparation to cure tuberculosis by itself but its use must be accompanied by the usual general treatment, including collapse therapy. The results of treatment with P.A.S. on various tuberculous conditions were summarised. Ten cases of pleural empyema were treated with improvement in four, no improvement in three, and death in three cases. The P.A.S. was injected as a solution into the empyema cavity. Vallentin does not give his definition of empyema. In some of the cases P.A.S. was also given orally. He could not record any really distinct result with local treatment and he observed that the anatomical circumstances were unfavourable. To conclude, Vallentin

made no extravagant claims for the remedy and was extremely non-committal in his views on its efficacy, stressing the need for much future work with it.

In 1947 Youmans, Raleigh and Youmans (155) reported that P.A.S. was highly bacteriostatic for virulent human type tubercle bacilli in vitro and that serum in the medium had no appreciable effect on this activity. The bacteriostatic activity of the compound (P.A.S.) was inversely proportional to the number of organisms in the medium, and one avirulent, rapidly growing, acid-fast organism had not been inhibited by 100 mg. per cent P.A.S. The authors found that experimental tuberculosis in mice was suppressed by P.A.S. when given in the diet in 1% and ½% concentration. P.A.S. and streptomycin when given to mice simultaneously appeared to exert a suppressive effect on the tuberculous process, greater than that of either substance alone.

Alin and Diffs (156) also reported their results in clinical trials with P.A.S. in 1947. They concluded that the drug was well tolerated in the doses mentioned above. Some patients had been observed to pass one or two loose stools daily. The writers summed up by stating that their observations justified continued investigation but did not permit of any other conclusion. They did not deal specifically with empyema.

Feldman, Karlson and Hinshaw (157) in 1947 gave an account of the effect of P.A.S. on experimental tuberculosis of guinea pigs. Contrary to the experience of Lehmann they found that the drug was well tolerated when given orally to guinea-pigs for a prolonged period. They concluded that P.A.S. was unmistakably able to exert a favourable influence on the course of tuberculous infections in guinea-pigs. Reading closely into the article, however, they state in one point that "it is true that several of the treated animals died with insufficient tuberculosis to account for death." Also a footnote states that from the tissues of several of the animals that died the virus of lymphocytic chorio-

meningitis was identified. The results of the tests are, therefore, open to doubt.

In December 1947 Dempsey and Logg (136) published early results with clinical trials of P.A.S. in 9 cases of tuberculous empyema. Three had open chest wall sinuses, one had a sinus and in addition a broncho-pleural fistula, and one had a broncho-pleural fistula with no sinus.

Their first patient had a chronic tuberculous empyema with a broncho-pleural fistula. There had been open drainage for two years before a thoracoplasty and then a further period of ten months when the fistula, sinus, and empyema cavity had remained open. A 5% suspension of the pure acid in water was inserted into the empyema cavity which was then sealed for three days, and this was repeated two weeks later. Within a month of the original injection of P.A.S. the chest wall sinus healed, the symptoms of broncho-pleural fistula vanished, and by X-ray the former empyema cavity could not be seen. Needling yielded no pus. Four months later a film showed no sign of relapse.

Another case showed healing of multiple sinuses and two further similar cases showed evidence of commencing healing. The remaining cases, according to the authors, have either been obliterated or are showing a definite tendency towards this end. The writers state that in these empyemata "radiological examination suggests a deviation from the usual mechanism of re-expansion and a proliferation of deposit (probably fibrinous) on the parietal pleura."

Apart from the fact that the authors give no definition of "empyema", their results are indeed remarkable, having excelled those obtained by most surgeons, and have stimulated many tuberculosis workers to try the drug. My own experience, using the sodium salt of P.A.S. on six cases, is that it has not been shown to benefit one of them. In accordance with the instructions issued by the firm (Herts Pharmaceuticals Ltd.) who supply P.A.S. in Britain, the pus has been aspirated as completely as possible and then 10 c.cs.

of 20% P.A.S. left in situ. This has been repeated every two days in most cases, which is more than was done by Dempsey and Logg. No constitutional upset was noted and no abnormality detected in the blood picture or urine.

A recent interview with a chemist, representing herts Pharmaceuticals Ltd., suggested that other workers are also having poor results with P.A.S. used locally in empyemata. This, according to the chemist, is because there is no oral administration of P.A.S. at the same time. However, the oral drug is in too short supply for general distribution and there is already a world-wide demand for the drug. Recent work by the makers shows that the sodium salt of P.A.S. is rapidly absorbed and almost all out of the pleural cavity within 2 hours of its injection. The brown colour of the solution supplied is an impurity, and because this colour remains it must not be confused with the P.A.S. proper.

On hearing those facts I set up an experiment with viable tubercle bacilli from a progressive case of pulmonary tuberculosis. A slope culture was heavily inoculated and a thick emulsion made with the resulting 3 week old smear. This was smashed up with beads and then centrifuged; the supernatant fluid was removed, replaced by pure 20% P.A.S. in the form of sodium salt as supplied for intrapleural use, and shaken. At varying intervals (quarter hour, half hour, one hour and two hours) 1 c.c. of the mixture was taken, centrifuged, and the deposit washed with saline and then plated on Loewenstein Jensen medium. The tubercle bacilli grew in both the one hour and two hour specimens that were in contact with P.A.S. This represents closer contact with a stronger P.A.S. solution than they would have in the pleural cavity.

Like Vallentin (138) I cannot see how the tubercle bacilli in subpleural caseous areas, often with endarteritis in the associated vessels, can be affected by the drug, and I am firmly convinced from my own experience that the expensive P.A.S., as with other conservative methods, is not

the answer in the treatment of truly purulent tuberculous empyemata. The problem is largely a mechanical one, and, as results with all the means of conservative treatment described above show, we must turn our attention to the surgical treatment of the condition.

The statement of McNally (158) on chemotherapy in relation to tuberculosis sums up the situation with regard to the conservative treatment of tuberculous empyema, i.e., until an effective chemo-therapeutic agent is found, the thoracic surgeon is still the person who will treat the condition.

The drawback with Nisin, P.A.S.,etc., is that they are acid in reaction while the body fluids are on the alkaline side of neutrality. Antibiotics, on the other hand, usually attack organisms when they are dividing. The tubercle bacillus, in its caseous focus, has merely to assume a resistant, non-proliferative, phase until the drug has been excreted or discontinued.

SURGICAL TREATMENT.

Most operative procedures for tuberculous empyema are undertaken when conservative methods of treatment have had an extended trial, often over many months or even years, and have failed. A minority of surgeons operate on tuberculous empyema within a few weeks of its onset. It is proposed to discuss the two views.

All cases of tuberculous empyema which have had operative treatment in Baguley Sanatorium have been of the long-standing variety (with the possible exception of case 8F). In the series under review 15 cases were thus treated. (See table 16 and the case histories).

TABLE 16.

Operation case numbers.	Duration of empyema before operation.	Total empyema. Duration before recovery or death.	Result and outlook.
2C	36 months	44 months	Death.
3A	15 "	144 "	Full recovery.
6A	48 "	108 "	Recovery.
8D	60 "	108 "	Persisting sinus on 3.4.48, otherwise well and in good general condition.
8F	5 "	13 "	Death after marked contralateral spread of disease and empyema persisting.
8G	48 "	108 "	Death.
8L	114 "	132 "	Death.
9B	60 "	72 "	Probable closure of space on 3.4.48. General condition only fair.
9C	36 "	46 "	Death.
9H	32 "	73 "	Outlook bad. On 3.4.48 has sinus and residual empyema. Oedema legs and albuminuria.
9L	10 "	16 "	Death.
10J	6 "	26 "	Recovery.
10K	44 "	52 "	On 3.4.48 patient is deteriorating. Outlook bad. Empyema persists with sinuses.
10.0	10½ "	63 "	Death.
10Q	20 "	37 "	Death.

A. Of the 15 cases treated by operation there were 8 deaths (53.3%), the average duration of empyema being 57.4 months.

B. There was full recovery without sinus in the chest wall in 4 cases (26.7%) - 3A, 6A, 9B and 10J).

C. Cases who had improved as regards general condition but in whom the sinus persisted number one (6.7%) - 8D.

D. In two cases the sinus persists and the general condition is deteriorating on 3.4.48 (13.3%) - 10K and 9H.

Therefore the good results with surgery amount to 33.4% (class B and C) while there is death or impending death in 66.6% (A and D). This is an improvement over the results with conservative treatment alone (Table 15) where the deaths or impending deaths are 82.2% and the cases with recovery only 17.8% of the total. Those results also correspond to fourteen cases I have seen recently in old-standing cases of empyema treated by surgery and not in the series under review.

Surgical measures for the cure of tuberculous empyema can only be undertaken if the state of the opposite lung will permit and if the general condition of the patient will allow of operative interference. It is a common event in bad choice thoracoplasty cases to see, after operation, a flare-up or extension of disease in the opposite lung. This may be due to a bronchogenic spread of disease from the operation side, (a) if the cavity which is to be compressed is of the "giant" variety or (b) in the case of empyema, where there is a broncho-pleural fistula present, and there has been no intubation prior to the thoracoplasty or inadequate aspiration between the stages. The fresh disease on the opposite lung may also be due to a true recrudescense of previous tuberculous foci which, prior to operation, were in a quiescent or latent phase. Those flare-ups are helped by a poor general condition of the patient after operation. In the series of cases under review it was possible from X-rays and dossier notes to assess the condition in the opposite lung at the time of onset of empyema in 78 of the cases who did not have operative treatment.

In 54 cases operative treatment was precluded from the start owing to contralateral disease. (In 17 cases this took the form of soft mottling, while in 35 cases there was cavitation as well. Two cases had an artificial pneumothorax present with airless lobe and no definite cavity). The contralateral lung was clear and considered safe for surgical interference on the empyema side in 24 cases. (It must be

stressed that in 5 of those cases (4J, 7F, 7H, 9I and 9P) there was some doubt of the stability of hard mottling present, and in another case (10R) there was marked, contralateral, bronchogenic spread of disease four months after the first X-ray).

Those figures show that, in spite of our best intentions, a large proportion of cases of tuberculous empyema (approximately 58% counting the 15 cases who had operation) are unfit for any but conservative treatment on account of contralateral disease. I include simple rib resection for intubation, with the conservative forms of treatment. Knowing the bad results with conservative treatment this figure envisages a poor over-all recovery rate from the outset, in spite of the best surgical efforts, when we are dealing with a hospital population in the R.B.2 and R.B.3 groups. In the ideal state of affairs, when empyemas follow on artificial pneumothoraces induced for unilateral disease, of course there would be a larger proportion of cases fit for surgery.

The reasons for failure with the operative treatment in long-standing cases are numerous. The successive severe stages of the operation leave their mark on some patients who either refuse to go on to the completion of the operation or are otherwise in too poor a condition for its completion. Some cases have a spread or flare-up of disease on the opposite side. The large majority of cases end up with a residual empyema space or a long sinus. There is no doubt that many are improved after operation, even with a small residual space, because they are relatively better off by virtue of a diminution in the large pleural surface from which septic absorption formerly took place. On the other hand, I have seen many such cases who lingered on, often with external sinuses, sometimes malodorous and necessitating daily dressings. They succumbed to slow toxaemia in the end.

<u>The use of a chemo-therapeutic "umbrella" during operation.</u>

Even though chemo-therapeutic agents, used intra-

pleurally, do not help the lung to re-expand, it has been suggested that they may be exhibited before operation to act as an "umbrella" to borrow a military phrase. It is thus hoped to prevent the spread of disease to the opposite lung following operation or by attacking the intrapleural organisms to lessen the risk of wound or extrapleural space infection.

In the case of a tuberculous empyema associated with secondary organisms sulphonamides or penicillin are useful, as I have often observed, provided the organisms are sensitive to those drugs.

In America streptomycin has been used as an "umbrella" before thoracoplasty and lung resections, but recent articles suggest that it is not finding universal favour.

My experience with P.A.S. intrapleurally indicates that it is of no use even to temporarily free the pleural fluid of tubercle bacilli (see previous cases).

Dr. Parker's results with Nisin in this Sanatorium show no improvement over what one would expect with thorough aspirations and saline washouts.

One class of organisms, the gram negative bacilli, are unaffected by penicillin or sulphonamides, e.g., Bacilli Pyocyaneus, Bacilli Coli and Bacilli Proteus, which latter is difficult to eradicate and causes an offensive type of pus. I was able to obtain from the firm of May and Baker a supply of the new drug Dibrompropamidine Isethionate (M.& B.1270) for testing its action on the secondary invaders in tuberculous empyemata. This drug has been used with success in killing gram positive and gram negative contaminants of skin grafting operations in the maxillo/facial and burns department of Baguley Emergency Hospital. In the autumn of 1947 I first obtained 4 grammes of M.& B.1270 from the firm's chief medical officer. Laboratory tests were made and it was found that the growth of Bacilli Proteus in a fluid medium was inhibited by a 1/2,000 dilution of the drug. Growth occurred

in 1/3,000 dilutions and above. The strain of Bacillus Proteus used was from the empyema pus of case (A.W.No.20,525). Low concentrations encourage the organisms to develop into drug resistant strains as we observed. Another point of note is that some coagulase positive staphylococci can be transformed to an avirulent type similar to a staphylococcus albus according to the makers.

In order to get a sufficient concentration in an empyema cavity 4 grammes were first tried on 17.12.47 (see case A.W. below). This was the first occasion on which this preparation had ever been used intrapleurally. The firm's medical officer had seen it used successfully in appendix abscesses in the dosage we first gave. The preparation is a diamidine, which has been developed and used for protozoal diseases abroad. As a general rule the maximum dosage of a diamidine is 4 mg. per kilo. of body weight intravenously.

Case A.W., 20,525.

Admitted 7.11.46. Weight 10st. 13½ lb. R.B.2. Aged 21 years. Bus conductress. ? Contact of father who died of chest disease (type not known).

Well until November 1945 when cough developed. First X-rayed January 1946. First hospital X-ray 13.11.46. Blood Sedimentation Rate 19 mms. per hour.

29.11.46. Right artificial pneumothorax induced with some difficulty.

7.2.47. Right Thoracoscopy and Adhesion Section.

Refill of 600 c.cs. Two punctures (axillary and scapular). There was a large, membraneous folded adhesion and three small cord ones at the apex. All were divided by enucleation. The operator left a small adhesion attached to the upper mediastinum purposely in order not to let the entire upper lobe collapse en masse.

After this the cavity remained patent and the sputum was still positive up to 18.4.47.

On 18.4.47 it was decided that a right phrenic crush would help and this was done on 5.5.47. Meanwhile fluid had appeared and 30 ounces of clear yellow fluid were removed on 2.5.47. Laboratory report "a slightly opalescent fluid containing a small amount of blood and a blood clot. Lymphocytes predominate in dried preparations. No organisms seen. Cultures remain sterile".

2.5.47. Pneumoperitoneum induced.

6.6.47. Last artificial pneumothorax refill. Then followed regular aspirations of fluid and progressive deterioration in the patient's general condition. By 21.6.47 she was seriously ill and the empyema could be said to be definitely present on that date. Blood Sedimentation Rate 21 m.ms./hr. Laboratory report of 5.7.47 on chest fluid -

"A purulent fluid. Tubercle Bacilli not seen. Considerable numbers of gram positive cocci in direct smears. Culture - a profuse growth of staphylococcus aureus (coagulase positive) has developed."

On 16.7.47 a diphtheroid bacillus was cultured and small members of tubercle bacilli were detected. The patient remained ill and toxic.

On 16.7.47 she was seen by me and I withdrew the air from the pneumoperitoneum to lower the right diaphragm. The lowest point of the empyema space was then drained in the anterior axillary line by means of an intercostal de-Pezzer catheter. A kidney dish full of thick, offensive yellow pus flowed out.

The lung began to re-expand and the tube drained well. Since 28.8.47, however, the lung expansion stopped. She had up to this time closed, under-water seal, drainage. Patient's general condition improved with tube drainage and her temperature fell to normal limits with occasional slight elevations.

Sinograms of 20.11.47 showed the extent of the pleural space. The lung was adherent to diaphragm below, but elsewhere was free from the thoracic cage, apart from the extreme apex. Ether run into the pleural cavity was faintly detected in the patient's mouth, suggesting the existence of a small broncho-pleural fistula. The site of insertion of the catheter to the chest was wrapped round with vaselined gauze to make an airtight joint and by an electric suction pump continuous drainage was applied to the empyema cavity from 20.11.47. An average of four ounces of pus was daily removed with great benefit to the patient's general condition. The patient altered her position regularly to ensure maximum drainage. Screening and X-ray photographs, however, showed that the lung was stationary and would not expand further. Laboratory report 4.12.47 - "Direct smears show cell debris, pus cells, gram negative bacilli and gram positive cocci. Tubercle bacilli not seen. Culture - Bacillus Proteus and Staphylococci have developed."

5.12.47. 4 grammes of M.& B.1270 in 10% solution was run into the empyema, the tube clipped, and left in position for 24 hours, and then suctioned off by electrical pump.

7.12.47. Report on fluid - "A purulent fluid. Tubercle Bacilli not seen. Culture - a few colonies only of Bacillus Proteus have developed. Approximately 99% of the viable organisms have been killed."

8.12.47. Laboratory report - "Bacillus Proteus grown. The number of organisms is greater than these in specimen of 7.12.47 but less than in specimen of 4.12.47".

The patient vomited several times for 2 days after this but soon became normal again.

16.12.47. Pus now described as "a thick purulent fluid. Tubercle Bacilli not seen. A large number of Bacillus Proteus and a small number of Staphylococcus Aureus have developed in culture."

17.12.47. M.& B.1270, 4 grammes instilled and left for 24 hours.

18.12.47. Chest emptied by pump and further 4 grammes of M. & B.1270 instilled.

19.12.47. Chest emptied by pump.
Patient had vomiting daily from now on (slight at first).

20.12.47. White Blood Cells 11,400/c.m.m. "The pus is more fluid than the previous specimen. Cultures demonstrate the presence of a very small number of Staphylococcus Albus and Bacillus Proteus.

21.12.47. 4 grammes of M. &B.1270 left in chest for 24 hours and removed.

22.12.47. "Material very fluid. 24 hour culture - no organisms grown."

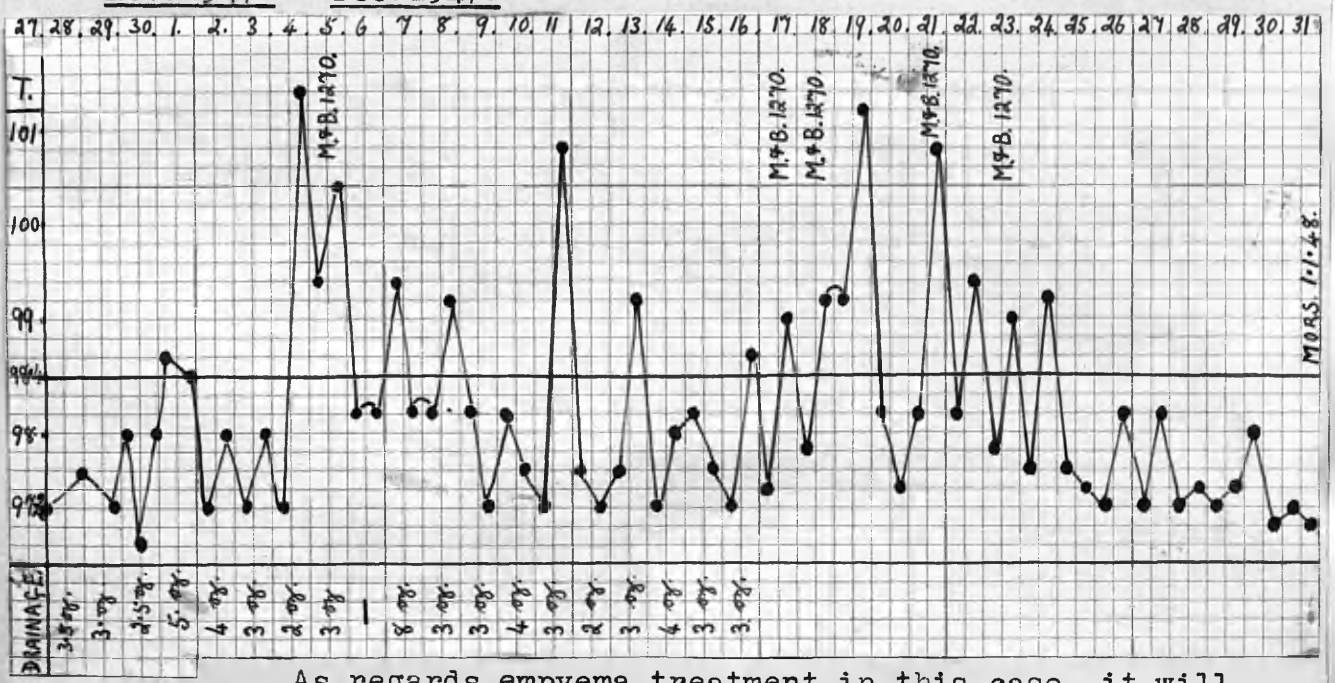
23.12.47. 4 grammes of M.&B.1270 left in chest for 24 hours and removed. (Total of 20 grammes M.& B.1270 in all). The patient now began to have troublesome sickness daily.

31.12.47. She was X-rayed. Patient was drowsy and had vague abdominal pains (right iliac fossa) and pains in the chest. No icterus visible. White Blood Cells 48,900/c.mm. (Normal differential count). Trace of albumen in the urine.
An enema showed putty-coloured stools. It was felt that she was developing acute yellow atrophy of the liver. She suddenly lapsed into unconsciousness in the early morning of 1.1.48 and died an hour later. Post-mortem (No.13) confirmed the presence of acute yellow atrophy of the liver.

Remarks: At the initial Jacoboeus operation all divisible adhesions were not divided. The artificial pneumothorax was maintained for two months, with negative pressures, in spite of a vomica in the right upper lobe still being visible at screening and on X-ray photographs. This cavity only finally closed when a massive empyema developed and the lung became

bound down by thickened visceral pleura. The cavity comes into category of a large peripheral one and it is doubtful if artificial pneumothorax should have been done).
The disease progressed to cavity formation in the left lung.

There is little doubt that the M.& B.1270 was responsible for the final issue. However, on 4.12.47, the day before the first dose of M.& B.1270, the patient did develop pyrexia of 101.4° fahrenheit. (See chart). This pyrexia settled by 6.12.47. Such temperature elevations are common in sputum positive cases where a tenacious mucous plug temporarily obstructs bronchial drainage. It is just possible, though I fear unlikely, that she was developing liver damage, apart from the M.& B.1270 preparation.



As regards empyema treatment in this case, it will be seen that frequent aspirations failed to stop pus formation or to make the lung expand.

Catheter drainage to a water seal alone had no effect on lung expansion after an initial slight expansion. Continuous suction by electric pump when the empyema had been established for five months caused the lung to expand very slightly, and it quickly gained a stationary position. The post-mortem showed that only decortication of the thick rind of visceral pleura would allow the lung to expand. At this stage too, sterilisation of the pleural contents will not aid expansion as mechanical factors now prevent lung re-expansion.

Although on 22.12.47 no Bacillus Proteus was grown from the pleural pus, the organism was recovered from the curd over the lung at post-mortem.

The analysts of the firm of May & Baker demonstrated M.& B.1270 in the liver by spectral analysis after death but were unable to say in what total concentration. There had, up to then, been no post-mortem on a case who was receiving the drug parenterally for Kala Azar so that at present we do not know the normal liver concentration of this drug, if any.

CHICK-EMBRYO CULTURE.

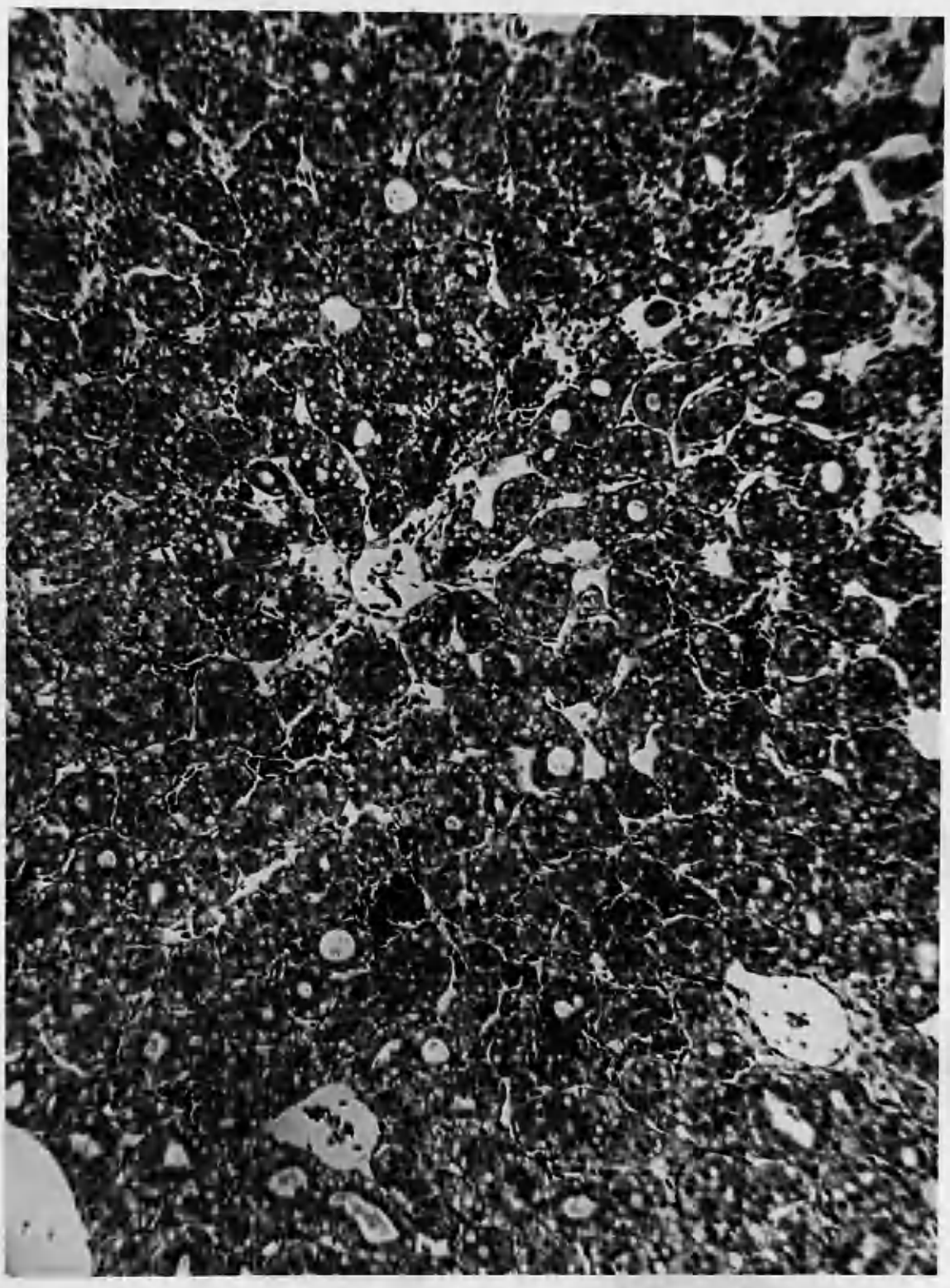
At this time I was working on the culture of tubercle bacilli on developing chick-embryos and decided to try the effect of M.& B.1270 on them. (Cuthbert and Davidson (159)). Various dilutions were tried*; .03 mlms. of 1/2,000 solution and .03 mlms. of 1/1,000 solution both produced a yellow, fatty looking liver. The latter killed the embryos in 24 hours with haemorrhages into the peritoneal cavity. The weaker dilution allowed the developing chick to live for 7 days when it was killed by me. Photo-micrographs of the liver from the human case and from a chick-embryo, which lived for 7 days, are attached for comparison. Both show fatty toxic changes and this suggests that M.& B.1270 has hepato-toxic properties and must, therefore, be used with care in a pleural cavity. The dose applied to the egg is, of course, a different concentration to what one might expect would reach the liver when the drug was put into a human pleural cavity. Also the delicate, developing chick liver would be almost certain to respond to any sort of toxic substance by fatty changes.

As the drug seemed to be of great value because of its ability to kill gram negative as well as gram positive organisms, it was cautiously tried on two other cases.

* By chorio-allantoic route.

Human Liver.

Negative magnification approx. x 300.
Final magnification. approx. x 900.



Chicks Embryo Liver. Negative magnification. approx. x 300.
Final magnification. approx. x 900.

Case L.H. 18,579.

This was an old-standing left empyema with drainage tube and secondary infection with B. Pyocyaneus, Staphylococcus Albus and a diphtheroid bacillus. Tested against B. Pyocyaneus, the first test on 7.1.48 showed that the organism was not killed by 1/1,000 dilution of the drug in a suspension.

20.1.48. Using dilutions of the M.& B.1270 in the medium, the organism was killed by 1/1,600 but not by 1/3,200. A third test was done on 21.1.48 for ascertaining the time necessary for the drug to act. The drug was not incorporated in the medium but the organism was kept in contact with a solution of M.& B.1270 and then loopfuls were cultured. 1/3,200 did not kill the B. Pyocyaneus in two hours and no stronger dilutions were then made.

Test 4 - 24.1.48. In 12 hours 1/2,000 killed the organism but 1/3,200 did not kill it. Those results were checked for the strain of B. Pyocyaneus in question.

Case History: Admitted 28.8.47 with left mixed infection, tuberculous empyema. Left artificial pneumothorax was induced 31.7.42 and patient was discharged. This had been a contraselective left artificial pneumothorax, and the resulting empyema was accompanied by broncho-pleural fistula. 30.9.47 left intercostal drain instituted. M.& B.1270 therapy commenced 16.1.48. Two ounces of 10% solution used on alternate days as a washout only, at first. From 11.2.48 the solution was retained for half an hour. On 19.2.48 the M.& B.1270 was stopped as she had nausea and sickness, and complained of pain over the liver. There was no jaundice. White blood cells were 15,300/cmm., which was its usual value. On 18.2.48 a growth of B. Pyocyaneus and diphtheroid organisms was obtained but no staphylococcus albus. This latter organism later re-appeared, Blood Van de Berg test was negative.

In this case I considered that the drug had been pushed to the limits of her tolerance.

Case M. McC. No.20,207.

This, too, was an old-standing case of mixed infection tuberculous empyema with a tube into the pleural cavity. The organisms were tubercle bacilli and bacillus proteus and a non-haemolytic streptococcus. Tests indicated that the strain of bacillus proteus here was killed in 15 minutes by 1/1,000 dilutions of M.& B.1270 but not by 1/2,000 dilutions even after 2½ hours contact.

Case history: Admitted 8.10.45. Left artificial pneumothorax 12.2.46 and left phrenic nerve crush 9.4.46. This was always a contraselective artificial pneumothorax with visible cavity and adhesions. Superadded left spontaneous pneumothorax on 28.12.46 while on Christmas leave and empyema developed with secondary infection.

24.2.47 intercostal stab drainage and Azochloramide washouts regularly with no improvement in the condition.

29.2.48 M.& B.1270 therapy commenced and the drug was given every two days for two weeks, i.e., four ounces of 10% solution which was kept in the pleural cavity for two hours. At the end of the period there was no effect on the patient's general or local condition and no change in the bacterial content of the pus which, at the end of the test, continued to yield tubercle bacilli, non-haemolytic streptococci and bacilli proteus. The while blood cells count at the commencement of the M.& B.1270 therapy was 28,500 and it remained around this level.

The drug in question is of great value for eliminating gram positive cocci. The three cases above, however, were sufficient to prove that as regards the elimination of gram negative cocci its use is attended with some danger, and the organisms cannot be banished from the pleural cavity within the limits of safety of the preparation. In all the cases the patients had their positions changed to spread the compound over the pleural surfaces. One of the causes of failure to kill all the organisms is probably a mechanical one - in the volume of solution used all the crevices and debris cannot possibly be probed by the drug.

Apart from the use of sulphonamides or penicillin for certain organisms, my endeavours to find another suitable chemo-therapeutic "umbrella" for operative inferference with the other remedies at my disposal have yielded, on the whole, disappointing results.

Outline of Operational Methods:

The cause of failure to cure chronic tuberculous empyema is the presence of greatly thickened parietal and visceral pleura. The tiling of ribs and the difficulty of removing them owing to fibrosis of the intercostal spaces, makes the various operations technically difficult. Only in rare cases will extensive thoracoplasty alone cure empyema cavities. As a rule a portion of empyema cavity, shielded by thickened pleura, remains patent and a final stage is necessary where some form of plastic operation is performed.

Delorme and Fowler first introduced decortication of the lung. The visceral pleura is cut through in a series of vertical strips, down to lung tissue. Those incisions are crossed by transverse cuts and the various intervening segments of thickened visceral pleura are removed by blunt dissection. The idea is that a healthy lung will always re-expand if it is unfettered, even though it has been collapsed for a considerable time. As I have previously shown, a diseased lung can quickly undergo carnification (within one month in some cases) and decortication would not help. The operation carries the risk of haemorrhage, air embolus, and the blood spread of tubercle bacilli to the meninges.

Gurd (160) recently described three successful cases of tuberculous empyema treated by decortication, but Meade at the discussion following Gurd's paper drew attention to several cases of widespread tuberculosis following the procedure. Dick (161) also stresses the risks of decortication and states that the procedure should be abandoned. Gurd advises that the lung be inflated following operation to help it to re-expand. He also states that where there is disease of the upper lobe alone, decortication should be done in the lower areas and the upper chest wall should be

collapsed by a thoracoplasty.

Joseph Ransohoff, an American surgeon (1853 - 1921), devised his operation of discission of the visceral pleura as a substitute for decortication. Grid iron incisions are made through visceral pleura down to lung tissue to allow of re-expansion of the lung. The method does not appear to have met with much success.

Jakob August Estlander, a Finnish surgeon (1831 - 1881), devised his operatiom which was used especially for small, flat empyema cavities. A series of incisions were made, parallel to and between adjacent ribs. Those gave access to the ribs above and below each incision. Those ribs were removed with a little more of the rib posteriorly and anteriorly than lay over the cavity. A large drainage tube was left in the extreme lower end of the space and changed every 3 or 4 days. Varieties of this operation are in use where a vertical incision is made, a skin and muscle flap elevated, and the ribs removed by this exposure.

For larger empyema cavities the final tent-like effect produced by the leathery, thickened parietal pleura over the cavity must be reckoned with. Hedblom (162), that authority on empyema, recognised this fact.

Max Schede, a German surgeon (1844 - 1902), devised an operation to deal with the hindering, parietal pleura. At the initial stages portions of upper ribs are usually removed and the regeneration ring of bone around any existing sinus. The empyema cavity is drained by a tube in its lowest part. A curving, vertical incision is then made over the residual cavity and the skin and muscles raised in two flaps from the ribs which are all removed over and a little beyond the limits of the residual cavity. The parietal pleurae and intercostal structures are removed from the roof of the cavity and the flaps of skin and underlying muscles allowed to fall inwards and stitched together. A drainage tube is left in the lowest part of the wound. The cavity should heal by granulation tissue, which is a long and

delayed process. In some cases, as in case No.4 (J.A.R., No.18,322), a partial scapulectomy is performed to allow the skin and soft tissues to fall into place easily. In other cases pockets of pus remain and supplementary operations are necessary to eradicate them.

Mr. H. Morriston Davies (163), when he finds a bronchial fistula communicating with a large empyema cavity, excises the margins and approximates the walls with a catgut purse-string suture and a second row of Lembert sutures.

Grow (164) has described a method used with success in four cases of tuberculous empyema where ribs over the cavity are resected but the intercostal bundles separated and preserved. The roofing parietal pleura is completely removed and the intercostal bundles replaced. This is obviously a modification of the Schede operation.

Many other operations have been devised to deal with the thickened, parietal pleura, among which should be mentioned that of Roberts. After decostalisation over the empyema cavity the parietal pleura is left. It is cut through, into empyema cavity, at the lateral margin of the empyema space all the way round. Posteriorly, however, where the visceral and parietal pleurae fuse at the costo-vertebral gutter, a long wedge of pleura is removed and the parietal, pleural flap allowed to swing inwards on to lung surface. As a rule a gauze pack is kept between the pleural layers for ten days and then removed, when the surfaces to be opposed should consist of granulation tissue.

Mr. Graham Bryce, at Baguley Sanatorium, treats each case on its merits. One method used by him with success, in smallish, localised cavities, is to decostalise the parietal area over the cavity. The vertebral aspect of the parietal pleura is incised and a wedge removed from the lateral and deep border of the cavities' parietal pleura (the opposite of a Robert's operation). The parietal pleural flap falls in and the latissimus-dorsi, trapezius, and other muscles, cut near the spine, are stitched into a new position, deep

to the erector spinae. This is an attempt to get rid of the cigar-like, residual empyema cavity which so often remains along the region of the paravertebral gutter and which a true Robert's or Schede operation does not always cure. Case No.10, H.J.M. (20,640) is a case of such an operation where the principle of stitching the spinal border of the trapezius under the erector spinae was tried. The man's wound and muscle layers are now widely septic, as so often happens in those extensive operations on tuberculous empyema. Local P.A.S. has been of no use in clearing the infection.

Other cases treated by the use of muscle flaps have had good results here. In some cases pedicled muscle flaps have to be used to obliterate the space or to block a broncho-pleural fistula. Where a permanent drainage is desired an Eloesser flap operation may be used. A 'U' shaped incision is made over the lowest part of the cavity and the skin and soft tissue flap elevated. The flap should be a little below the rib to be resected. Three or four inches of rib are removed and the flap turned inwards and stitched to parietal pleura, the lower edges of the wound are undercut and the skin edges brought together as well as possible up towards the opening. A drainage tube is left in situ until the skin edges that are in apposition have healed. Such an operation only allows for better drainage and lessens toxaemia. It rarely leads to cure of a tuberculous empyema.

The above is a brief outline of the principles of the operations on long-standing tuberculous empyema. The results and prognosis are always worse in the case of secondarily infected empyemas. It is important, of course, that before thoracoplasty operations, either an anterior tube be introduced (well away from posterior incisions and this if also anterior to the line where skin meets mattress, with the patient on his back, allows of a comfortable resting position) or that there is adequate aspiration of pus between the stages. If this is not done pus may be forced into the lung through a broncho-pleural fistula and the patient either drowned or a severe bronchogenic spread of pulmonary tuberculosis result. Coryllos (166) advocates the use of a tracheal tube and con-

tinuous suction during operation in case of such an event. Hoyle (121) believes in aspiration between the stages of operation.

In old-standing cases of empyema the mediastinum is usually fixed, and therefore more ribs can usually be removed at the first stages of operation than in an ordinary thoracoplasty without post-operative paradoxical chest movement. Thus all of the first three ribs and as long lengths of the 4th, 5th, 6th and 7th ribs as can be obtained from the back may be removed. The anterior ends will be later removed by an antero-lateral stage. Apical mobilisation at the first stage of the thoracoplasty is only advised if the apex of the lung is adherent to the dome of the thorax - Price Thomas (167). The upper rib beds are formalinised but not the lower ones in case of a permanently mobile chest wall resulting. The final stage is planned in relation to the position of the sinus or residual cavity on the lines described above.

Lambert (168) in 1946 described some success in three cases with partial claviculectomy as an adjunct to the usual means of surgical collapse of the chest wall mentioned above. He found partial removal of the clavicle of use where the empyema space extended anteriorly and laterally, because the extrathoracic tissues are, to some extent, held out by the clavicle anteriorly.

On the whole the results with operative measures in old-standing cases of tuberculous empyema (mixed or otherwise) are disappointing. Ornstein and Herman (123) treated 86 cases surgically and only 34% were cured, while there were 46.5% of deaths in the series. Klassen, Miller and Curtis (122) found that only 38% of their thoracoplasty cases were completely well but 50% more were well apart from a residual sinus.

Woodruff's (2) results are as follows, using operational treatment:-

	Well	Living	Dead	TOTAL.
Group A. (No secondary infection).	7 (43.7%)	6	3(18.8%)	16.
Group B. (Secondary infection present).	5 (42%)	3	4(33.0%)	12.

Smyth (124) observes that before the advent of penicillin the mortality in contaminated tuberculous empyema, without surgery, was practically 100%. Now thoracoplasty, with drainage and the use of penicillin, offers, in competent hands, around 75% chance of recovery.

THE USE OF EARLY SURGICAL MEASURES IN TUBERCULOUS EMPYEMA.

The bad results with conservative treatment of the condition have been seen. The results with surgery when the empyema is of long-standing are not ideal because of the thickened pleura which tends to make residual spaces prevalent. The operations are technically laborious and difficult, too, in those advanced cases, and the trauma and shock to the patient severe. Is there any justification for early surgical measures as soon as the empyema is diagnosed? Although I, personally, have no experience of such early surgery, I believe that there is a very strong case for it. My reasons are as follows:-

A. Many cases, both in the series I have reviewed, and in others I have seen, have developed a spread of disease to the opposite healthy lung during the long period when futile conservative treatment was being pushed. With that fresh, contralateral disease they lost the only hope they had of surgical therapy.

B. I can recollect many thoracoplasty cases done where there was a persistent non-purulent effusion in the pleural space of the same side. Those cases, with judicious aspiration between the stages, do very well as a rule.

'B' (continued):-

The condition of the pleura in them is less thick than in the case of purulent effusions and corresponds to the state of affairs that would be present if early surgery were undertaken in empyema cases.

C. With gas analysis it is possible to ascertain the approximate size of the bronchopleural fistula present. Only in the case of the smallest fistulae is it justifiable to try vigorous conservative methods of treatment, and even the presence of a punctiform or a healed fistula is no guarantee that the lung will come out because, if the parenchymal disease is extensive, rapid pneumonitis and carnification may ensue. In the case of the larger fistulae it is logical that delay will gain nothing. Indeed it will further thicken the pleura and tend to cause chest wall sinuses, secondary infection, and a progressive deterioration in the patient's general condition.

D. My experience has been that after three months of conservative treatment very few lungs, if not expanded, finally re-expand because of pleural thickening. It is this same thickening which leads to the poor results with surgery after that period. Therefore it behoves us to operate early.

E. Finally, the results of such an eminent surgeon as Brock (120) prove that the use of early surgery should be given wider consideration than it is in most places at present. Brock is not alone in advocating early surgery. Archibald (169) laid it down at a meeting of thoracic surgeons in America that if the patient were not well on the way to recovery in from one to three weeks with aspiration and lavage, then thoracoplasty should be done.

At the Inaugural Meeting of the Irish Society for Thoracic Surgeons the speakers stressed the necessity for earlier operations in tuberculous empyema, and Henry showed

how, if early re-expansion of the lung was not forthcoming, the surgeon should not wait too long before operating.

Brock's (120) paper was designed to show (i) that tuberculous empyema is a serious condition which must be treated with respect and (ii) that conservative treatment fails in most cases, and that surgical treatment must be prompt and vigorous. Brock's definition of an empyema is "a purulent effusion which strengthens the argument when his results are seen." Dealing with 90 cases of tuberculous empyema, 60 of which progressed to secondary infection, he classified them as follows:-

Conservative treatment or drainage alone:	42.	
Died	33	}
Alive	9	}
Mortality	77%.	}
Thoracoplasty:	48.	
Died	7	}
Alive	41	}
Mortality	14%.	}

The total mortality was 40 out of 90 or 44%.

In the case of thoracoplasty done on 21 cases of uncontaminated tuberculous empyema, 1 case died but the remaining 20 cases did well.

An analysis of 84 cases of pyogenic tuberculous empyema was made:-

i.e., Drained only:	35	Thoracoplasty and drainage:	49
Dead:	32	Dead:	14 (29%).
Alive (awaiting thoracoplasty):	3	Alive:	35 (71%).
Mortality:	91%.		

Brock's analysis from the point of view of disease in the underlying lung shows that the results of thoracoplasty are less good than when the disease in the lung is controlled. Brock concludes that, whenever even remotely possible, thoracoplasty should be begun within a very short time of drainage measured in days, not weeks, and certainly

not in months. He states that contrary to the practice of waiting for the fever to fall before operation, persistent fever is an indication.

Finally, Brock, in a personal communication to me (1.6.48) gives his recent results with early operation in eleven cases of secondarily infected tuberculous empyemata. Those were drained and had thoracoplasty commenced within a few weeks of the onset in all cases. Of those patients one died after a fourth stage operation, one has died since, but seven are well and healed, and two are well and working and with a small discharging sinus. Those results far outstrip the cures with extensive surgery on old-standing mixed infection empyemata. Brock now proceeds to drainage and thoracoplasty immediately he sees the case, if suitable for operation, but he observes that the trouble in most cases is that he does not see the patient until many months have passed.

Mr. A.L. d'Abreu, thoracic surgeon of Birmingham, has also kindly brought to my notice four cases of tuberculous empyema done within three months of the empyema onset. All were cured without sinuses and no intercostal drainage was required. Mr. d'Abreu likes, if possible, to avoid intercostal drainage but prefers instead thorough needle aspiration before and between the operative stages.

It is evident then that a study of the mode of onset of tuberculous empyema and of the resultant pathological changes, as it persists for many months, shows that conservative treatment is doomed to give poor results from the start. Operation to be of maximum benefit must be early, and for the surgeon to be able to institute early treatment entails close co-operation or team-work with the physician as usually prevails in the thoracic centres. The trouble will arise where artificial pneumothorax therapy is conducted in smaller institutions with no surgical staff, and it will be necessary for those workers in the field to learn the urgency of the condition and to enlist the help of the surgeon as soon as the empyema develops.

I believe, from a close personal observation of many cases of tuberculous empyema treated conservatively, that 6 to 8 weeks is the maximum one should wait. If the lung has not completely re-expanded then, and no later, should the case be pushed for surgery. This allows for a delay of two weeks at the least for review of the films and arranging a surgical bed and a place on the operating list, and it brings the operation to the patient when his empyema has lasted just under three months. Surgery after the three month period is bound to have the same results as in long-standing casss of empyema.

SUMMARY AND CONCLUSIONS.

CHAPTER I.

Definition: The definition of tuberculous empyema varies widely in the literature, from "any collection of fluid in the pleural space, containing tubercle bacilli" through various grades of turbidity to frankly purulent fluids.

I have taken as my definition, only cases of pulmonary tuberculosis with pus in the pleural cavity. The dividing line between turbid fluids and pus is hard to determine, but a test which can be used is to note whether the scale markings of a new, 2 c.c. record syringe can be seen in a good light through the fluid in the syringe barrell. If not visible the fluid, when not haemorrhagic, is purulent.

Initial Investigation: With empyema clearly defined the first step was to ascertain its incidence and causation. The case records of 7,206 patients (deaths 2,157; discharges 5,049) who were in hospital between 1.1.29 and 1.1.48 were studied and empyema cases abstracted. It was observed that during this period an advanced type of case was being admitted to this Sanatorium of 420 beds, i.e., using the Ministry of Health's most recent classification, 82.4% of all admissions were R.B.2 or R.B.3 cases, R.B.1 cases comprised 2%, and all negative cases (mostly R.A.2) were 15.6% of admissions. The problem of poor social conditions and overcrowding in the district determines this incidence.

The numbers only of empyemas following spontaneous pneumothorax were noted, as the cause is obvious. The number of known spontaneous pneumothoraces escaping empyema was also recorded. A more detailed analysis was made of all empyemas not due to spontaneous pneumothorax and the abbreviated case records are included in this thesis, along with a reduction of the important X-rays where those were available. Those photographs were made by myself. Table 1 (page 6) gives the overall figures extracted from this preliminary investigation.

Findings re incidence of empyema:

There were 146 cases of empyema during the period under review. In 7,206 cases of pulmonary tuberculosis analysed there were 82 cases of spontaneous pneumothorax (an incidence of 1.13%). Empyema developed in 51.2% of the cases of spontaneous pneumothorax and their course was the same as that of empyema developing from other causes. The courses of the 40 cases of spontaneous pneumothorax which did not develop empyema are recorded. 104 empyemas or 71.2% of the total were not due to spontaneous pneumothorax. All but 11 of those were due to artificial pneumothorax therapy which, therefore, causes the bulk of empyemas. Table 3 (page 9) gives a summary of the position. Table 4 (page 10) is a correction so that cases who have been admitted with empyema following artificial pneumothorax will not be included with the cases who follow on artificial pneumothorax therapy in Baguley Sanatorium. Graphs (page 11) show that the trend of empyemas due to spontaneous pneumothoraces is level and averages about 4.4 per 2 year period. With increasing artificial pneumothorax therapy so has there been a rise in the incidence of empyemas following artificial pneumothoraces.

The criterion adopted, as to when an empyema is attributable to adhesion section, is that it should appear within three months of the operation. The empyema incidence in artificial pneumothoraces where there was no adhesion section, or where the adhesion section was not attributable, was 9.5%.

In artificial pneumothoraces, where the empyema was attributable to adhesion section, the incidence of the complication was 18.3%. As a rule most writers record a lower incidence of empyema in artificial pneumothoraces where adhesion section has been done, but, as events prove, in our series the cases were advanced at the start of active therapy and very few of the adhesions were totally divided, with the result that there were very few "free artificial pneumothoraces."

Several points were made concerning non-purulent pleural fluids.

A study of all the artificial pneumothorax cases (610) not associated with empyema in the 19 year period was made, to ascertain whether refills to positive pressures were more dangerous than refills which were always on the negative side of atmospheric pressure. This is important in view of conflicting opinions on the matter. It was seen that the numbers of negative pressure artificial pneumothoraces developing fluid, calculated as a percentage of all negative pressure artificial pneumothoraces, was 35%. The corresponding figure for positive pressure artificial pneumothoraces developing fluid was 53.2%. Table 5 (page 13) shows the figures. In all cases only fluid of sufficient quantity to cover the hemi-diaphragm, or to warrant aspiration, was counted.

Table 6 (page 14), and its accompanying graph, shows that from year to year there appears to be no close parallel, or constant relationship, between the number of artificial pneumothoraces developing non-purulent fluid and those developing true empyemata. This is possibly due to personal factors with regard to the medical officers treating the cases. Those medical officers have changed during a nineteen year period and each has a slightly different technique of artificial pneumothorax refill as regards manipulation of the needle, amount of air given, final pressure reading, etc. Also some medical officers hold on to contraselective artificial pneumothoraces longer than others.

Chapter I is concluded with notes of the recorded incidence of empyema in various workers hands. The incidence of the complaint as a complication of lobectomy and pneumonectomy is not omitted.

In reviewing a representative sample of the literature it is seen how the question of empyema incidence in artificial pneumothorax therapy has come to be that of its incidence in relation to thoracoscopy and adhesion section.

This shows how it is now widely recognised that internal pneumonolysis is an essential part of any programme of artificial pneumothorax therapy.

One very important point which has emerged from this initial investigation is that it is quite impossible to compare incidence rates of empyema, in series recorded by various workers, unless several factors are standardised, i.e.,

(a) the definition of empyema must be determined;

(b) the case classification of the entire tuberculous population at risk should be stated;

(c) there should be a fixed and constant time within which an empyema, if it develops after adhesion section, is attributable to the operation;

(d) artificial pneumothorax refills to negative pressures only, should be the rule.

CHAPTER II.

As a preliminary the small group of 11 cases which were not due to artificial pneumothorax therapy and apparently not due to spontaneous pneumothorax, were investigated. The conclusions as regards their mode of onset are that one was due to Pott's disease of the thoracic spine, two followed on operative removal of tuberculous lung tissue, and two were due to silent spontaneous pneumothoraces with no clinical evidence of this event. In the remaining six cases the exact mode of onset was not known, but five of the cases had extensive caseo-pneumonic types of disease before the empyema developed, and the other case had her empyema slowly developing in the place of greenish pleural fluid.

The question of whether or not there is always a communication between alveolar air and pleural space when an empyema develops is more than an academic one, as it affects the treatment of the condition. From a close personal study of many tuberculous empyemas I am convinced that there is a spontaneous pneumothorax present in the vast majority of

cases of empyema of insidious onset.

The final analysis, therefore, shows that out of 146 cases of purulent empyema in the 19 year period under review we can explain the exact nature of the onset of all but six of them, i.e., (spontaneous pneumothoraces - 44 cases, artificial pneumothoraces - 93 cases, operative removal of diseased lung - 2 cases, and Pott's disease of the thoracic spine - 1 case).

It now remained to analyse the data relating to the 104 cases of empyema which did not follow spontaneous pneumothorax. This was done in detail under various headings.

Age and sex: It was concluded that, like pulmonary tuberculosis as a whole, tuberculous empyema is affecting persons in the young adult age groups. The average male age at onset of empyema (28.2 years) in 45 cases was slightly higher than the corresponding female age (25.5 years) in 59 cases.

Case classification at the onset of empyema:

Table 7 (page 29) shows the numbers of empyemas in each class of case calculated as a percentage of the total empyemas being analysed. Ninety-seven percent of the empyemas following artificial pneumothoraces did so in R.B.2 and R.B.3 cases. However, when the 610 cases with artificial pneumothorax, who did not develop empyema, are analysed it is seen that 96.8% of the subjects were also in the R.B.2 and R.B.3 groups. Thus we cannot, from this table, say that empyemas are necessarily commoner when artificial pneumothoraces are done on advanced cases, when the population at risk is also largely in this advanced category. However, I hope to prove later in the pathological approach to the problem that it is bad policy to induce artificial pneumothoraces in those advanced cases.

The pleural cavity involved was on the right side in 42 cases and the left in 62 cases. In men the numbers of all other artificial pneumothoraces was approximately equal on each side, while in women 39% were on the right and 61% on the left side.

Refills to mean positive pressures: It has previously been shown that artificial pneumothoraces that are conducted with mean positive pressures develop half as many non-purulent fluids again as artificial pneumothoraces conducted with refills to negative pressures. Of the 93 cases of artificial pneumothorax developing empyema 24 had refills to mean positive pressures. Several cases are quoted where, without doubt, a refill to positive pressure was responsible for a superadded spontaneous pneumothorax or adhesion rupture and rapid empyema onset. In other cases it is impossible to say that the refill to a positive pressure had any relationship to the onset of empyema.

On the whole the investigation into this aspect of the problem points to two factors, namely -

(a) the undesirability of using positive pressure refills in artificial pneumothorax therapy;

(b) the potential danger of uncut pleural adhesions in artificial pneumothorax therapy.

It has been my experience that "free artificial pneumothoraces" give much less trouble than those with uncut adhesions whether in an apico-mediastinal position or elsewhere.

Blood Sedimentation Rate at onset of empyema.

This record was available in only 53 cases of empyema. There was wide variation in the figure from 10 mms./hour in two cases to 45 mms./hour in one case. The average reading was 23 mms./hour. No conclusions can be drawn from those values as it is a common event to find an attendant rise in Blood Sedimentation Rate values with the onset of pleural inflammation in pulmonary tuberculosis.

Pyrexia at induction of artificial pneumothorax.

Allowing for adhesion sections or positive pressure refills as possible causes of empyema, the trend, when those cases are omitted, is for the onset of empyema to be earlier where there is pyrexia at induction (mean of 4.4 months) than where the patient had no elevation of temperature (mean of 9.6 months). This finding agrees with the views of several authoritative workers.

Finally, the radiological appearances of the artificial pneumothoraces which were responsible for the empyemata were analysed and the findings are most important for they give us the clue to the two factors which above all others are responsible for the final events leading to empyema onset. Table 8 (page 34) describes the types of artificial pneumothoraces commonly seen before empyema developed. It was possible to obtain the appropriate artificial pneumothorax X-rays of 78 of the cases. The table shows how many empyemas developed under six months and how many over six months, from the onset of the artificial pneumothorax. A sample of 201 artificial pneumothoraces from the series, which did not develop empyema, is similarly analysed and, short of pus formation, the non-purulent fluid formation tends to be more marked in the two types of artificial pneumothoraces (Groups IV and II) where pus is likewise more common. Only eleven artificial pneumothoraces are "free" in the samples analysed but they have no fluid formation.

We have already ascertained that over 90% of all the artificial pneumothoracesare in advanced cases (R.B.2 and R.B.3) and we now know that most of the resulting X-ray appearances fall into the Groups II, III and IV described in Table 8, i.e., the induction of artificial pneumothoraces on advanced cases leads most commonly to this type of artificial pneumothorax. The most dangerous type of artificial pneumothorax from a radiological aspect is in Group IV and has adhesions present with an opaque, "ground-glass" appearance of lobe and a patent cavity. Group II cases are less dangerous and have patent cavities and adhesions visible but lack the "ground-glass" appearance. Similarly, Group III cases are less dangerous than those of Group IV. They have adhesions visible and opaque, "ground-glass" like lobes but no visible cavities.

It is, therefore, necessary to study (a) the role of adhesions and (b) the significance of the radiologically opaque, milky-looking lobes which develop so readily in artificial pneumothoraces done on unsuitable or advanced cases.

CHAPTER III.

This section is devoted to a study of the role of tuberculous tracheo-bronchitis in causing the opaque, milky-looking lobes in the X-ray appearances of the artificial pneumothoraces which lead to disaster if maintained.

A representative sample of the literature on the subject is reviewed. It is at once evident that most of the work refers to the trachea and those bronchi within bronchoscopic vision. Very little attention has been paid in the past to the role of the smallest bronchi.

A higher incidence of tuberculous tracheo-bronchitis is on the whole reported where post-mortem studies have been made, compared with those accounts of bronchoscopic findings. The former incidence is perhaps raised because the cases who die are mostly advanced, cavernous cases with copious sputum. Those cases who are bronchoscoped are not necessarily so advanced. I observed that post-mortem degenerative changes quickly occur in the mucosa of the air passages in the presence of pus. Therefore, in observing mucosal ulcers, post-mortem examinations should be made as soon after death as possible, as I have done.

The only real way of assessing the incidence of tracheo-bronchial involvement in doubtful cases is by microscopic examination which I have done in my series of cases which were examined in detail. At an early period in the investigation I observed that secondary bronchi draining cavities and the finer bronchi and bronchioles in the vicinity of diseased areas were invariably involved in the tuberculous process even though the bronchi which could be seen by the bronchoscope were free in many of those cases. It was decided, therefore, to assess the relative importance of the disease in each class of bronchus.

Disease in the Trachea and Bronchi within Bronchoscopic Vision.

Involvement of the larynx was not included as it is so often affected owing to the fact that it offers a site where sputum may easily lodge.

The study was made by clinical, post-mortem and a limited number of bronchoscopic examinations.

Examples of tracheo-bronchial tuberculosis which I personally have seen can be listed under the following headings:-

A. Generalised bronchitis with here and there small, specific ulcers.

B. Solitary ulcerations in the absence of gross general bronchitis.

C. Complete fibrous stenosis of a bronchus.

D. Partial fibrous stenosis, often with resulting intermittent atelectasis of distal lung, bronchiectasis and a large septic element.

E. Nodules of tuberculous granulation tissue.

F. Endobronchial ulceration from outside pressure and erosion by a caseous, softening, hilar lymph gland.

G. Stenosis of a bronchus from pressure from without and no endobronchial ulceration.

Examples of the above types of tracheo-bronchial tuberculosis are quoted and photo-micrographs of sections included for descriptive purposes. The role that they play in leading to empyema formation in artificial pneumothoraces is described, and it is undoubtedly of great importance. However, my point is that the role of the diseased bronchiole and finer bronchus and the draining bronchus from a cavity, is even more important because it is so much more often diseased than its large, main counterpart. When one sees tracheo-bronchitis bronchoscopically one can safely say that the finer bronchi are diseased because disease in the main bronchi in most cases is caused by the passage of tubercle bacilli positive sputum. A negative bronchoscopic examination, on the other hand, is no guarantee that the finer bronchi and bronchioles are free from disease.

Tuberculous Bronchitis in Bronchi and Bronchioles beyond Bronchoscopic Vision.

In the discussion on the "ground-glass" radiological appearance one sees in certain artificial pneumothorax cases,

the term "atelectasis" is taken to mean collapse of previously aerated lung. The various views on the milky, opaque X-ray appearance are discussed, and the mechanism of gas absorption from distal alveoli when a bronchus is obstructed, described.

An example, with photographs, is given of the danger of the tension type of cavity which results in certain artificial pneumothoraces or cases of adhesion section. This case showed no tracheo-bronchial abnormality, yet from post-mortem examinations I have found that the draining bronchus from such a cavity is invariably diseased and liable to be kinked or obstructed when its alignment is altered.

Post-mortem study has revealed a high incidence of tuberculous disease in the smallest bronchi and bronchioles in the vicinity of cavity walls and caseo-pneumonic patches. The changes have been classified into 3 stages for descriptive purposes and diagrams and photomicrographs are appended to illustrate them.

I have made bronchograms on many opaque lobes following artificial pneumothorax therapy, and a typical example is reproduced by photographs (page 64A.). Using warmed lipiodol it is seen that the bronchial block is not in those bronchi visible by the bronchoscope as is stated by so many writers, but is, in fact, in the terminal bronchi (third or fourth degree) and outwith bronchoscopic vision.

This opaque, "ground-glass" like appearance seen in some cases of artificial pneumothorax does not represent irreversible pathological changes. If the artificial pneumothorax is abandoned at once, in some cases re-aeration of the lobe occurs. This is presumably so when the changes in the finest bronchioles are slight (stage 1 described). If collapse is maintained a pneumonitis ensues and the caseous areas have a chance to extend, and carnification may ensue, too, as is illustrated in some of the post-mortem cases. A photomicrograph of such a carnified lung is reproduced.

The chain of events when a tension cavity forms is

described, as it leads to collapse of lung tissue, spread of caseous foci, and final rupture into the free pleural space. The prognosis as regards empyema formation is less grave where there is no persistent cavity in the opaque lobe. A case is described (B.W. - page 70) where such a cavity in an opaque lobe may suddenly vanish, presumably after complete bronchial blockage, and the resulting collapsed lobe shrink, and after several years, when the artificial pneumothorax is abandoned, be pushed upwards and inwards alongside the mediastinum. It is stressed that those contra-selective artificial pneumothoraces should not be kept in the slender hope that this favourable chain of events may occur.

Post-mortem findings:

The post-mortem technique is described, and the results in twenty-eight thorough chest examinations recorded in Tables 9 and 10.

The chapter is concluded with some recorded notes on the treatment of tracheo-bronchial tuberculosis.

CHAPTER IV.

The Role of Pleural Adhesions in Empyema Onset.

A brief historical outline of the development of artificial pneumothorax therapy is given.

Some observations on the peripheral spread of tuberculosis from the lungs to pleural surfaces, endothoracic fascia, intercostal lymph glands and sub-diaphragmatic tissues are recorded.

The formation of adhesions and their commonest distribution is mentioned. An analysis of post-mortem records from Baguley Sanatorium was done to determine the number of cases with adhesions and the number of cases in which artificial pneumothoraces could have been induced had it been so desired. The results of my own post-mortem series are recorded in Table II.

A photomicrograph of an organising caseous focus which was situated in the endothoracic fascia is reproduced.

The presence of such nodules and of intercostal gland involvement seen at post-mortem and thoracoplasty operations shows that even enucleation of adhesions may light up foci of infection, and the mechanism of extrapleural space infection with tubercle bacilli, after the formation of extrapleural pneumothorax or Semb's apicolysis, can be similarly explained.

The traumatic effect of adhesions on the lung surface, and their role in holding cavities open, is described. The dangers of incomplete division of adhesions are mentioned. The ideal aim in artificial pneumothorax therapy is discussed.

B. An account is given of the dangers of large or small subpleural caseous areas with little overlying pleural reaction.

Photomicrographs reveal the very close proximity of masses of tubercle bacilli to a free pleural surface in such cases.

Finally, figures are given showing that if adhesion section is going to be a determining factor on the onset of pus in the pleural cavity, it does so early in the average case, i.e., 1.21 months, as opposed to a mean of 8.4 months where no adhesion section was undertaken.

An example is given of a case of empyema which cleared after complete division of adhesions and aspiration. The artificial pneumothorax was continued and has been dry for a year. This is an exceptional event.

CHAPTER V.

The clinical course of empyema is discussed. An analysis of the pleural fluids sent to the hospital laboratory between January/1944 and December/1947 is of interest. (Table 12). There are approximately equal numbers of pure tuberculous and secondarily infected, tuberculous empyemas. In only 5 cases did a pure tuberculous empyema of several months' standing progress to a mixed infection empyema. This

suggests that the ultimate size of the broncho-pleural fistula is determined at its onset. It is probable that fistulae which communicate with bronchi directly as opposed to alveoli, become secondarily infected readily.

The relative frequence of occurrence of secondary infecting organisms is noted in Table 13. The clinical course of a tuberculous empyema having been described, the results of treatment by all the methods used are given in Table 14 (page 101). It is seen how serious the condition is. In 105 cases 67.6% died under 5 years while only 19.05% recovered completely. The fate of the rest is indicated.

CHAPTER VI.

TREATMENT:

The admission of a number of cases of tuberculous empyema to Baguley Sanatorium from another institution which closed its chest wards in May/1947 confirmed the opinions on causation and treatment already formed from the study of the Baguley Hospital empyema series.

There are two main approaches to the problem of treatment - conservative and surgical.

A. Conservative. The case may be left alone and aspirations done when the fluid by its pressure and volume is causing dyspnoea or cardiac embarrassment.

B. Regular and frequent aspirations may be done and intra-pleural irrigations employed or negative pressure suction applied.

The surgical methods resolve themselves into -

C. Operations on cases of empyema of several months' standing, which is the usual procedure in most sanatoria at the present time.

D. Operations on recent cases of empyema of only a few weeks and certainly not more than 3 months from the empyema onset.

Each of those lines of treatment has its advocates. I hope to show, and I am convinced in my own mind, that the early surgical treatment is the best line of attack and the most sensible if useful lives are to be saved.

Table 15 (page 104) shows the results in 90 cases of purulent, tuberculous empyema treated by conservative methods. The total deaths or impending deaths are 81.1% of the total. Only 16 cases or 17.8% recovered completely. The exact method of treatment in each case is included in a table at the end of the volume.

Results with conservative treatment in other workers' hands are given and the commoner irrigating substances that have been used are mentioned. There is such diversity of irrigating fluids as to suggest that the results with conservative treatment have been poor in each worker's hands.

The correct management of a case in which aspirations are done is described, and it is shown that there are two main reasons for the failure of such cases.

A. The mechanical factor of grossly thickened visceral and parietal pleura which holds down the lung. After 3 months it is extremely rare for a lung, so bound down, even if healthy, to be able to re-expand.

B. The nature of the disease in the underlying lung may prevent it from ever re-expanding.

Photomicrographic studies of thickened visceral pleura from a case of empyema are added to prove the points raised.

The role of broncho-pleural fistulae in preventing lung expansion is discussed as well as the methods used to diagnose this event.

The most scientific approach to the problem is by a gas analysis of the pleural air. This is described. The work of Coryllos (3) is quoted. It is shown that gas analysis does indeed yield an accurate means of detecting broncho-pleural fistulae but with the same type of apparatus as Coryllos used, in my hands, it has not been possible to

state definitely what size the fistulae were. I am of the opinion that only small and large varieties may be diagnosed and the intermediate sizes fall into one of those two groups as regards gas analysis results. Also the presence of the smallest fistula is no guarantee that the lung is bound to re-expand with vigorous conservative methods of treatment. As previously stated, the parenchymal disease may be of such an extent that carnification ensues quickly. This is all the more reason for earlier surgical approach to the problem before there is a contralateral spread of disease or the patient loses condition.

The use of two new drugs - RAS. and Nisin, in the conservative treatment of empyema, is described. The latter drug has, and is being used exclusively by Dr. L. Parker of this Hospital. Neither drug offers any advantage over the other numerous applications previously mentioned.

Surgical methods are next discussed. There are 15 cases in the series under review and I personally have been acquainted with 14 similar cases recently, treated by operation when the empyema was of many months standing.

Results in the series are noted in Table 16 (page 138). The death rate is 53.3%, the average duration of empyema being 57.4 months. The complete recovery rate, with no remaining sinuses, was 26.7%. The other cases are described.

Those results are an improvement on conservative therapy but still leave much to be desired. Examples of the severe nature of the operations, which are technically difficult owing to tiled ribs and fibrosed intercostal structures, are given in the case reports at the end of the volume.

In 78 of the empyema cases of the series who did not have operative treatment, it was possible to analyse the state of the opposite lung. Surgery on the empyema side was ruled out, even at the onset of empyema, in 54 cases, owing to contralateral disease. This shows that even with the best surgical intentions there will still be a high death

rate once empyema develops, as many of the cases will be doomed to conservative treatment only.

Our attempts to establish a chemo-therapeutic "umbrella" to operative procedures, with the few agents at our disposal, failed.

P.A.S., Nisin, and Dibrompropamidine Isethionate (M.& B.1270)were tried. It was considered, with only 3 cases, that effective doses of the latter drug were dangerous and might lead to liver damage. An experiment with M.& B.1270 in developing chick-embryos is mentioned.

A brief outline of the usual operational methods used in chronic tuberculous empyema is given. Each case has to be treated on its own merits.

Finally, the reasons why early surgical methods of treatment (within a matter of weeks from empyema onset) should be adopted, are discussed. I am convinced from studying the results in the series under review and from a wide clinical experience of the condition, that early surgical treatment should be used more often than at present. The inevitable pathological events in the pleura whereby it becomes rigid and thickened also point in this direction.

Mr. Brock is the great exponent of early surgical treatment in this country and his published results are quoted as well as recent results sent to me in a personal communication. They are an improvement over surgical results in advanced cases of empyema. Similarly 4 cases of Mr. A.L. d'Abreu of Birmingham, operated on within 3 months of empyema onset, were all cured without sinuses.

At the end of the volume case reports are added and the records in post-mortem examinations.

This study has revealed two broad lines of attack in the problem of tuberculous empyema,

(a) prevention;

(b) treatment.

There will always be a certain number of cases of

empyema admitted to sanatoria, i.e., cases following spontaneous pneumothorax.

There is no doubt that the incidence of empyemas following artificial pneumothorax therapy can be cut down by better choice of cases for artificial pneumothorax treatment and by a realization of the pathological events in a compressed and airless lobe or when a cavity balloons, with the prompt abandonment of such artificial pneumothoraces. The only safe artificial pneumothorax is one which is "free" and unfettered by adhesions. Such artificial pneumothoraces will very rarely be obtained when R.B.2 and R.B.3 cases are being admitted to sanatoria.

As I stated in my introduction, a worker in a tuberculosis sanatorium of this size is brought into contact with medical, surgical and social problems. The admission of advanced cases is a sign of the times. Waiting lists are long and most cases remain under dispensary care for 10 months before reaching hospital. Many of them, treatable at the outset, are beyond hope of sputum conversion by the time they are admitted. Many of the existing hospital beds are filled by chronic cases who have no homes. Advanced cases take priority on waiting lists if they are diagnosed in common lodging houses or overcrowded houses containing young children. The nursing shortage throughout the country does not help matters.

Nevertheless, something drastic will have to be done in the country as a whole, to get early cases under treatment as soon as they are diagnosed or we will go on merely marking time. When early cases are received, the empyema rate should fall because better artificial pneumothoraces will be procurable.

The study of treatment shows the universally poor results with anything but surgery, which should be instituted early to be of maximum benefit.

I hope that I have been able to contribute something of value to our knowledge of this condition.

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90 CASES TREATED BY CONSERVATIVE METHODS.

Case No.	Duration of empyema.	Result.	Treatment.
1A.	6 months	Death	No aspirations.
1B.	3 "	Death.	No aspirations.
2A.	12 "	Death.	Air replacement.
2B.	18 "	Death.	Regular air replacement. Repeated aspirations for three years.
2D.	5 "	Death.	Aspirations, air-replacement and finally gelatine thorax.
2E.	2 "	Death.	Repeated aspirations and air replacements.
2F.	2 "	Death.	Air replacement.
2G.	12 "	Death.	Infrequent aspirations.
3B.	44 "	Death.	Regular air replacement.
3C.	3 "	Death.	One aspiration.
3D.	7 "	Death.	Air replacement. Later gelatine and flavine injections.
3E.	26 "	Death.	Irregular aspirations. Sanocrysin.
3F.	2 "	Death.	Monthly aspirations.
3G.	11 "	Death.	Regular aspirations and air replacement.
3H.	14 "	Death.	One diagnostic aspiration only.
3I.	4 "	Death.	Air replacements. Sanocrysin.
4A.	No trace (over 3 months).	Poor general condition.	Air replacements.
4B.	10 months	Recovery from empyema.	Right Phrenic Crush. Continuous aspiration (air replacement at first).
4C.	1 month	Death.	Air replacement.
4D.	7 months	Death.	Monthly aspirations.
4E.	9 "	Death.	Irregular aspirations.
4F.	26 "	Death.	5 gas replacements.
4G.	42 "	Death.	Monthly air replacements.
4H.	31 "	Re-expansion of lung.	Many aspirations.
4I.	12 "	Death.	Weekly Eusol washouts and air replacements.

Case No.	Duration of empyema.	Result.	Treatment.
4J.	6 months	Death.	1 air replacement, 1 aspiration and 1 air removal.
4K.	1 month	Death.	1 aspiration.
5A.	11 months	Death.	Irregular aspirations at first. Later regular aspirations at weekly intervals.
6B.	13 years	Partial recovery.	Several months air replacement. Later saline washouts and repeated aspirations.
6C.	36 months +	Death.	Frequent air replacements. Later many aspirations and flavine washouts.
6D.	71 months	Death.	Weekly air replacements.
6E.	3 "	Death.	Aspirations with gas replacement.
6F.	2 "	Death.	Repeated aspirations.
7A.	50 "	Death.	Left phrenic crush. Frequent aspirations and saline irrigations.
7B.	8 "	Re-expansion of lung.	Regular aspirations and Soluseptasine washouts.
7C.	4 "	Re-expansion of lung.	Regular aspirations.
7D.	38 "	Death.	Regular aspirations.
7E.	41 "	Death.	Repeated aspirations and Soluseptasine washouts.
7F.	16 "	Death.	Aspirations and saline irrigations. Methylene Blue and Ol. Menth.Pip.
7G.	12 "	Death.	In last month aspirations and intercostal drain. No previous treatment.
7H.	4 "	Death.	Irregular aspirations.
7I.	7 "	Death.	Bi-weekly aspirations. 12 injections ½ c.c. Benzyl Cinnamate.
7J.	2 weeks	Death.	Aspirations and air withdrawal.
7K.	54 months	Death.	Aspirations at first.
7L.	90 months (7½ years).	Cachexia and death.	Regular air replacements.
7M.	10 months	Death.	Regular aspirations. Saline irrigations and Soluseptasine instillations.
8A.	72 months (6 years).	Probable recovery.	Repeated aspirations and Azochloramide washouts for 15 months.

Case No.	Duration of empyema.	Result.	Treatment.
8B.	6½ months	Death.	Regular aspirations.
8C.	120 months (10 years).	Recovery. Lung expansion.	Air replacements 2 years. Then no aspirations for 8 years.
8E.	16 months	Death.	Aspirations. Saline washouts.
8H.	20 "	Recovery. Lung expanded.	Frequent aspirations and Soluseptasine washouts.
8I.	5 "	Death.	Two aspirations.
8J.	6 "	Re-expansion of lung.	Frequent aspirations.
8K.	11 "	Death.	Regular aspirations.
8M.	2½ "	Death.	Aspirations and Azochloramide washouts.
8N.	4½ "	Death.	Intercostal stab drain. Daily Azochloramide washouts.
8.0	14 "	Death.	Regular aspirations. Azochloramide washouts and saline washouts.
8P	15 "	Death.	Repeated aspirations. Intubation and Azochloramide washouts.
9A.	17½ "	Death.	Frequent aspirations. Stab drain.
9D.	4 "	Complete recovery.	Vigorous aspirations.
9E.	24 "	Doubtful re-expansion. Pleurae thick.	Air replacements - 1 year. Aspirations. Azochloramide and formaline washouts.
9F.	40 "	Death.	Stab drain and washouts.
9G.	16 "	Lung re-expanded.	Repeated aspirations and Azochloramide washouts.
9I.	114 " (9½ years).	Death.	Air replacement 17 months. Lapse of 8 years. Stab drain.
9J.	4 days	Death.	Aspiration.
9K.	6 months	Death.	Repeated aspirations.
9M.	60 "	Death.	Repeated aspirations.
9N.	24 "	Death.	Repeated aspirations and Azochloramide washouts. Stab drain 1½ years.
9.0	4 "	Death.	Repeated aspirations. Azochloramide washouts. Penicillin.

Case No.	Duration of empyema.	Result.	Treatment.
9P.	30 months	Death.	Repeated aspirations. Antiseptic washouts. Monaldi catheter drain.
10A.	38 "	Death.	Right phrenic crush and frequent aspirations.
10B.	30 "	Death.	Frequent aspirations and saline washouts. Finally Penicillin.
10C.	47 "	Death.	Stab drain. Saline washouts. Penicillin finally.
10D.	8 "	Cure of empyema. Lung expanded.	Vigorous aspirations.
10E.	36 "	Cured.	Left phrenic crush and repeated aspirations.
10F.	30 "	Death.	Air replacements. Stab drain and Azochloramide washouts.
10G.	30 " to 3.4.48.	Still empyema deteriorating.	Repeated aspirations and Azochloramide washouts.
10H.	10 months	Death.	Repeated aspirations six months. Stab drain and Azchloramide 4 months.
10I.	11 "	Recovery.	Vigorous aspirations. Cannula suction of debris. Penicillin.
10L.	33 "	Recovery.	Repeated aspirations. Penicillin instillations.
10M.	32 " to 3.4.48.	Deteriorating rapidly.	Stab drain and Azochloramide.
10N.	7 months	Death.	Occasional aspirations. Stab drain. 4 stage thoracoplasty and plastic operation. (Operation on old case fails to close space).
10P.	7 "	Death.	Aspirations.
10R.	10 "	Death.	Aspirations. Stab drain. Washouts.
10S.	4 "	Death.	Aspirations. Blood and Penicillin.
10T.	8 "	Lung re-expanded.	Aspirations. Blood and Penicillin.
10U.	13 "	Death.	Aspirations. Intercostal tube. Azochloramide. Breathing exercises.
10V.	$8\frac{1}{2}$ "	Death.	Repeated aspirations.
10W.	6 "	Death.	Repeated aspirations. Drain and Penicillin washouts.
10X.	3 weeks	Death.	Aspirations. Stab drain. Penicillin.

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CASE HISTORIES.

The serial numbers correspond to the periods of discharge or death of the cases, i.e., No.1 ... 1929-30; No.2 ... 1930-32; No.3 ... 1932-34, etc. Alphabetical order is used for the cases in each two yearly period, therefore each case has a serial and an alphabetical number, and this is reproduced on the X-ray photographs where available.

1929-30.

1A. E.M.B. No.11343. Female, 28 years at empyema onset and R.B.3 classification.

Empyema was the sequel of a right artificial pneumothorax and came on 4 weeks from induction and from positive pressure refills which were present from the outset.

Patient pyrexial at induction of artificial pneumothorax.

No record of Blood Sedimentation Rate at onset.

Treatment was by abandoning artificial pneumothorax - no aspirations. Death in six months.

X-ray after induction showed -

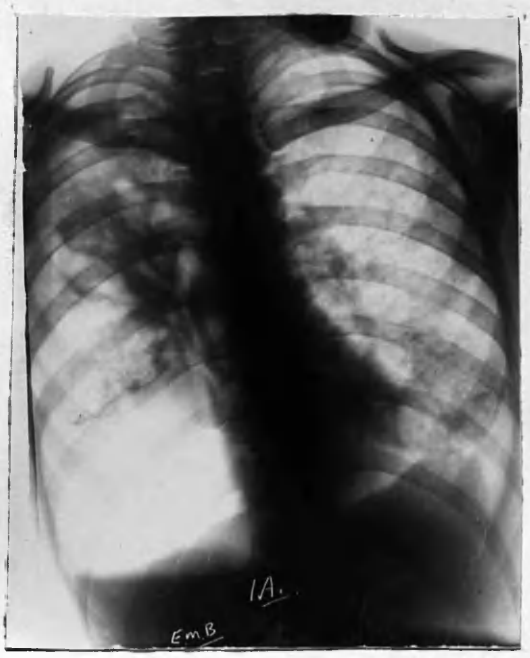
(R) side. An artificial pneumothorax present mostly basal and contraselective. Right upper lobe is opaque and milky in appearance (atelectatic). Several patent cavities are visible and tortuous draining bronchi. Upper and mid.zones of right lung widely adherent to chest wall. Trace of fluid at right base.

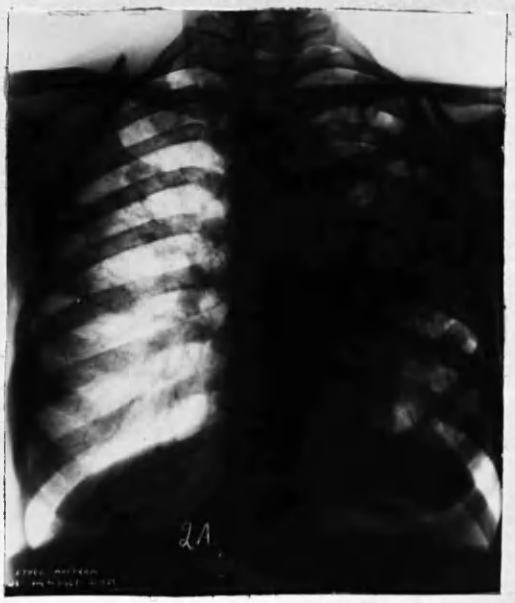
(L) lung shows extensive mottling all zones with early mid.zone cavitation. Elevation of left diaphragm present.

This was a "soft" film and the patient was badly positioned. A contraselective artificial pneumothorax where disease in right upper lobe draining bronchi is highly probable. No adhesion section was done.

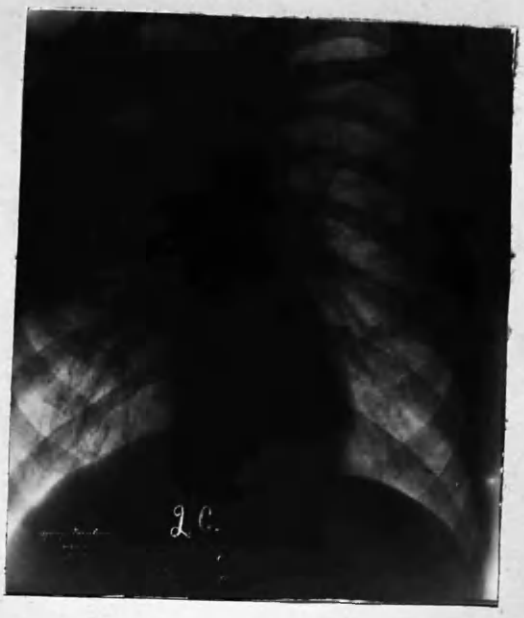
1B. T.A.E. No.12207. Male, aged 44 years at empyema onset.

The dossier shows that no artificial pneumothorax was induced but that the left empyema was the sequel of advanced bilateral pulmonary tuberculosis (R.B.3 classification). No clinical history suggesting a sudden


1A.
E.m.B.


2A.


2.B.
LILY GLEDHILL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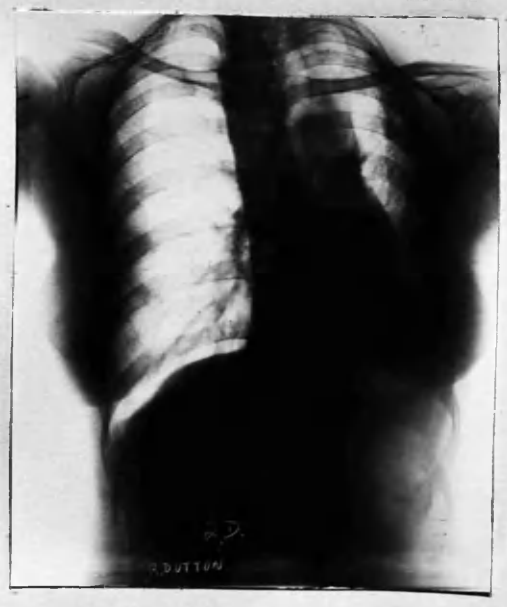

2.D.
E.DUTTON


2.E
H. COWAN.
6.9.32.

1B. T.A.E. No.12207 (continued):-

spontaneous pneumothorax and temperature records do not indicate a sudden lung rupture. Treatment was by bed rest only and no aspirations. Death occurred in 3 months.

No record of Blood Sedimentation Rate.

No X-rays available.

(During 1929-30 screening was in common use in the hospital so that the records are accurate as regards the presence of collapsed lungs, cavities, spontaneous pneumothoraces, and the presence or otherwise of fluid).

1930 - 32.

2A. E.M. No.11443. A female, aged 18 years at onset of empyema. Case classification at onset R.B.3. The empyema was the sequel of a left artificial pneumothorax. There was pyrexia of 99.6°F. at induction. Time of onset from induction was 10½ months but only two months from a positive pressure refill of +2, +10 c.cs.water. No adhesion section done and no records of Blood Sedimentation Rate available.

Treatment was by air-replacement of the fluid for eight months when the patient took her own discharge.

Final result - death after empyema had existed for one year.

The only available film taken just before the induction of left artificial pneumothorax shows -

Right lung - mottling, scanty, in mid.and upper zones, with apical cavitation;

Left lung - dense mottling, of pneumonic type, in the mid. and upper zones, with numerous apical cavities.

The heart and mediastinum are slightly displaced to the left. There is peaking of the left diaphragm.

(This, the only available film in this case, was taken just before left artificial pneumothorax induction).

2B. L.G. No.12,510. A female, aged 18 years at empyema onset. Case classification at onset was R.B.3.

Pyrexia of 99°F present when left artificial pneumo-

2B. L.G. No.12,510 (continued):-

thorax was induced. No adhesiotomy performed. Empyema onset was two months from induction and five weeks from the start of positive pressure refills averaging +5 cms. water. Treatment was by repeated aspirations and air replacement. Death followed in eighteen months with the empyema persisting. No Blood Sedimentation Rate records. A faded film taken just before the left artificial pneumothorax was induced is the only X-ray record and shows dense, soft woolly mottling all zones right lung but most marked in the mid. and upper zones. Probable cavitation right apex. Heart over to right. Left lung has soft mottling in the upper and mid.zones but not so extensive as on the right side. Left apical vomica is clearly seen. Both costo-phrenic angles are clear.

(Screening records after induction show that the left lung quickly became opaque with patent vomica visible and the left artificial pneumothorax refills were continued).

2C. S.F. No.11,355. A male, aged 32 years at empyema onset. R.B.2 classification at onset of empyema. Evening temperatures of 101°F. at induction of right artificial pneumothorax. No positive pressure refills and no adhesion section done. Empyema followed five weeks after induction of right artificial pneumothorax. Treated by repeated aspiration and then a total right thoracoplasty after 3 years at the Manchester Royal Infirmary. A residual space was left, sinuses formed in the thoracoplasty scar and toxaemia caused death. Duration of empyema was 3 years 8 months. No Blood Sedimentation Rate records.

An X-ray photograph before the right artificial pneumothorax was induced shows the trachea is displaced to the right. Large vomica visible in right apex and much soft mottling below and around this in right mid.zone. Prominent left hilar markings and some, scanty, dis-

2C. S.F. No.11,355 (continued):-
creet mottling in left mid.zone. The records show that this is an example of failure of extensive thoracoplasty to close a chronic empyema space in the face of grossly thickened pleura.

2D. R.D. No.12,836. A female, aged 30 years at empyema onset. Case classification R.B.2 at start of empyema. No artificial pneumothorax was induced here. No records of Blood Sedimentation Rate present. The history is of the insiduous onset of a clear, left pleural effusion which gradually became purulent. Treatment was by aspirations, air replacement, and finally a gelatino-thorax. The patient died 5 months after the onset of empyema proper. The only film available from this case shows marked kypho-scoliosis. The right lung field is clear. The trachea and heart are displaced to the left and the appearances suggest fluid in the left costo-phrenic angle. On the original film the appearances suggest erosion of the body of fourth thoracic vertebra, and there is an eliptical shadow over the spine below this suggesting a Pott's disease abscess.
This case is interesting as being one of tuberculous empyema secondary to tuberculous disease of the spine - one of the rarer causes of empyema.

2E. H.C. No.13,524. A female, aged 24 years at empyema onset and with case classification R.B.3. Admitted with a left empyema following left artificial pneumothorax of which no record is available. Treatment was by repeated aspirations and air replacements, but death ensued two months from the empyema onset. No Blood Sedimentation Rate records made. The patient was highly pyrexial on admission here where no refills were done.
The film taken on admission here shows some scoliosis present with the upper convexity to the right. Right mid. zone has scanty, soft mottling which has not reproduced well. On the left side a hydro-pneumothorax is present. There are two layers of fluid and the "ground-glass"

2E. H.C. No.13,524 (continued):-

appearance of the left upper lobe which is cavitated and apically adherent to chest wall, is seen. There is tiling of the left ribs.

This has been a contraselective left artificial pneumothorax with patent cavities visible during the course of the artificial pneumothorax. Although no exact dates are given, the records show that the empyema was of rapid onset after the artificial pneumothorax was induced (i.e. under six months).

2F. E.A. No.13,085. A female, aged 39 years and case classification R.B.3 at empyema onset. Pyrexia of 99.6°F. at induction of right artificial pneumothorax. Empyema followed two months from induction but one month from a positive pressure refill of +9, +13 cms. water. No Blood Sedimentation Rate records present. Treatment was by air replacement but death from toxaemia resulted in two months.

No films are available but adequate screening notes indicate a contraselective right artificial pneumothorax with opaque upper lobe containing a visible cavity; there was tuberculous disease present on the left side too, in the form of soft mottling.

2G. J.S. No.13,003. A female, aged 24 years, and case classification R.B.1 at empyema onset. No Blood Sedimentation records. No artificial pneumothorax was induced here but the empyema began as a left pleural effusion without clinical evidence of a spontaneous pneumothorax. Empyema slowly developed in the place of greenish pleural fluid. Treatment was by aspirations at irregular and infrequent intervals dictated by dyspnoea of the patient. The empyema persisted and death occurred in one year.

No film is available in this case.

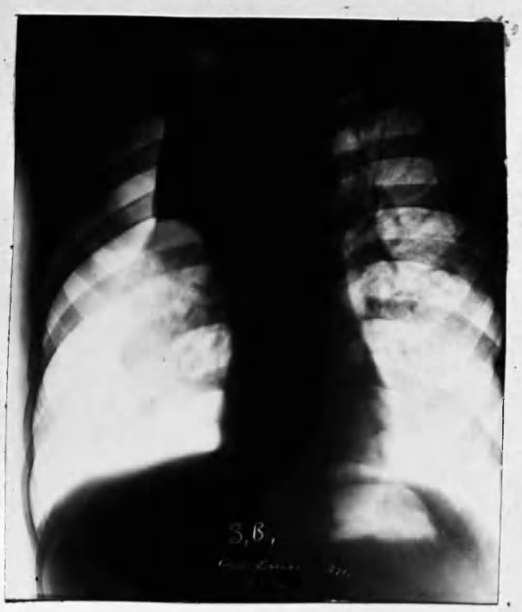
3A. W.G. No.12,957. A female, case classification R.B.2 and aged 20 years at onset of empyema. No artificial pneumothorax induced in this case which started as a right pleural effusion which became purulent. There was no clinical evidence of spontaneous pneumothorax but a film taken before any needling or aspiration shows a faint but definite fluid level (marked by arrows and difficult to reproduce) in the fluid collection at the right base. The right lung has scattered, soft mottling, especially in the mid. zone. Left hilar markings are prominent and there is left apical mottling with honeycomb cavitation.

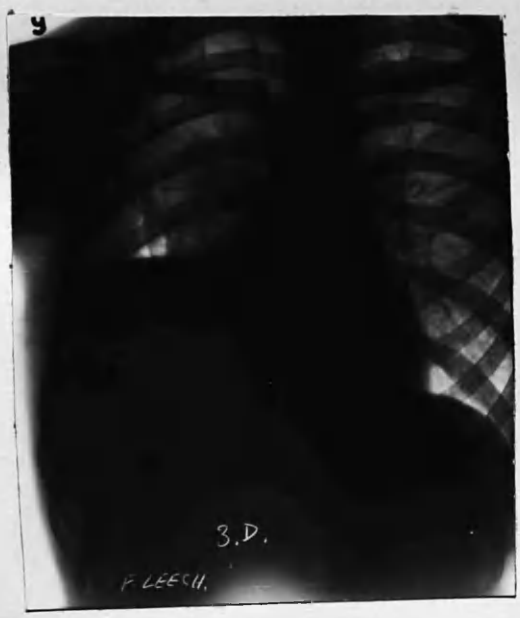
This is probably a case of symptomless and localised spontaneous pneumothorax as shown by the fluid level and slight air cap over it. No Blood Sedimentation Rate records available.

Treatment was by repeated aspirations and Eusol washouts for fifteen months, followed by rib resection over the empyema cavity. She was discharged, with a small residual space, at her own request, and a follow-up shows that she was fully recovered by 1943 and off the register. The total duration of empyema was 12 years but after operation it took ten years nine months for the sinus to dry up.

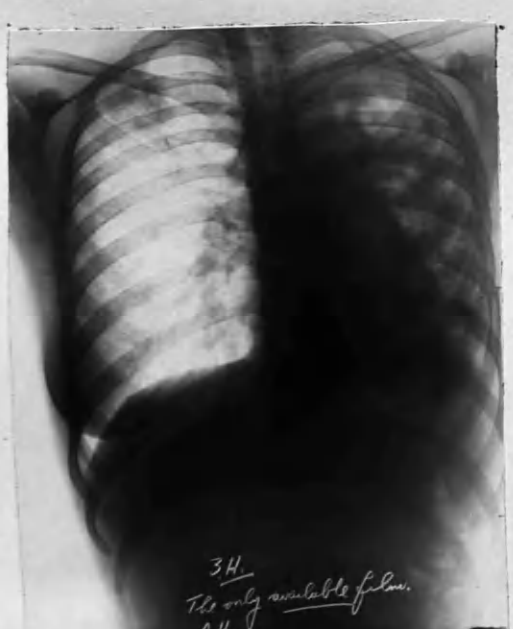
3B. C.R. No.13,520. A male, aged 34 years, and R.B.2 classification at empyema onset. Had pyrexia of 99.6°F. at induction of right artificial pneumothorax. Empyema onset was four months after induction and one month after positive pressure refill to 0 + 7 cms. water. Blood Sedimentation Rate was 23 mms./hour at empyema onset. Treatment was by regular air placement.
Final result was death after the empyema had lasted three years eight months.
An X-ray taken after induction shows a right artificial pneumothorax present with the right upper lobe completely airless and "ground-glass" like and strung up to the

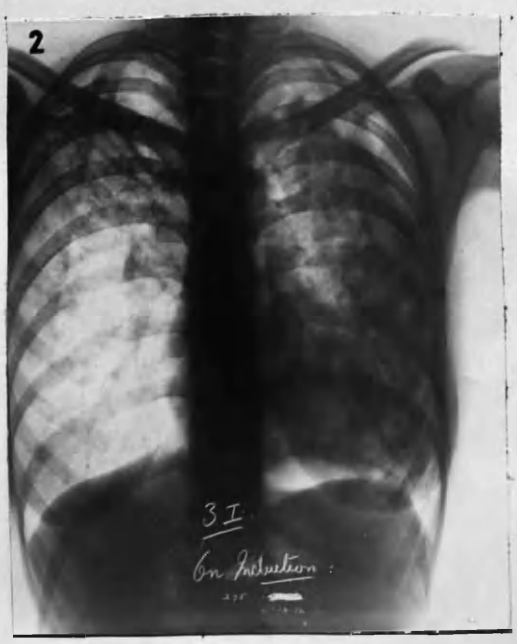

3A.


3.B.


3.D.
F LEECH.


3E.
A. KELLY.


3H.
The only available film.
A.H.


3 I
On Instructions

3B. C.R. No.13,520 (continued):-

apex of the chest. There is a large vomica in its superior part. The right lower lobe is aerated but small in volume and has a small round focus (not nipple) in its lower margin. Costo-phrenic angle is clear. This is a contraselective artificial pneumothorax and taken before tuberculous empyema developed. The left lung field has emphysema at the base and prominent apical vascular markings.

3C. H.S. No.13,152. A male, aged 26 years, and R.B.2 classification at empyema onset.

Pyrexia of 99.2°F. at left artificial pneumothorax induction. Empyema came on 5½ months after induction but two weeks from a positive pressure refill of -2 +4 cms. water. No Blood Sedimentation Rate record available. Treatment was by one aspiration. The dossier notes indicate that this was a contraselective artificial pneumothorax with cavity still patent. There was also tuberculous disease at the right apex with honeycomb cavitation. No X-rays are available and the patient took his discharge, against advice, still with his empyema.

A follow-up shows that he died in a month, the empyema having lasted for three months.

3D. F.L. No.13,024. A male, aged 18 years, and R.B.2 classification at empyema onset. Pyrexia of 100°F. at induction of right artificial pneumothorax. Empyema onset one month from induction. No positive pressure refills and no adhesiotomy. Treatment was by air replacement at first and later gelatine and flavine injections. No Blood Sedimentation Rate records.

A film taken soon after the right artificial pneumothorax induction shows a right hydro-pneumothorax is present. The right artificial pneumothorax is densely adherent in the upper and clavicular areas. There is air between the right upper lobe and upper mediastinum. A large vomica is evident level with right hilum and

3D. F.L. No.13,024 (continued):-

above the fluid level. Left lung appears clear.

This patient took his discharge, against advice, but a follow-up reveals that he died the following day, his empyema having existed for seven months.

3E. A.K. No.12,481. A male, 27 years, and R.B.2 classification at onset of empyema. Pyrexia of 101°F. at left artificial pneumothorax induction. Empyema onset was nine months after induction but three weeks after positive pressure refill of -3 +6 cms. water. No Blood Sedimentation records. Treatment was by a few irregular aspirations when necessary, and Sanocrysin systemically. Death ensued in 26 months from empyema onset.

The only available film taken when the empyema is established shows marked scoliosis - concavity to left and tiling of left ribs. The right apex has fine calcareous nodules. Right lung is extremely emphysematous and has not reproduced well in the photograph. Trachea, heart and mediastinum displaced to left.

The picture is one of a chronic pyo-pneumothorax. The left upper lobe margin is seen. There is great thickening of parietal and visceral pleura.

The left lower lobe bronchi are apparently bronchiectatic in the original X-ray; it looks as though the left lower lobe has become atelectatic on production of the artificial pneumothorax with probable resulting pneumonitis, empyema, carnification of left lower lobe and bronchiectasis.

3F. H.K. No.13,146. A male, 28 years, and R.A.1 at the onset of the empyema. This empyema commenced as a right pleural effusion with minimal parenchymal disease. The dossier records indicate that the empyema developed pari-passu with the spread of pneumonic parenchymal disease. He was eventually a proved tuberculous case. No Blood Sedimentation Rate records available and no X-ray photographs. Treatment was by 7 aspirations in all, for original fluid and later pus, at monthly

3F. H.K. No.13,146 (continued):-

intervals. The empyema proper only lasted for two months and toxaemia and death ensued. There is no clinical indication that this was a case of spontaneous pneumothorax.

3G. T.W.H. No.13,676. A female, 24 years of age, and R.B.3 classification at empyema onset. Temperature of approximately 98.8°F. each evening at induction of left artificial pneumothorax. Empyema came on four months from induction but only two weeks from a refill of -3 +4 cms. water. Treatment was by regular aspirations and air replacement. No Blood Sedimentation Rate records. Death from toxaemia resulted in eleven months.

No X-ray films are available but the dossier screening records indicate a contraselective left artificial pneumothorax with atelectasis and cavitation.

3H. A.H. No.13,590. A female, 17 years, and R.B.3 classification at empyema onset. Pyrexia of 100°F. at left artificial pneumothorax induction. Onset of empyema was six months from induction but only six weeks from a positive pressure refill of +4 +6 cms. water. No Blood Sedimentation Rate records. The treatment was to abandon the artificial pneumothorax on the onset of empyema. No aspirations were made after one for diagnosis. Death resulted from toxaemia fourteen months from the development of empyema. An X-ray when the empyema had existed for some months shows some fine mottling throughout right lung with apical cavitation. Trachea, heart and mediastinum displaced to the left where the appearances suggest grossly thickened pleura with a peripheral zone of fluid present. Extensive cavitation seen in left upper and mid.zones. Bronchus draining left upper lobe vomica is tortuous.

3I. L.W. No.13,506. A female, aged 32 years, and R.B.3 classification at empyema onset. Pyrexia of 99°F. was present when the left artificial pneumothorax was induced. Empyema resulted six weeks from induction and

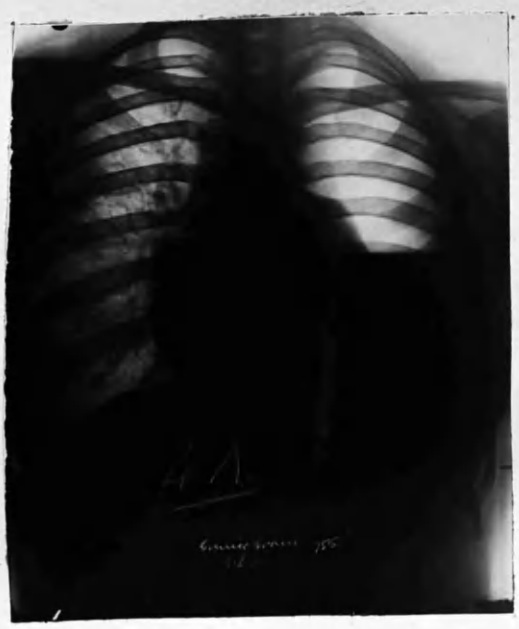
31. L.W. No.13,506 (continued):-

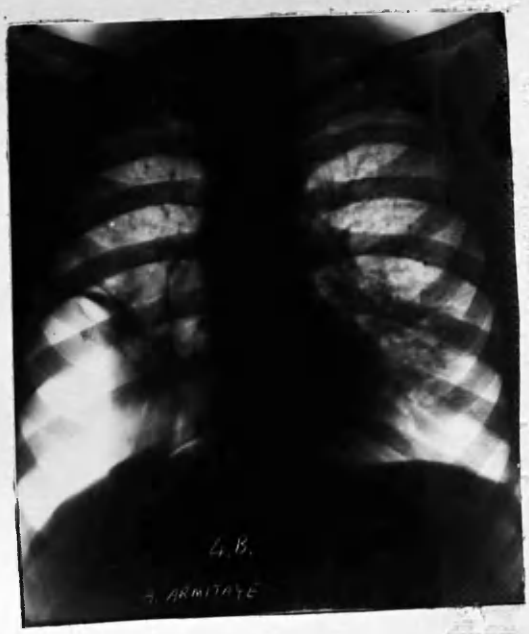
one week from a positive pressure refill of $\pm$0, +9 cms. water. No adhesiotomy done. No Blood Sedimentation Rate records available. Treatment was by air replacements and Sanocrysin systemically. Death from toxaemia ensued in four months. A film taken after induction shows extensive soft mottling of the right upper and mid. zones with early sub-clavicular cavitation. A shallow left artificial pneumothorax is present mostly infra-clavicular in position. Left lung is widely adherent apically and at mid.zone to chest wall. Left lung has extensive soft mottling and is opaque and like "ground-glass" at the apex around a patent vomica.

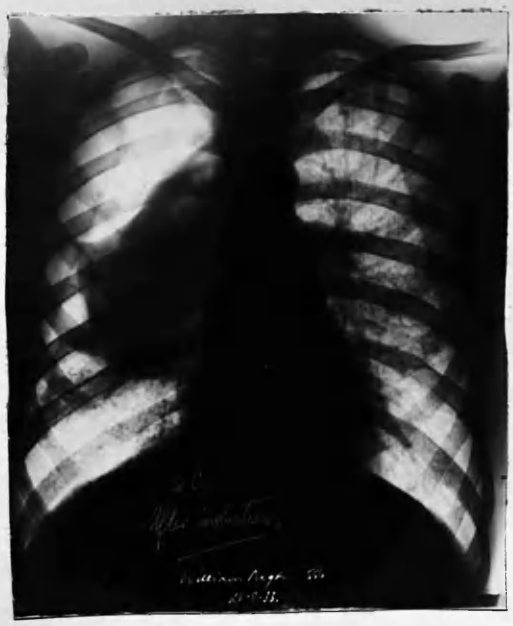
1934 - 36.

4A. E.E. No.14,264. A female, aged 27 years, and R.B.2 classification at onset of empyema. This empyema began after an illness diagnosed as left pneumonia and pleurisy. No sudden catastrophe to indicate spontaneous pneumothorax. Blood Sedimentation Rate 21 mms./hr. at onset. A film before aspiration shows (right lung) minimal mottling of mid.zone with early cavitation in the third interspace. A left hydro-pneumothorax is present with an opaque and milky collapsed left lung. This then is presumably a case of silent spontaneous pneumothorax. Treatment was by four aspirations and gas replacement over three months. She took her own discharge with empyema present and general condition deteriorating. A follow-up reveals that she could not be traced by the Clinic after her discharge.

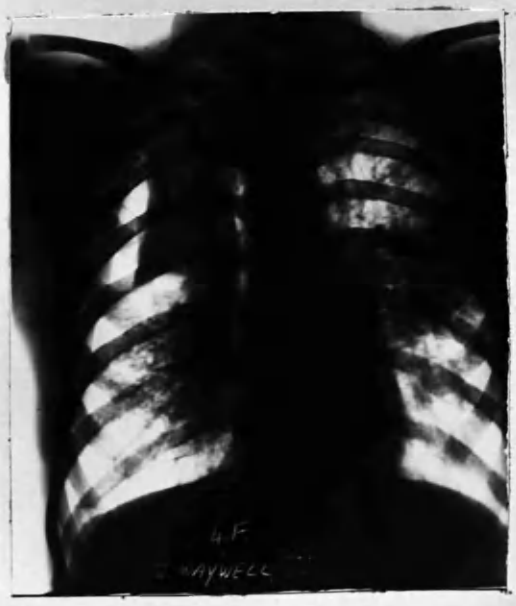
4B. A.A. No.12,889. A male, aged 25 years, and R.B.2 classification at onset of empyema. Blood Sedimentation Rate 18 mms./hour when empyema resulted. Pyrexia of 99.6^{o}F. at induction of right artificial pneumothorax. Clear fluid resulted in 2$\frac{1}{2}$ months and was treated for two years by regular aspirations and gas replacements. Then, two months after a single positive pressure refill to -3 + 5 cms. water, pus developed. Treatment

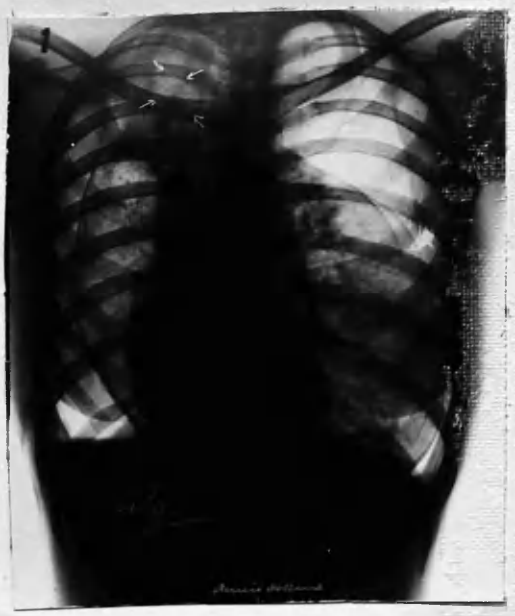



4 B.
4 ARMITAGE




4 D.
After induction & Before
injection of dilated S.P.T


4 F
HAYWELL



4B. A.A. No.12,889 (continued):-

was continued by aspiration and gas replacement. A film taken just after induction shows a basal right artificial pneumothorax is present with no visible fluid as yet. The right lower lobe is collapsed, probably cavitated, adherent to diaphragm and "ground-glass" like. There is slight mottling at right apex. Left side shows mottling in lower zone suggesting a bronchogenic spread.

The patient was transferred to another hospital for right phrenic nerve evulsion. He did not return here but a follow-up reveals that the lung re-expanded after the empyema was in existence for ten months. On 3.4.48 there was no empyema or sinuses. He had absence of toxaemia and fairly good general condition.

4C. W.P. No.13,940. A male, aged 25 years, and R.B.2 classification at empyema onset. Blood Sedimentation Rate 20 mms./hour at onset. Pyrexia of 99.4°F. at the induction of a right artificial pneumothorax. Fluid appeared in four months from induction and pus in one year. There was a single refill to 0, + 10 cms. water, eleven months before the empyema onset. (This is probably not attributable). Treatment was by gas replacement of the fluid and pus. Death occurred in one month from the onset of pus. A film taken after induction shows a right artificial pneumothorax is present with adhesions between base of right upper lobe and apex of right lower lobe and lateral chest wall. Right upper lobe is opaque and has an apical cavity present. There may be collapse of right middle lobe too, in this case. There are calcified nodes at left hilum and scanty mottling left mid.zone. (This artificial pneumothorax was induced for a pneumonic type of disease in entire right upper lobe. A film before the artificial pneumothorax induction shows right apical cavity clearly).

4D. J.J. No.14,490. A male, aged 23 years, and R.B.2 classification at onset of empyema. Blood Sedimentation

4D. J.J. No.14,490 (continued):-

Rate at onset 27 mms./hour. Right artificial pneumothorax induced and no pyrexia at induction. No positive pressure refills. One month after induction there was a clinically demonstrable superadded spontaneous pneumothorax and empyema formed. No adhesion section was done. Treatment was by five aspirations and death ensued in seven months. An X-ray taken after induction shows a right artificial pneumothorax is present. There is extensive calcification of the costal cartilages but apart from this the lower lobe shows some mottling. A trace of fluid is present in the right costo-phrenic angle. Right upper lobe has several patent, large cavities with opaque and "ground-glass" like edges. Apico-mediastinal adhesions are present. The pleura is thickened over the left lung (there has been an artificial pneumothorax here) and a mid.zone cavity with fluid level is seen in the left lung. Left apical cavities are present too. The heart is displaced to the left.

4E. V.H. No.14,566. A female, aged 36 years, and R.B.2 classification at empyema onset. Blood Sedimentation Rate at onset 25 mms./hour. Pyrexia of 101°F. at induction of left artificiial pneumothorax. No positive pressure refills. After nineteen months a superadded spontaneous pneumothorax was evident and empyema developed with bronchopleural fistula. There had been greenish fluid present before this.

Treatment was by irregular aspirations as necessary. Death ensued in nine months.

Films of this case could not be traced.

4F. J.W. No.13,499. A male, aged 30 years, and R.B.3 classification at onset. No Blood Sedimentation Rate records. Pyrexia of 100°F. at induction of right artificial pneumothorax. Empyema ensued six months from induction and two months from a positive pressure refill of -4 +6 cms. water. Treatment was by five gas

4F. J.W. No.13,499 (continued):-

replacements in two years two months when death ensued. A film taken after induction shows a right artificial pneumothorax is present. The right upper lobe is cavitated extensively and of "ground-glass" appearance and adherent extensively in the apico-mediastinal positions. The right lower lobe is aerated but has considerable soft mottling. No free fluid visible. Left lung field has much soft mottling in the mid.zone with probable early cavitation and a small vomica over the second rib anteriorly.

4G. A.H. No.12,411. A female, aged 21 years, and R.B.2 classification at onset of empyema. No Blood Sedimentation records. No pyrexia at induction of right artificial pneumothorax and no positive pressure refills. Empyema developed within three weeks of induction of right artificial pneumothorax. Treatment was by monthly aspirations and air replacements. (She died in $3\frac{1}{2}$ years.. There was a left artificial pneumothorax with greenish fluid at the base when the right artificial pneumothorax was induced.)

A film taken after right artificial pneumothorax induction shows a bilateral artificial pneumothorax is present with fluid at each base but worse on the right. Right upper lobe is semi-opaque and cavitated (arrows) and extensively adherent to chest wall. Trachea is displaced to the right. There is better collapse of left lung but a long apical vomica is visible. There is some mottling of left upper lobe. (The opacity at left lung edge is an artefact).

4H. Z.C. No.12,979. A female, aged 18 years, and R.B.3 classification at empyema onset. No Blood Sedimentation Rate records. Occasional pyrexia of 99.4°F. when right artificial pneumothorax was induced. Empyema developed seven months from induction and six months from the first of several positive pressure refills of approximately -3 +4 cms. water. Treatment was by immed-

4H. Z.C. No.12,979 (continued):-

iately abandoning the right artificial pneumothorax and many aspirations at irregular intervals.

X-rays show that the lung re-expanded, with residual thickened pleura, in two years seven months.

X-ray films show -

4H(1). Pre-artificial pneumothorax film - considerable soft mottling at the periphery and apex of the right lung, with apical cavitation. The inter-lobar septum is elevated and trachea over to the right. There is much emphysema in the left lung which has scanty apical mottling.

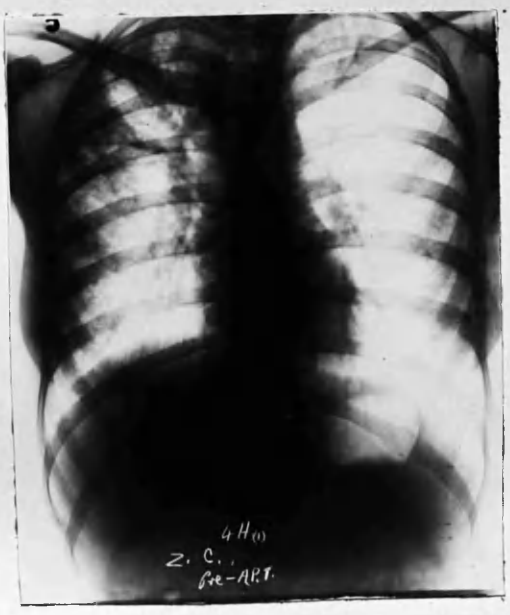
4H(2). 1.8.34. This film was taken after treatment for right empyema, the only remains of which are thickened pleura in the costo-phrenic angle and at the mid.zone where it overlaps the scapular markings. There has been a large spread or flare-up of disease on the left side where there is apical and mid.zone mottling and cavitation. Both hemi-diaphragms are elevated, suggesting intra-abdominal pressure.

Screening records show that the right upper lobe was opaque and cavitated after induction of artificial pneumothorax.

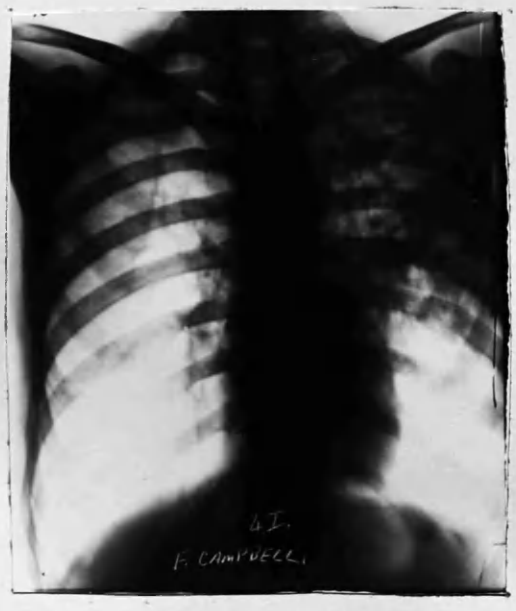
4I. F.C. No.13,606. A female, aged 31 years, and R.B.2 classification at empyema onset. Blood Sedimentation Rate 24 mms./hour at onset. No pyrexia at induction of left artificial pneumothorax. Clear fluid formed five months from the date of induction and pus in nineteen months. Pus formed fifteen months after the onset of positive pressure refills averaging +2 +6 cms. water.

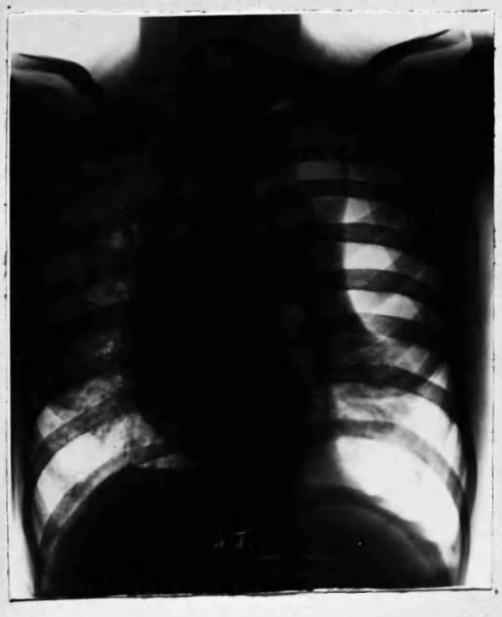
Treatment was by weekly Eusol washouts and air replacements. Multiple chest wall sinuses eventually formed and death from toxaemia a year from empyema onset.

A pre-artificial pneumothorax film which is "soft" and badly focussed, taken before the left artificial pneumothorax was induced, shows scanty mottling at the extreme


4 H (1)
Z. C.,
Pre-A.P.T.


4 H (2)


4 I.
F. CAMPBELL



4I. F.C. No.13,606 (continued):-

right apex. The right and left bases are emphysematous. The left upper and mid.zones have dense pneumonic type of disease and numerous cavities.

4J. J.W. No.13,545. A female, aged 20 years, and R.B.3 classification at empyema onset. Blood Sedimentation Rate 26 mms./hour at empyema onset. No pyrexia at left artificial pneumothorax induction. Empyema developed nine months from induction and eight months after a single positive pressure refill of -2 +4 cms. water which is probably not attributable. Treatment was by a gas replacement of fluid. Later one aspiration was done and finally two air removals. Death occurred from toxaemia six months from the empyema onset.

A film taken after induction shows heart, trachea and mediastinum are displaced to the right. Slight peaking of right diaphragm. Fine hard mottling throughout most of right lung. Left artificial pneumothorax present and no fluid visible yet. The entire left upper lobe is opaque and "ground-glass" like. It has a large apical vomica and is adherent apico-mediastinally.

4K. W.O.P. No.14,122. A male, aged 31 years, and R.B.2 classification at empyema onset. Blood Sedimentation Rate 12 mms./hour at onset. Pyrexia of 102°F. at induction of left artificial pneumothorax. No positive pressure refills. Empyema developed in four weeks. High pyrexia, spread of parenchymal disease in each lung, and death ensued one month from the start of empyema.

Treatment was the immediate cessation of the artificial pneumothorax and one aspiration.

No films are available but the dossier notes from screening records indicate that the left lung was the seat of extensive, progressing and cavitary disease. The right lung also had apical cavitation.

1936 - 38.

5A. E.L. No.15,986. A male, aged 37years, and classification R.B.2 at onset of empyema. Blood Sedimentation Rate at

5A. E.L. No.15,986 (continued):-

onset 22 mms./hour. No pyrexia at induction of left artificial pneumothorax and no positive pressure refills at any time. Fifteen months after induction there was a superadded spontaneous pneumothorax and rapid empyema onset with clinical broncho-pleural fistula. Treatment was by aspirations at irregular intervals at first and later weekly aspirations. The lung failed to expand and she died from toxaemia in eleven months.

No X-ray pictures available but dossier notes indicate a contraselective artificial pneumothorax with patent vomica, throughout.

1938 - 40.

6A. A.T. No.16,908. A female, aged 30 years.

This empyema followed on spontaneous pneumothorax without empyema. X-rays show -

6A(1) 21.6.37. Numerous calcified foci in the right lung field. There is a left hydro-pneumothorax present, the lung being semi-collapsed. Calcified foci can be seen in left mid.zone.

6A(2) 22.8.46. A more penetrating film, 9½ years after her last operation, shows that the right side has remained stationary. The radical nature of the left thoracoplasty is clearly seen.

First in hospital 2.11.36 - 20.11.37. History of pulmonary tuberculosis for previous seven years and spontaneous pneumothorax and empyema in 1933. On admission had left empyema and broncho-pleural fistula, deformity of left chest and sinuses. R.B.3 classification.

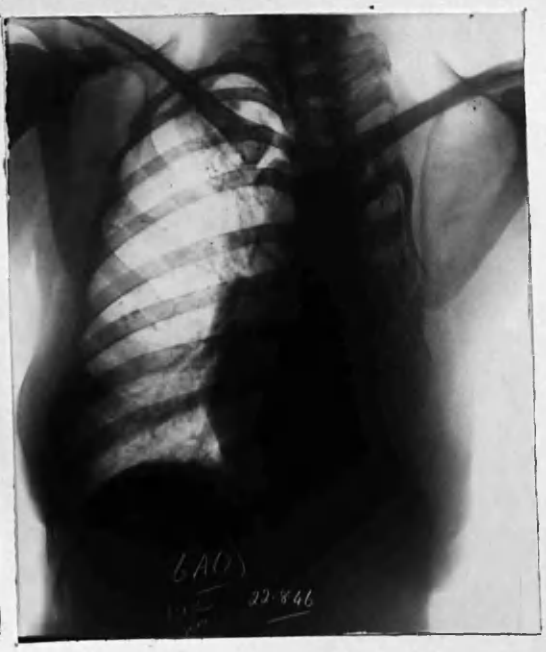
Operation at Withington Hospital (Mr. G. Bryce).

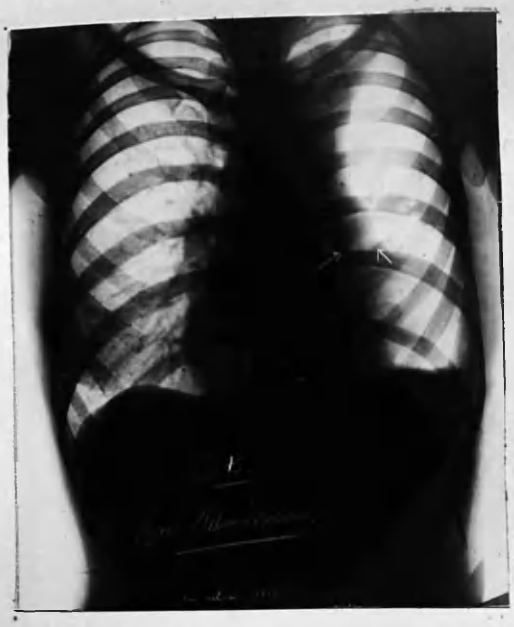
October 28th/1937. Empyema drained in left anterior axillary line. Then total left thoracoplasty in four stages (last operation February 17th/1938). Patient lost over one stone in weight.

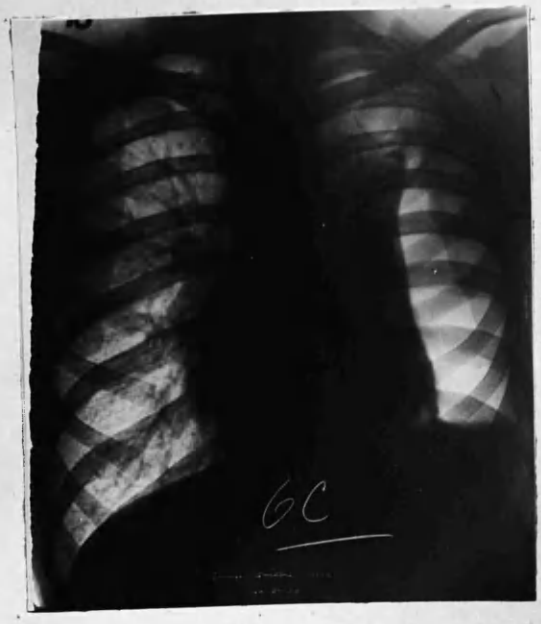
Re-admitted here 4.4.38 - 6.10.38.

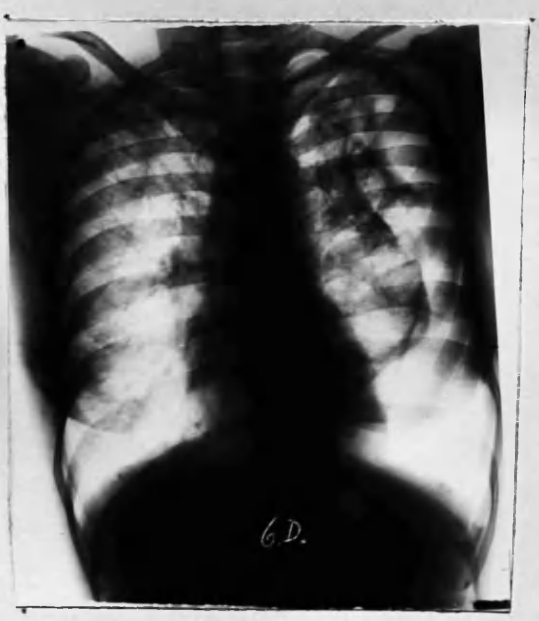
By 24th August/1938 - still tubercle bacilli positive sputum.

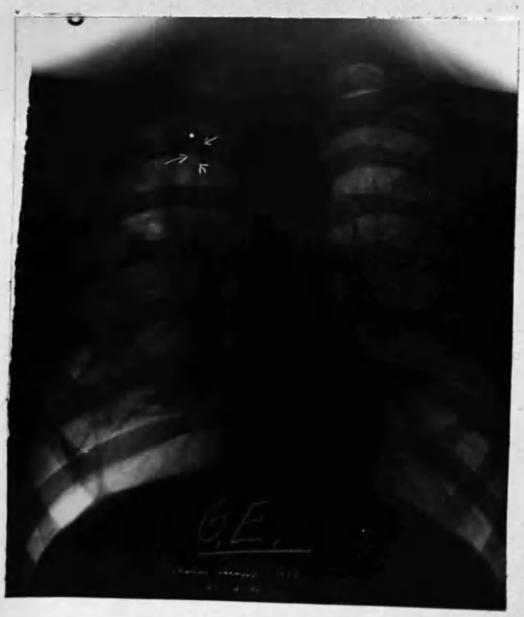

6A(1)


6A(2)
22-846




6C


6D.


6E.

6A. A.T. No.16,908 (continued):-

Sinograms show no evidence of bronchial fistula and a great reduction in size of empyema cavity. Chest sinus still discharges. For an intra-pleural plastic operation at a later date. Clinically, however, at this stage the blowing of a bronchial fistula could be heard.

27.10.38. Operation at Manchester Royal Infirmary. Outer wall of small remaining empyema cavity mobilised and allowed to fall in on the visceral wall. Lung at top end of cavity (site of bronchial fistula) was mobilised so that it was freed from the chest wall.

Baguley 27.10.38 - 1.9.39.

20th.June/1939 still a discharging sinus at lower end of wound.

The patient was evacuated from Baguley on 1.9.39.

Two drachms sputum. Tubercle Bacilli minus. Discharging sinus, weight 11st.1½ lb. compared with 8st.6 lb. on 2.11.36. The sinus healed by November/1942.

A follow-up reveals that by 22.8.46 there were no sinus or toxaemia, no active signs, and her general condition was good.

The empyema lasted for nine years all told, before being cured by the operation.

6B. W.M. No.16,469. A female, aged 40 years, and R.B.2 classification at onset of empyema. Blood Sedimentation Rate not recorded. This patient's left empyema followed a left artificial pneumothorax induced in another institution. No detailed records of the early artificial pneumothorax are available but a film on admission here shows a clear right lung field. A left hydro-pneumothorax was present. The left lung was extensively collapsed and of "ground-glass" opacity. No apical vomica was seen but extensive apico-mediastinal adhesions. There was a mid.zone, large vomica, present with an adhesion holding it out to lateral chest wall (arrows).

This then had been a contraselective artificial pneumo-

6B. W.M. No.16,469 (continued):-

thorax with opacity and patent vomica. There was no thoracoscopy. Empyema followed within six months of induction of left artificial pneumothorax.

Treatment was by gas replacements for several months at first and later saline washouts and repeated aspirations. The lung failed to re-expand. A cold abscess and two sinuses developed in left chest wall.

She was evacuated in September 1939 and by January/1941 was described as being in "fair general condition only". Alive on 3.4.48 and a follow-up shows that "she attends Tuberculosis Dispensary regularly. Weight maintained and general condition is satisfactory. She is able to do a moderate amount of housework. One of the sinuses healed up just after she was discharged from Baguley but the other is still active and produces about two ounces of discharge in the 24 hours. X-ray examination shows an opacity over the left side due to thickened and calcified pleura. The right lung remains clear."

This empyema then has lasted for 13 years to 3.4.48.

6C. E.G. No.14,786. A male, aged 25 years, R.B.2 classification and Blood Sedimentation Rate 19 mms./hour at onset of empyema. No pyrexia at induction of left artificial pneumothorax. A film taken prior to induction shows extensive soft, broncho-pneumonic type of disease with early cavitation in the left mid.zone. Soon after induction there was a superadded spontaneous pneumothorax on the left side and empyema developed in six days from then, or two months from induction. No positive pressures used at any time.

A film taken at this time shows in the right lung field early cavitation in the second interspace and fine mottling around. A left hydro-pneumothorax is present, the left lung being opaque and milky and extensively adherent apico-mediastinally. Probable cavitation is present.

6C. E.G. No.14,786 (continued):-

Clinically there was a broncho-pleural fistula present and chest wall sinuses and toxaemia eventually formed. Treatment was by frequent aspirations and air replacements at first and later flavine washouts. (He had approximately 80 needlings). The lung failed to expand. On discharge he was deteriorating rapidly, the empyema having existed for over three years then and he died in two weeks.

6D. S.A.E. No.16,286. A female, aged 39 years, R.B.2 classification, and Blood Sedimentation Rate 28 mms./hour at empyema onset.

No pyrexia at induction of left artificial pneumothorax. She had positive pressure refills of -4 +5 for a few weeks on one occasion. Fluid, non-purulent, developed within one month of induction and pus in a further nine months, i.e., the empyema onset was ten months from induction or eight months from positive pressure refill. Treatment was by weekly air replacements but the lung failed to expand and she died from toxaemia in five years eleven months from empyema onset.

A faded film, taken after induction, shows discreet mottling in the right upper and mid.zones. A contra-selective left artificial pneumothorax is present and a trace of fluid in the costo-phrenic angle. There are numerous apical adhesions and several lateral adhesions holding open a cavity in the left upper lobe. The edges of the cavity are semi-opaque and like "ground-glass".

6E. T.H. No.14,803. A male, aged 27 years, and R.B.3 classification at empyema onset. No Blood Sedimentation Rate records available. No pyrexia at induction of right artificial pneumothorax which continued for several years. He was refilled at an outside clinic and had a superadded spontaneous pneumothorax (there is no record of positive pressure refills). An empyema quickly followed, four years after right artificial pneumothorax induction. He had, as treatment, three aspirations with

6E. T.H. No.14,803 (continued):-

gas replacement and died in three months from the onset of empyema with high fever and toxaemia.

An X-ray taken after induction shows on the right side a contraselective artificial pneumothorax with no fluid present. The apex is semi-opaque and cavitated. Lung edge of right upper lobe is hazy but numerous apico-mediastinal adhesions are visible. There is mottling of the right lower lobe and several adhesions to diaphragm. Left hilar markings are accentuated and there is minimal mottling in first and second left inter-spaces.

6F. T.T. No.17,386. A male, aged 16 years, and R.B.2 classification at empyema onset. No Blood Sedimentation Rate records.

A contraselective right artificial pneumothorax was maintained and Tubercle Bacilli positive fluid formed in two months and pus six months, from induction. No positive pressure refills. Treatment was by repeated aspirations but pyrexia persisted and death ensued in two months.

No X-rays available but the dossier notes indicate a contraselective artificial pneumothorax with patent cavity persisting.

<u>1940 - 42.</u>

7A. A.C. No.17,543. A female, aged 21 years, R.B.2 classification and Blood Sedimentation Rate 18 mms./hour at empyema onset. Pyrexia of 99.4°F. at left artificial pneumothorax induction. No positive pressure refills. Fluid soon appeared and empyema developed six months from induction and was treated by left phrenic nerve evulsion, frequent aspirations, and saline irrigations. A sinus formed in the chest wall and the patient took her discharge against advice. A follow-up reveals that she died from toxaemia after the empyema had persisted for four years and two months.

7A. A.C. No.17,543 (continued):-

An X-ray taken after induction shows scanty mottling in the right lung in the first, second and third interspaces. A contraselective left artificial pneumothorax is present with a fluid level over the diaphragm. The left upper lobe is collapsed and like "ground-glass" and adherent apically to chest wall and laterally over a large vomica. It has several visible vomicae in its substance. The left upper lobe has obvious infiltration. Arrows demonstrate one large vomica and its adjacent adhesions.

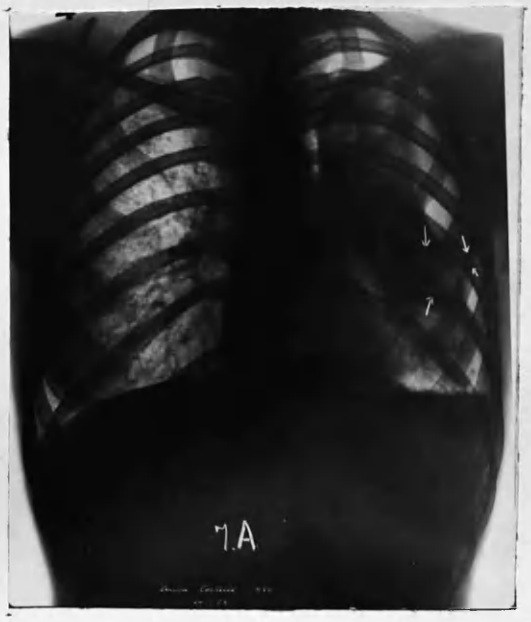
7B. W.C. No.17,270. A female, aged 21 years, and R.B.2 classification and Blood Sedimentation Rate 18 mms./hour at empyema onset. Pyrexia of 99.2°F. at induction of a left artificial pneumothorax. No positive pressure refills. Empyema developed six months from induction and 2½ months from adhesion section when a clinical superadded spontaneous pneumothorax occurred.

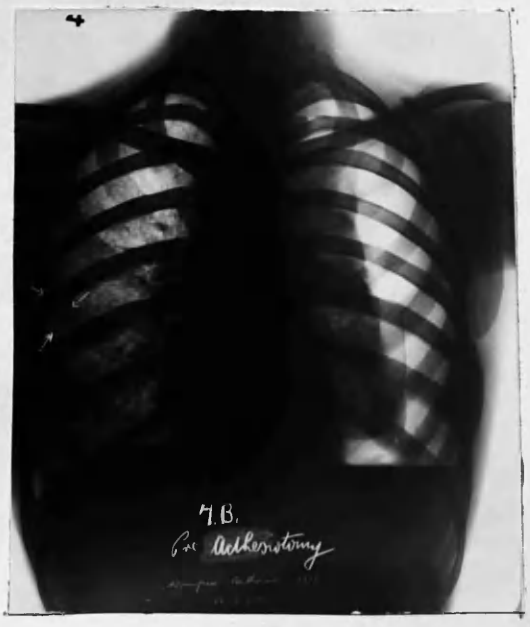
Treatment was by regular aspirations and washouts with Soluseptasine solution. The lung slowly and completely re-expanded, the empyema having lasted for eight months.

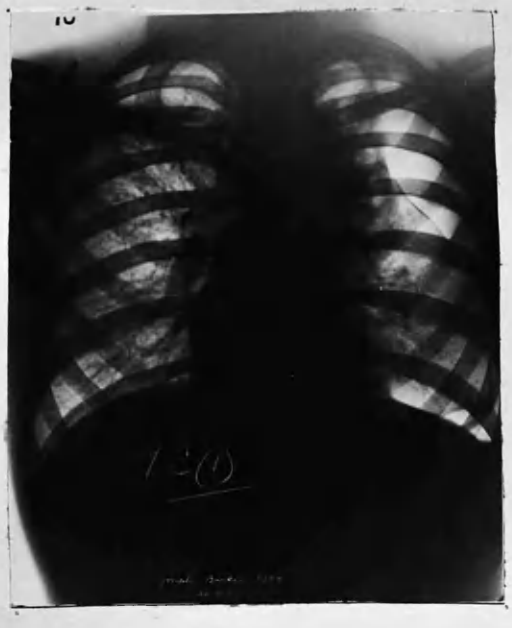
An X-ray shows on the right side the transverse fissure is prominent. It is an old film but a vomica is seen at the site denoted by arrows.

A left artificial pneumothorax is present with a fluid level. Adhesions are seen in the mid.zone area and extensively in the apico-mediastinal field. Part of the left upper lobe around a patent cavity is of "ground-glass" consistency. (A film of 29.4.40, after adhesiotomy, shows increase of fluid. The cavities in the left upper lobe are accentuated and the whole left upper lobe is more collapsed).

A later film on 28.4.41 shows apparently complete re-expansion of the left lung with slight residual obliteration of left costo-phrenic angle.


7.A


7.B.
Pre Adhesiotomy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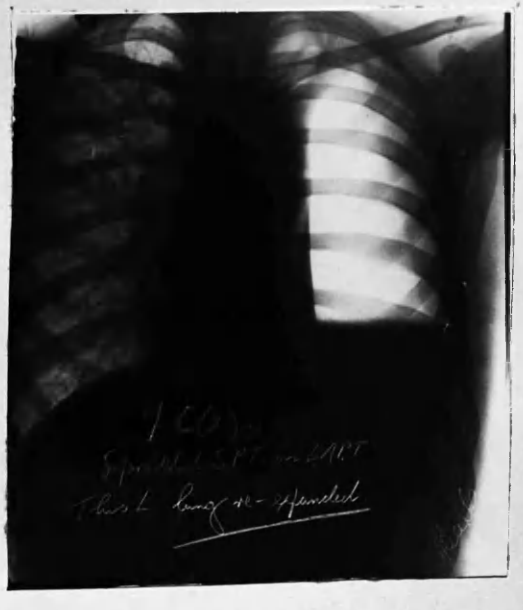
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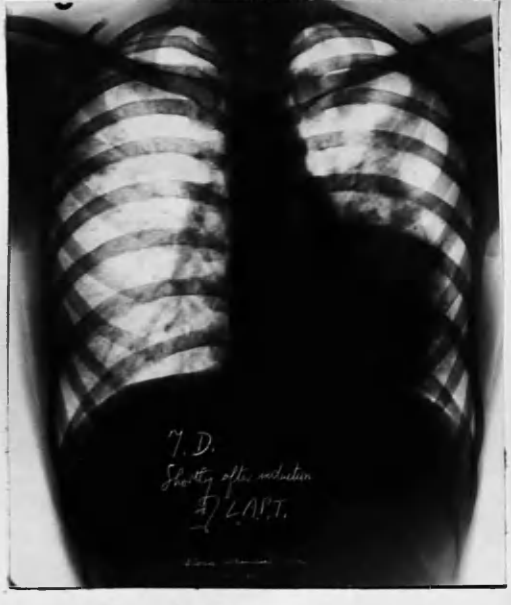

7.D.
Shortly after evacuation
of L.A.P.T.


7.E

7C. J.B. No.17,577. A male, aged 17 years, R.B.2 classification. Blood Sedimentation Rate 12 mms/hour at empyema onset. No pyrexia at the induction of a left artificial pneumothorax. There was superadded spontaneous pneumothorax and fluid formation, pus slowly formed, and the empyema was present three months from induction. No adhesiotomy done.

Treatment was by regular aspirations and the lung completely re-expanded, the empyema having lasted four months.

X-ray films show -

7C(1) 20.10.37. Taken after induction - right lung field shows mottling in the mid.and upper zones with honeycomb cavitation behind the clavicle. A left artificial pneumothorax is present with a trace of fluid. The left upper lobe has several patent cavities with "ground-glass" edges and is adherent apico-mediastinally. There is a small vomica in left lower lobe.

7C(2) Allowing for differences in the texture of the films the right lung field is unchanged. A left hydro-pneumothorax is present with a "ground-glass" like left lung collapsed against the mediastinum which, with heart and trachea, is displaced to the right. A large left upper lobe cavity is obvious.

7D. C.T. No.17,375. A female, aged 21 years, R.B.2 classification, and Blood Sedimentation Rate 26 mms./Hour at empyema onset. Occasional pyrexia of 99°F. when left artificial pneumothorax was induced. Empyema followed in seven months. No positive pressure refills and no apicolysis.

Treatment was by regular aspirations and the lung was almost expanded when the patient took her discharge against advice. A follow-up showed that the empyema continued and she died after three years two months' empyema duration.

7D. C.T. No.17,375 (continued):-

A film taken shortly after left artificial pneumothorax induction shows, in the right lung field, areas of mottling with cavitation behind the clavicle and in the second interspace. A contraselective left artificial pneumothorax is present. There is no free fluid visible. Apical and lateral chest wall adhesions are visible and honeycomb cavitation of the left upper lobe which is opaque in places around the vomicae.

7E. J.S. No.17,699. A male, aged 23 years, and R.B.2 classification at empyema onset. No Blood Sedimentation Records. There was no artificial pneumothorax in this case. The history is that the patient developed a tuberculous pneumonia followed by a simple effusion in India. One air replacement in an army hospital in India was followed by pus. The patient was admitted here with a mixed infection left-sided empyema. A film taken on admission here shows mottling of the pneumonic type in the right mid.zone. There is a left hydro-pneumothorax present (the man has had many aspirations and washouts since his original X-ray). The part of left upper lobe visible shows slight, soft mottling but no gross pneumonia now. The visceral and parietal pleurae are thickening. The original films are not available so that one can only speculate on the cause of this empyema, i.e., ? spontaneous pneumothorax or ? contamination at the first aspiration of a simple effusion. Treatment here was by repeated aspirations and Soluseptasine washouts. The patient took his discharge against advice but a follow-up shows that he died after the empyema had lasted for three years five months.

7F. L.M. No.18,501. A female, aged 20 years, and R.B.2 classification at onset of empyema. No Blood Sedimentation Rate records. The left artificial pneumothorax was induced in another institution and it is not known whether there was pyrexia at induction or positive pressure refills. No adhesion section done. Empyema followed in

7F. L.M. No.18,501 (continued):-

one week and the patient was admitted here. An X-ray shows right lung markings are heavier than normal. Left hydro-pneumothorax present. This left artificial pneumothorax is completely contraselective and the lung is widely adherent apically and laterally to the chest wall. Several visible left upper lobe cavities seen whose edges are milky and "ground-glass" like.

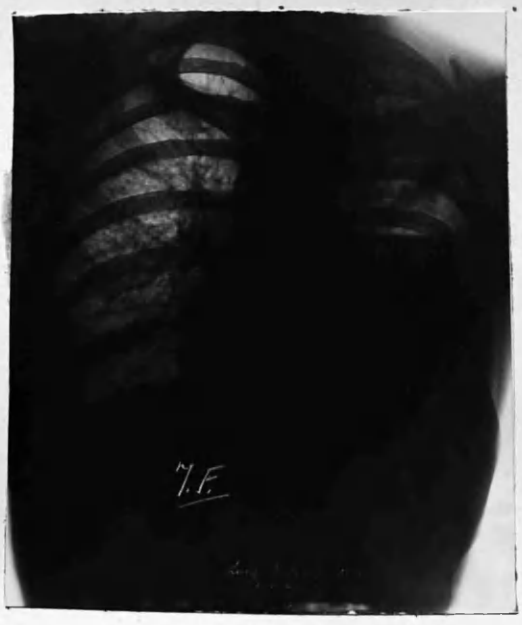
Treatment was by aspirations and saline irrigations; also frequent washouts with methylene blue and ol.menth. pip. for the odour, were used. The empyema persisted, chest wall sinuses formed and death in sixteen months.

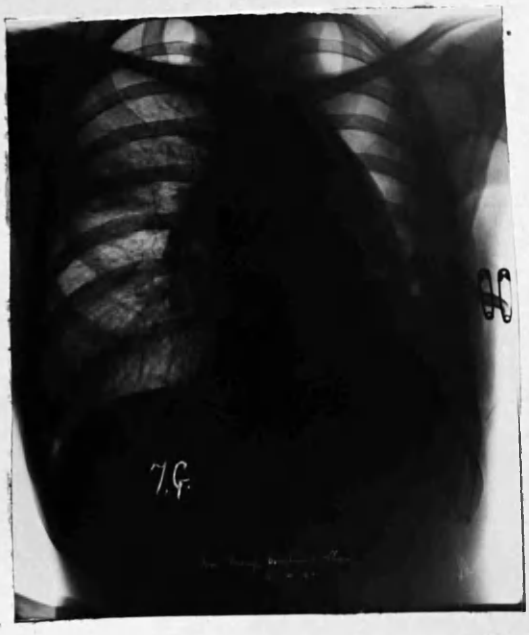
7G. M.C.S. No.18,491. A female, aged 36 years, and R.B.2 classification at empyema onset. No Blood Sedimentation Rate records. This patient had a left artificial pneumothorax present for a year before empyema developed following refills at a clinic. No record of positive pressure refills or temperature at induction.

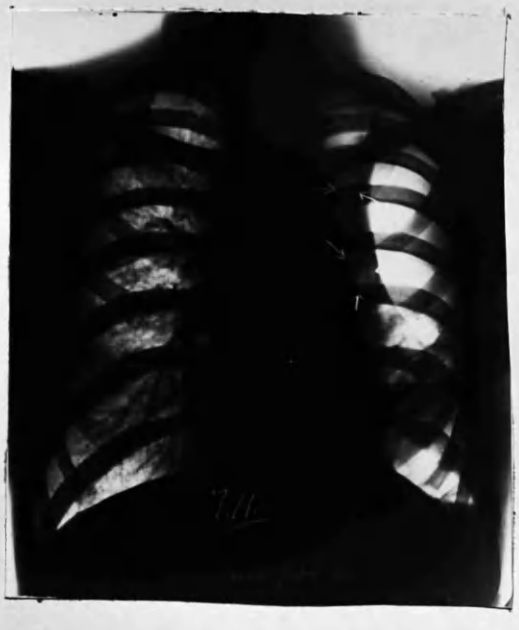
Admitted with an empyema she had previously had no aspirations for eleven months but had secondary infection. She was only in Baguley for one month when she died. Aspiration, followed by the insertion of an intercostal drain, was done on admission. Soluseptasine and acriflavine washouts were used for a month and M.& B.693 tablets orally. There were several chest wall sinuses present finally.

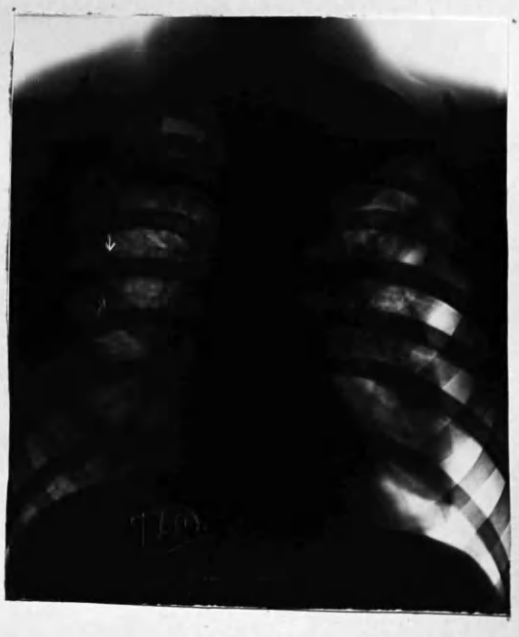
An X-ray taken shortly after admission shows a recent right mid.zone spread of disease with early cavitation. A left artificial pneumothorax is present with fluid level and a self-retaining catheter above the fluid layer. The left lung is collapsed, opaque (like "ground-glass") and adherent apically. At least two large vomicae are present in its length. The empyema persisted for twelve months.

7H. F.B. No.17,688. A male, aged 18 years, R.B.2 classification and Blood Sedimentation Rate 25 mms./hour at empyema onset. Pyrexia of 99°F. at left artificial


7.F.


7.G.





7H. F.B. No.17,688 (continued):-

pneumothorax induction. No adhesion section or positive pressure refills. Empyema onset was 2½ months from induction.

Treatment was by irregular aspirations. Amyloid disease quickly supervened and death in four months.

Films show -

20.5.40 - all zones of the left lung to be extensively infiltrated with soft-looking disease and the appearances suggest many small cavities.

This film(15.10.40) shows scanty, hard mottling on the right side. A contraselective left artificial pneumothorax is present in which there are two fluid levels (one basal and one at level of lower angle of scapula). The left upper lobe is opaque and like"ground-glass" and has at least two large patent vomicae (shown by arrows). There are dense left apical adhesions and adhesions between left lower lobe and chest wall.

7I. J.B. No.17,563. A male, aged 26 years, R.B.2 classification and Blood Sedimentation Rate 23 mms./hour at onset of empyema. Pyrexia of 99.2°F. at induction of left artificial pneumothorax. No positive pressure refills and no adhesion section. Empyema developed in four months. Treatment was by twice weekly aspirations and twelve injections of half cc. of Benzyl Cinnamate. The empyema persisted. There was contralateral spread and death in seven months.

This is an interesting case as it shows the course of events in an artificial pneumothorax done in the face of rapidly spreading disease.

X-rays show -

7I(1) taken shortly after left artificial pneumothorax induction - a definite vomica (arrows) on the right side. A contraselective left artificial pneumothorax is present with a widely adherent left upper lobe and a ballooning vomica whose edges are "ground-glass" like. No free fluid visible.

7I. J.B. No.17,563 (continued):-

7I(2) taken eighteen days later shows further spread of disease in the right mid.zone and the left apical vomica has assumed alarming proportions. (On rupture of this vomica the patient lived for seven months after the empyema formed).

7J. E.B. No.17,720. A male, aged 22 years, R.B.2 classification and Blood Sedimentation Rate 26 mms./hour at empyema onset. Temperature of only 98.6°F. at left artificial pneumothorax induction. No adhesion section and no positive pressure refills.

An X-ray taken soon after left artificial pneumothorax induction shows considerable soft mottling of the right mid. and upper zones with cavitation below the clavicle. A contraselective left artificial pneumothorax is present. The left upper lobe is semi-collapsed and like "ground-glass." It is adherent apically and laterally over a large tense-looking vomica. Left lower lobe is widely infiltrated too. No fluid is present.

There was a superadded spontaneous pneumothorax, and after five months empyema formed. Treatment was by several aspirations and withdrawal of air. Death occurred in two weeks.

7K. S.K. No.17,604. A male, aged 49 years, R.B.2 classification and Blood Sedimentation Rate 15 mms./hour at onset of empyema. No pyrexia at the induction of left artificial pneumothorax which ran for two years with a trace of turbid fluid present, before empyema developed. No positive pressure refills or adhesion section.

An X-ray after induction shows extensive mottling in the right lung. A left artificial pneumothorax is present with a trace of fluid at the base. Left upper lobe is semi-collapsed and opaque. Appearances suggest mid.zone cavitation where the lung is adherent to lateral chest wall. Apico-mediastinal adhesions are extensive.

Treatment was to aspirate the pus at first for several months. Then the empyema was left alone. Pleural thicken-

7K. S.K. No.17,604 (continued):-

ing occurred but the empyema persisted. Chronic bronchitis was evident and death was hastened by haemoptysis. Duration of empyema was 4½ years.

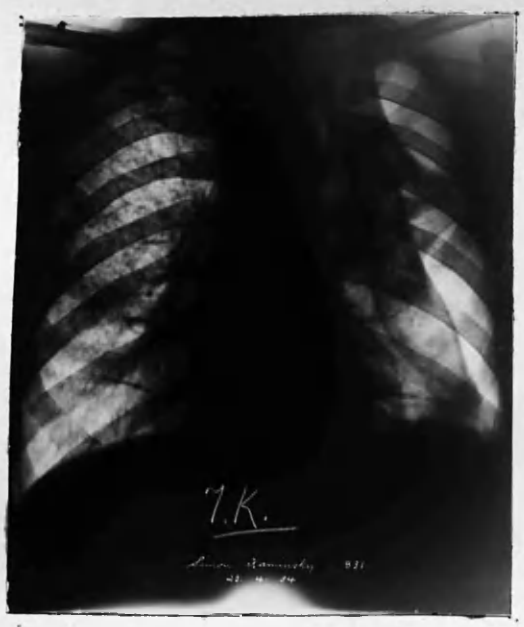
7L. N.P. No.17,866. A female, aged 41 years, and R.B.2 classification at onset of empyema. No Blood Sedimentation records. Pyrexia of 100°F. at induction of right artificial pneumothorax. No adhesion section or positive pressure refills. Regular air replacements were made of the pus, which formed three months from induction. Unexpandable lung ensued and lingering cachexia and death in 7½ years.

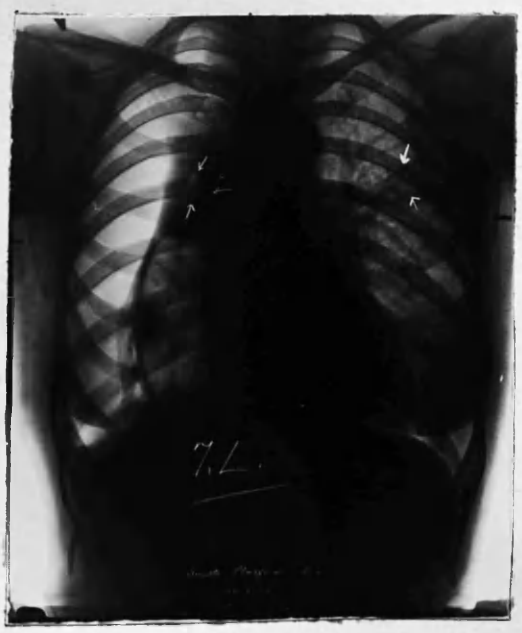
A film before induction shows a broncho-pneumonic lesion with central cavitation in right upper lobe. This film shows a right artificial pneumothorax with early fluid formation in costo-phrenic angle. Visceral pleura is already thickening over right lower lobe where there is a dense area of mottling like a pneumonic focus. Right upper lobe is like "ground-glass" and cavitated. It is adherent apico-mediastinally. There is early cavitation in the left mid.zone. (This increases in subsequent films).

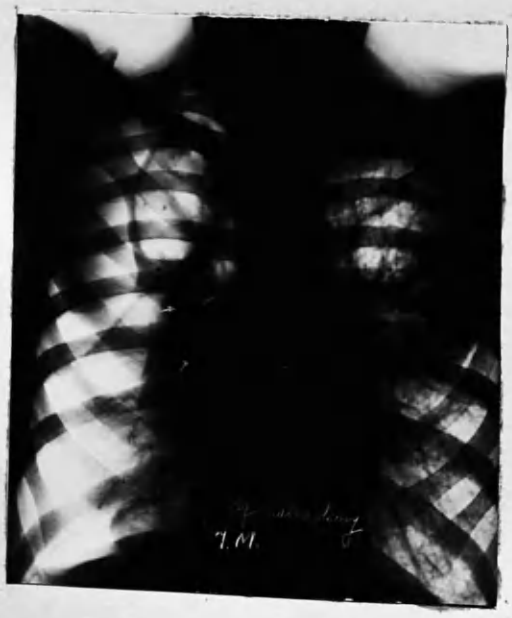
7M. H.S. No.17,295. A male, aged 27 years, R.B.2 classification and Blood Sedimentation Rate 21 mms./hour at empyema onset. Pyrexia of 99.0°F. at right artificial pneumothorax induction. No positive pressure refills. Empyema onset was seven months from induction and three weeks from adhesion section. It is, therefore, attributable to the latter procedure. Massive collapse of lung ensued and cachexia and death in ten months.

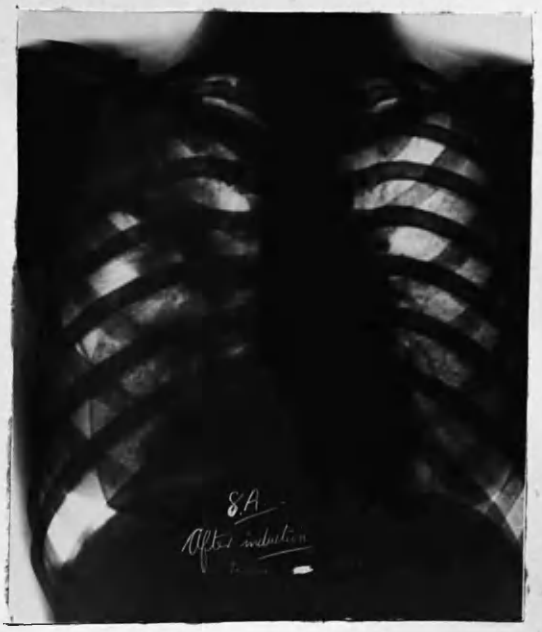
Treatment was by regular aspirations, saline irrigation and Soluseptasine instillations.

An X-ray taken soon after induction shows a contra-selective right artificial pneumothorax is present with a trace of fluid only in the costo-phrenic angle. The right upper lobe is the seat of a giant cavity whose walls are thin and milky-looking and held out to the apex and lateral chest wall by numerous, thin adhesions. The

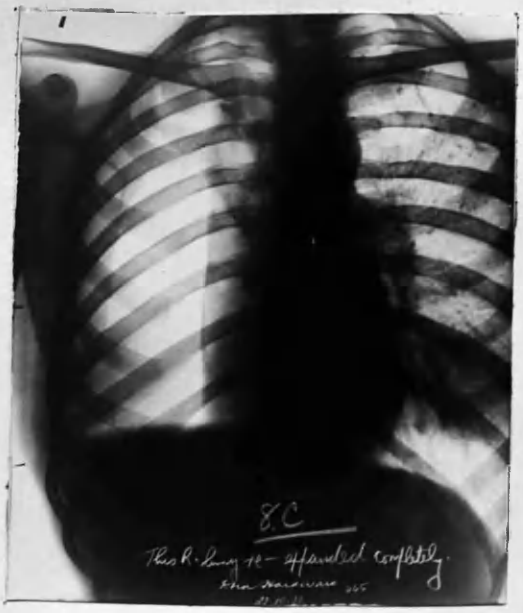

7.K.


7.L.


7.M.


8.A.
After induction


8.B.


8.C.

8B. J.J. No.19,015 (continued):-

up to then. No adhesion sections were done. On the first occasion on which she was given a positive pressure refill of -2 +8 cms. water she developed pus within three weeks and was re-admitted at once.

Treatment was by aspirations until she took her discharge against advice and died in one month, the empyema having lasted for $6\frac{1}{2}$ months.

A film shows right lower lobe is emphysematous and adherent in its lower part to chest wall. There is a fluid level at the mid. zone. Right upper lobe is widely cavitated. The walls of the cavity are opaque and like "ground-glass". Trachea is slightly over to right. The appearances over the left apex suggest a localised pleural effusion. There is much cavitation in the left upper and mid. zones below the pleural opacity. Left lower lobe bronchi are clearly visible and dilated in parts. Slight peaking of the left hemi-diaphragm.

8C. D.H. No.19,196. A female, aged 30 years, R.B.2 classification and Blood Sedimentation Rate 22 mms./hour at empyema onset. There was no pyrexia when a right artificial pneumothorax was induced and no adhesion section or positive pressure refills. Non-purulent fluid was present for a year and then pus formed $1\frac{1}{2}$ years from induction. Treatment was by air replacements regularly at first for two years and then no aspirations from 1935 to 1943. A rib abscess formed at one point and was drained and healed. Eventually, after ten years, the empyema cleared, the lung re-expanded and her general condition was good.

An X-ray taken not long after induction shows a right artificial pneumothorax is present with fluid level. Right lung is collapsed but semi-aerated. Right upper lobe is cavitated and the walls of the vomica are thick and milky. The innermost walls of the cavity are like scar tissue in appearance. Numerous apico-mediastinal adhesions are present. Left lung has scattered calcium

8C. D.H. No.19,196 (continued):-

nodules and lines of hard mottling showing old disease. (Subsequent films show a completely re-expanded right lung with apical cavity persisting and left lung unchanged over several years).

8D. M.B. No.19,197. A female, aged 33 years, and R.B.2 classification at empyema onset. This left artificial pneumothorax developed empyema in six months. She was admitted for treatment with empyema and chest wall sinus, having had aspirations at irregular intervals. Total thoracoplasty was done here.

A follow-up reveals that on 7.4.48 she has a persisting sinus and residual empyema space. Apart from this she is well, free from toxaemia and symptoms of lung activity, and is quiescent otherwise. To date (7.4.48) the empyema has lasted nine years.

In hospital 23.9.1943 - 10.12.1943.

History of left artificial pneumothorax in 1934 and purulent fluid in six months (R.B.2 case). Aspirations done irregularly. Left chest wall sinuses formed 1939. First stage left thoracoplasty at Manchester Royal Infirmary 1939 and then stage two thoracoplasty. Treated at home with pus discharging periodically from her sinuses. Weight 6st. 9 lb. on admission. Sputum T.B. Minus.

24.9.43. Third stage thoracoplasty (Mr. G. Bryce). Pus found during operation by an exploratory aspiration. "U" shaped incision left scapula line at level of tenth rib. Parts of 9th, 10th and 11th ribs resected as far back as the transverse processes. Empyema cavity had anterior and posterior angles in the costo-phrenic sinus. Roof of cavity was $\frac{3}{4}$" thick. A flap of it, about 2 inches square, including rib periosteum, intercostal muscles and pleura, was cut, based posteriorly. A wedge was cut from the inner posterior angle of the flap (Robert's method) and the flap was turned inwards so as to obliterate the posterior end of the lower portion of the cavity. The prepared "U" shaped skin flap was then sewn

to the parietal pleura by Eloesser's method and two gauze packs inserted into the cavity. It was decided that, at the next stage, it would be necessary to mobilise the roof of the anterior and lower pocket, and attempt to close, probably by Robert's method.

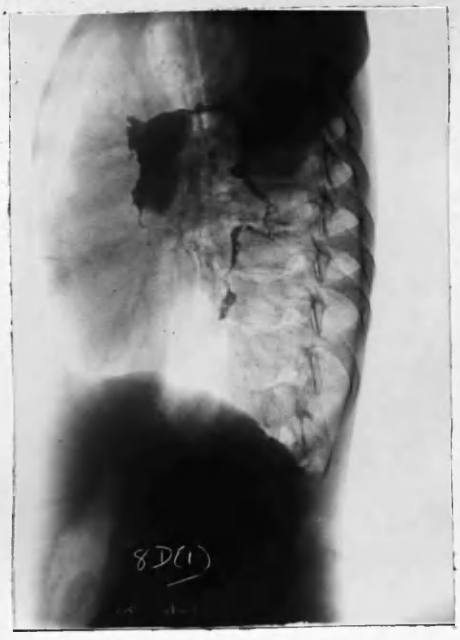
22.10.43. Operation continued (Mr. G. Bryce.) General anaesthesia.

Short perimammary incision connected by curved oblique incision backwards and downwards to the side of Eloesser's flap. Anterior portions of 5th, 6th and 7th ribs completely removed with small portions of cartilage. Roof incised along the anterior border by Robert's method and cut prolonged into the lower and anterior angles so that the roof fell in completely. In the extreme corner of the anterior angle a minute portion of dead bone was found. A large portion of the anterior and lower part of the cavity should have been obliterated by this operation. At the extreme upper end in the axillary region it seemed as if small portions of two more ribs might have to be resected and to do this the approach would have to be made through the upper line of the perimammary incision. The remaining portion of the cavity extended from the axilla downwards and backwards to the Eloesser's flap which afforded adequate drainage. It was thought possible that this drainage might result in the complete obliteration of the remaining portion of the cavity. The wound healed well.

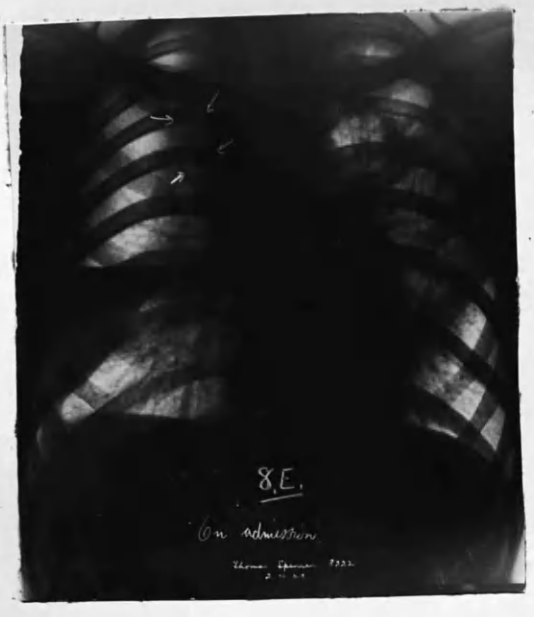
29.11.43. Barium sinogram showed a small space from the axilla down to the diaphragm. It was hoped that this would obliterate.

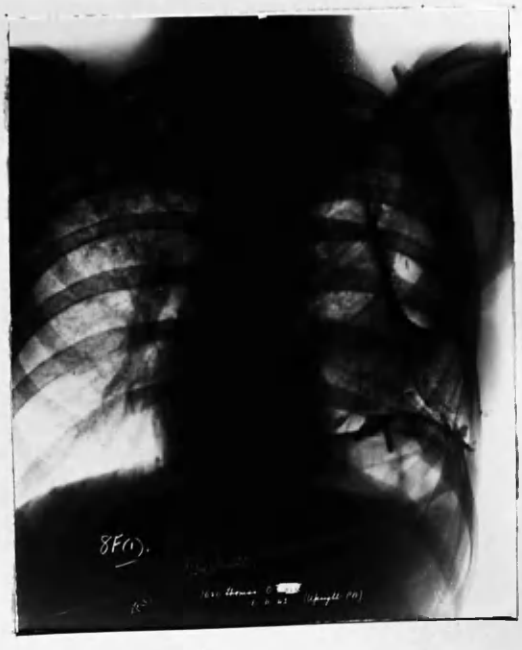
10.12.43. Weight 7st. 1½ lb. Discharged home.

X-rays (sinograms) of 7.4.48 done 4½ years after operation are the only films available here. The extent of the remaining sinus is seen and the radical nature of the previous thoracoplasty.

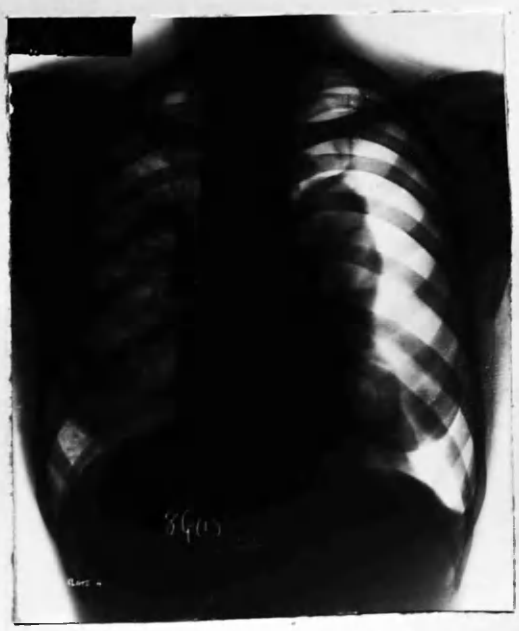

8D(1)


L
8D(2)


8.E.
On admission

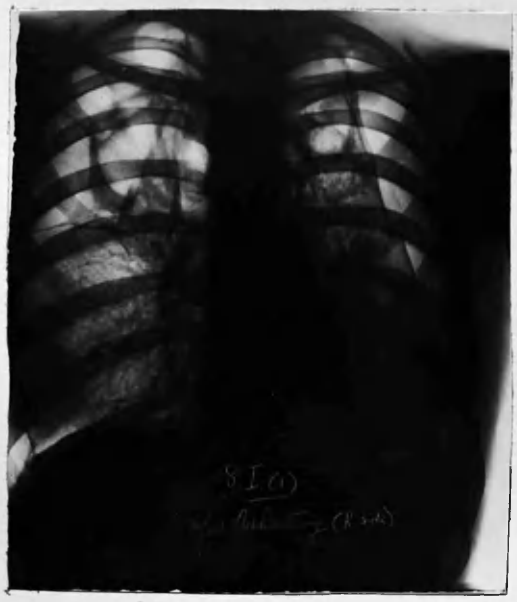

8F(1).

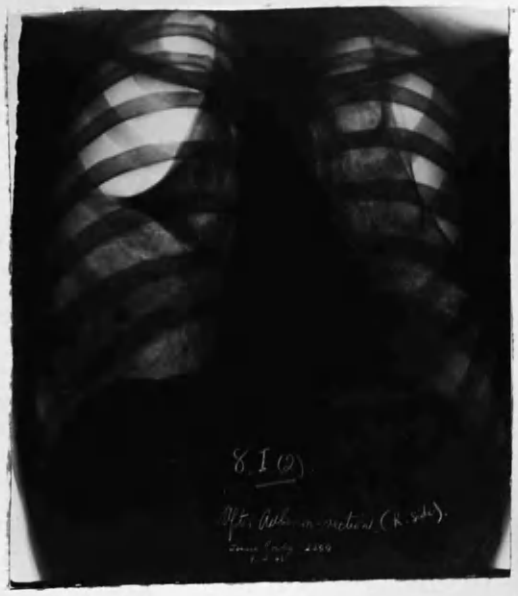

8F(2)


8G(1)

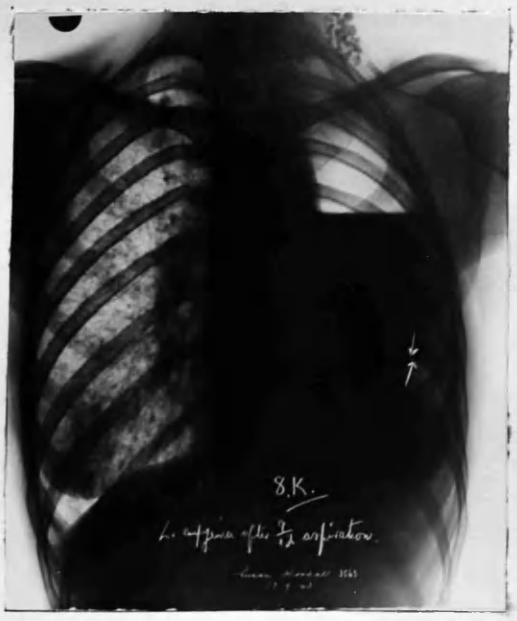

8 G (1).


1
8. H.
Con Induration.


8 I (1)


8 I (2).


8 J
Phas. R lung re-expanded in 6 months


8.K.

8E. T.S. No.18,702. A male, aged 18 years, R.B.2 classification at empyema onset. No Blood Sedimentation Rate record. Admitted with an empyema which developed one year after right artificial pneumothorax induction, he had never had adhesion section or positive pressure refills. Treatment was by aspirations as fluid formed and saline washouts. The lung failed to expand and a chest wall sinus developed. He took his discharge against advice but follow-up reveals that he died in two months, the empyema having lasted sixteen months.
An X-ray taken on admission shows a right artificial pneumothorax with fluid level. The base of the lung has expanded to thickened pleura on the lateral chest wall. The right upper lobe lung markings extend out to chest wall in the infraclavicular region. A cavity is visible below the inner end of the right clavicle (denoted by arrows), i.e., a contraselective artificial pneumothorax. There is much infiltration in the left upper and mid. zones and a large apical vomica is present.

8F. T.O. No.19,166. A male, aged 23 years, and R.B.2 classification at onset of empyema. No Blood Sedimentation records. This man was admitted with an empyema which followed on a tuberculous broncho-pneumonia in a Royal Naval Hospital with no history of spontaneous pneumothorax and no artificial pneumothorax concerned. Aspirations before admission had caused a chest wall sinus.
Treatment here was by continuous suction drain for four months from admission and then an Eloesser's operation. Chest wall sinus and residual space persisted, with cachexia and death, the empyema having lasted thirteen months.
Admitted Baguley 3.9.1943 for operation. Sinus left chest and tender around. Toxic and ill.

6.9.43. Eloesser's Operation for left empyema (Mr.G. Bryce).
General anaesthesia.

"U" shaped incision on top of the eighth rib in the anterior scapular line, with the old discharging sinus

8F. T.O. No.19,166 (continued):-

caught along the incision. Direction and extent of cavity ascertained by probing. Three inches of rib (8th) resected and its periosteal bed incised. Skin flap sutured inwards and upwards. Lower part of wound sutured with thread. Packing of gauze inserted. After this there was slow but definite improvement in his general condition until 15.11.43 when he was transferred to another sanatorium. He deteriorated and died, with marked spread of tuberculosis on the right side on 9.5.1944, the empyema having lasted thirteen months. X-rays taken two months after operation show the extent of the residual empyema space. (The large vomica in right upper zone precludes any further operative treatment).

8G. E.S. No.18,778. A female, aged 26 years, and R.B.3 (larynx) classification at empyema onset. No Blood Sedimentation Rate records. Admitted with an empyema which followed in one month a left artificial pneumothorax induction. No positive pressure refills or adhesion section. Treatment was by closed intercostal drain for thirteen months, then a two stage thoracoplasty and Eloesser's flap operation. The result was persisting chest wall sinus, massive albuminuria (amyloid) and death, the empyema having lasted for nine years. First in another sanatorium 19.11.38 - 9.6.39 (R.B.3) with tubercular laryngitis. Left artificial pneumothorax induced but abandoned as empyema developed in 1½ months. Adhesions numerous in this artificial pneumothorax. 22.11.41. Closed intercostal drainage (continued for thirteen months). Her general condition improved considerably when this was abandoned. Albuminuria marked on admission to Baguley Sanatorium on 19.11.42. First stage thoracoplasty on 12.3.43 (Mr. G. Bryce). (Empyema was four years old). Local anaesthesia. All of the first rib was removed and rib 2 (6.1/3") and rib 3 (8¾"). The first costal cartilage was bitten

8G. E.S. No.18,778 (continued):-

away with Rongeur forceps. A partial mobilisation of the apex was done by division of the periosteum and intercostal bundles of the first, second, and third spaces posteriorly. No extensive strip was attempted in view of the thickening of the tissues and the marked peri-pleuritis. The periosteum was treated with 20% tannic acid.

21.5.43. Second stage left thoracoplasty (Mr.G.Bryce). General anaesthesia.

Old incision re-opened. Ribs 4th (5"), 5th ($4\frac{1}{2}$"), 6th ($7\frac{1}{4}$") and 7th resected partially with their transverse processes. The intercostal bundles were divided and ligatured. Albuminuria persisted.

11.7.43 marked deterioration in physical condition. Several sinuses left chest wall.

16.7.43. Eloesser's Flap Drainage Operation (Mr.G.Bryce). General anaesthesia.

Sinuses explored and enlarged with a probe. "U" shaped incision around the posterior sinus in posterior axillary line. Serratus muscle divided. As the ribs were very oblique it was necessary to resect portions of three ribs and the intercostal bundles in order to turn the skin flap inwards later on. Empyema cavity was opened up and several ounces of pus were sucked out. The anterior chest wall sinus drained the anterior tunnel-like prolongation of the empyema cavity. Skin flap stitched to parietal pleura and serratus muscle sutured. Wound packed with gauze.

She recovered well and by 22.9.43 had a granulating discharging sinus, had gained $10\frac{3}{4}$ lb. in weight since admission. Discharged to Ladywell Sanatorium. She eventually died on 9.1.48, the empyema having lasted for nine years.

X-rays show -

8G(1) the right lung field has scattered mottling in the

8G. E.S. No.18,778 (continued):-

upper and mid.zones but no definite cavitation. A left artificial pneumothorax is present with a trace of fluid in the costo-phrenic angle. The left upper lobe is opaque and has a large vomica in the periphery and "ground-glass" like edges. Some adhesions between the left upper lobe and chest wall are visible;

8G(2) this X-ray, taken $3\frac{1}{2}$ years after her last operation, shows that the disease on her right side has "hardened" considerably with no fresh spread. The extent of her left residual empyema cavity is considerable.

8H. B.A. No.17,475. A female, aged 20 years, R.B.2 classification and Blood Sedimentation Rate 15 mms./hour at empyema onset. No pyrexia at right artificial pneumothorax induction; no positive pressure refills or adhesion section. Empyema developed fifteen months from induction and was treated by frequent aspirations and Soluseptasine washouts. The lung re-expanded after the empyema had lasted twenty months. A follow-up revealed that the patient was able to resume nursing duties. An X-ray after the right artificial pneumothorax was established shows a right artificial pneumothorax is present with fluid at the base. The right middle lobe appears collapsed and like "ground-glass", and an adhesion between it and chest wall is visible. No obvious cavity on X-ray. Left lung field appears clear.

8I. L.R. No.17,627. A female, aged 21 years, R.B.3 classification and Blood Sedimentation Rate 30 mms./hour at onset of empyema. No pyrexia at right artificial pneumothorax induction. Empyema came on fourteen months from induction and eleven months from adhesion section which was only partial, leaving apical adhesions and patent cavities. Nevertheless adhesion section is not considered the causal factor in empyema onset here. A

8I. L.R. No.17,627 (continued):-

refill to positive pressures was given ten months before the empyema onset. Treatment was to abandon the artificial pneumothorax. Two aspirations were done but the patient took her discharge and died in four months, the empyema having lasted for five months.

Films show -

8I(1) taken before adhesion section - a contraselective right artificial pneumothorax with the right upper lobe extensively cavitated. The edges of the vomicae are opaque and like "ground-glass". Numerous adhesions hold out the lung margin to chest wall. In the right lower lobe there is an opaque calcareous focus (? calcified primary focus). There is a trace of fluid in the right costo-phrenic angle. A contraselective left artificial pneumothorax is present with large apical vomica but no fluid visible in the pleural cavity.

8I(2) The right lower lobe has expanded to chest wall. There is more collapse of the right upper lobe but an apical adhesion remains. Right upper lobe cavities are still visible. The left upper lobe vomica is slightly larger than in the previous film.

8J. K.J. No.18,202. A male, aged 19 years, and R.B.2 classification at empyema onset. No Blood Sedimentation Rate records. This man was admitted with empyema which came on eight months from the start of a right artificial pneumothorax induced for haemoptysis. (Non-purulent fluid had existed from a month of induction). There were no positive pressure refills or adhesion sections.

An X-ray taken on admission shows a right hydropneumothorax is present, the lung being flattened against the mediastinum. There is some mottling in left middle and lower zones but no definite cavitation.

8J. K.J. No.18,202 (continued):-

Treatment was by frequent aspirations and the lung finally re-expanded, the empyema having lasted six months (confirmed by X-ray reviews).

8K. S.W. No.19,182. A female, aged 39 years, R.B.2 classification at empyema onset. No Blood Sedimentation Rate records. Empyema onset 2½ years after left artificial pneumothorax induction and two years from adhesion section. No positive pressure refills.

Treatment by regular aspirations. Death in eleven months.

X-rays (13.9.43) show considerable mottling of a soft nature in all zones of the right lung but no definite cavitation. A left hydro-pneumothorax is present, the lung being greatly collapsed. The apex of left lung is adherent to chest wall and there is obvious thickening of parietal and visceral pleura. Left hemi-diaphragm is raised (arrows). (Note the calcified glands on the left side of neck).

8L. I.D. No.18,903. A female, aged 32 years, and R.B.2 classification at onset of empyema. No Blood Sedimentation Rate records. Admitted with empyema for operation. Empyema followed a right artificial pneumothorax done elsewhere.

Admitted 24.2.43. R.B.2 on admission.

In 1932 spent eleven months at Market Drayton. No history of artificial pneumothorax. Attended Tuberculosis Dispensary at Altrincham for several years. Prior to 1932 had bilateral pneumonia.

On admission had right empyema - pus containing tubercle bacilli and pneumonococci. General condition very poor. Blood Sedimentation Rate 37 mms./hour. Pyrexial. Duration of empyema nine years. Treatment was by regular aspiration and lavage with Azochloramide solution, no antiseptic being left in the empyema cavity. The lung expanded up to a point.

30.6.43. Pyrexial upset and signs of increased activity

8L. I.D. No.18,903 (continued):-

right upper zone but this settled by 17.10.43 and remained so until her operation.

29.10.43. Eloesser's operation (Mr. G. Bryce).

General anaesthesia.

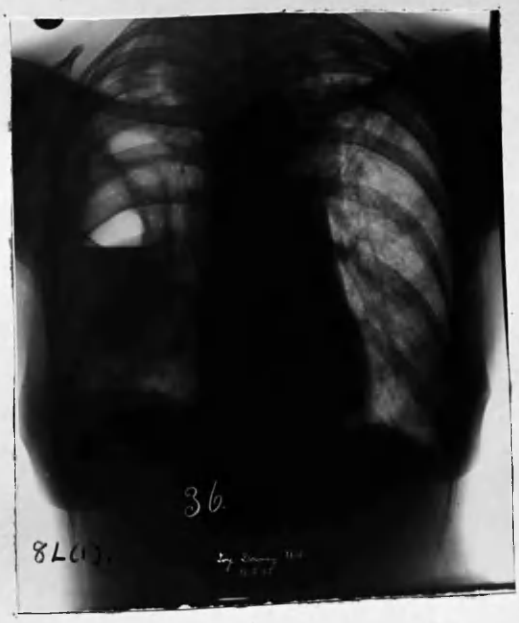
"U" shaped incision in the axillary line along the fourth rib. Skin flap retracted upwards. A few inches of the fourth rib resected. Periosteal bed incised. Pus drained. Skin flap sutured to parietal pleura. Lower part of incision sutured with silk. Small opening left. Cavity packed with gauze. The empyematous cavity seems to be somewhat posterior to the opening underneath the lower part of latissimus dorsi.

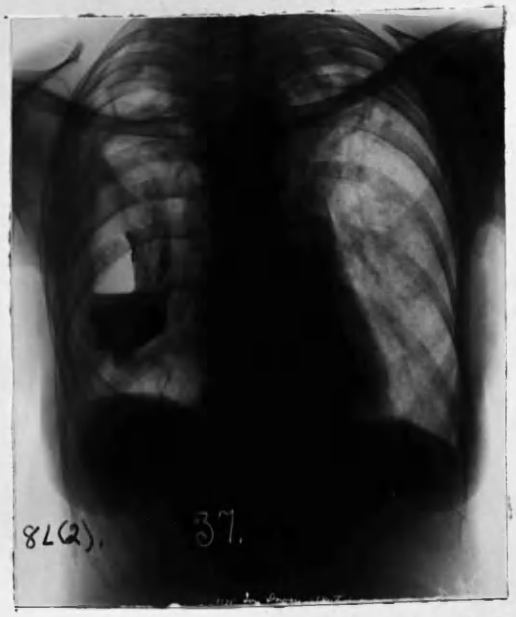
15.5.44. Sinograms show definite diminution in size of cavity.

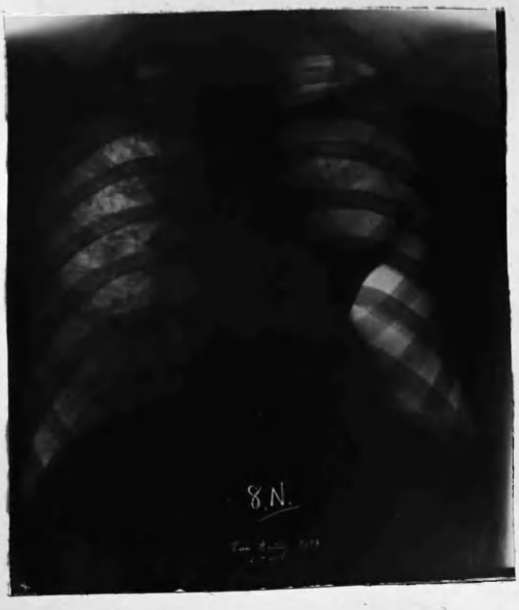
X-ray No.36, 14.6.43. The right lung is collapsed in its mid.zone but expanded to the chest wall inferiorly. Below and above the clavicle the right lung is adherent to chest wall. Marked visceral and parietal pleural thickening is present and fluid in the pleural space (hydropneumothorax). The appearances suggest a right apical vomica. Trachea is displaced to the right. Some "hard" infiltration with cavitation at the left apex.

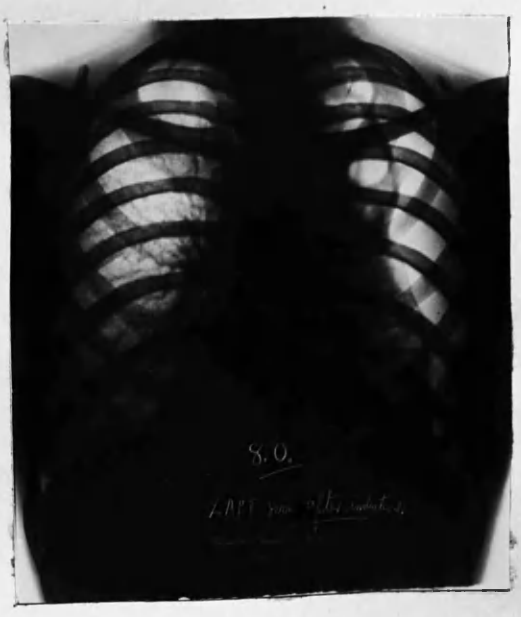
No.37. 11.5.44. Appearances are unchanged, apart from increased right parietal pleural thickening. Lipiodol demonstrates the lower level of the right empyema cavity. After the operation the sinus continued to discharge profusely and she became increasingly pyrexial and cachectic and died on 4.6.1944.

8M. M.K. No.19,151. A female, aged 20 years, R.B.2 classification and Blood Sedimentation Rate 33 mms./hour at onset of empyema. Pyrexia of 100°F. at right artificial pneumothorax induction. No positive pressure refills. Empyema followed 3½ months from induction and 1½ months from adhesion section and is, therefore, attributable to the latter operation. Immediately after adhesion

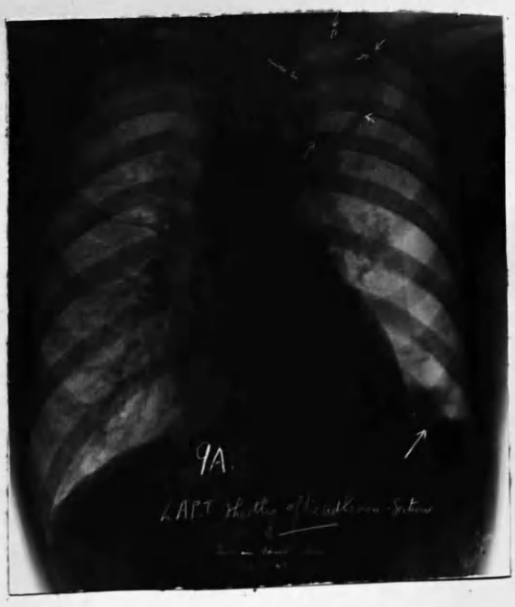

36.
8L(1).


8L(2).
37.


8.N.


8.O.


8.P.


9A.

8M. M.K. No.19,151 (continued):-

section there was increased pyrexia and fluid formation. Treatment was by aspirations and Azochloramide washouts but toxaemia progressed and death ensued in 2½ months. There are no X-rays available but dossier records indicate pneumonic type of disease before right artificial pneumothorax induction and a subsequent contraselective artificial pneumothorax.

8N. E.B. No.18,447. A male, aged 24 years, R.B.2 classification and Blood Sedimentation Rate of 45 mms./hour at empyema onset. A left artificial pneumothorax was induced and carried on uneventfully for three years with no positive pressure refills. Partial adhesion section was then attempted and a mixed infection empyema ensued in one month. Treatment was by intercostal stab drainage and Azochloramide washouts daily. Abscess of the chest wall formed and death 4½ months from the onset of empyema. A film taken after induction shows in the right lung fine mottling in all zones but no definite cavitation. The hilar markings are prominent. A contraselective left artificial pneumothorax is present. In the mid.zone there is a giant vomica with fluid level. Left lower lobe is apparently collapsed. No gross fluid. No lateral film is available to check the site of this vomica.

8.O.S.M. No.18,640. A female, aged 19 years, and R.B.2 classification at onset of empyema. No Blood Sedimentation records. There was no pyrexia at the induction of this left artificial pneumothorax and no positive pressure refills at any time. Empyema ensued nineteen months from induction and ten months from adhesion section and is, therefore, not attributable to the latter operation. After adhesion section the patient was discharged but the empyema came on after clinic treatment and she was re-admitted.

Treatment was by regular aspirations with Azochloramide and saline washouts. Death from toxaemia followed fourteen months from the empyema onset. In the films note

8.O.S.M. No.18,640 (continued):-

a contralateral spread two years after the induction of the left artificial pneumothorax.

A film taken after induction shows evidence of a calcified primary focus in right lower lobe and calcified nodes at right hilum. Increased mottling right lower lobe is probably due to breast shadows. There is slight mottling in the second right interspace. A contra-selective left artificial pneumothorax is present with apical ballooning cavity adherent to chest wall laterally and at the apex. The edges of this cavity are milky and opaque. There is much mottling in the left lower lobe. The left costo-phrenic angle appears clear. (A later film of 15.2.43 shows two cavities in the second right interspace and one at the right apex).

8P. D.S. No.18,530. A female, aged 19 years, and R.B.2 classification at empyema onset. No Blood Sedimentation Rate records. Admitted when her empyema developed, this patient had a left artificial pneumothorax induced in another hospital. No record of pyrexia at induction or of positive pressure refills. Empyema followed in one month. Treatment was by repeated aspirations at first and finally intubation and Azochloramide washouts. Deformity of the chest was marked and death from cachexia ensued when the empyema had lasted for fifteen months.

A film, taken just after her empyema formed (29.5.42), shows right hilar markings are prominent. The mottling from here extends along the line of a prominent right transverse septum. The right upper lobe has fine honeycomb cavitation. A left artificial pneumothorax is present with a fluid level. The left upper lobe is opaque and like "ground-glass" in much of its substance. It is impossible to say from this film (her earliest here) whether there is a vomica in the left upper lobe which is adherent apically and to lateral chest wall.

9A. N.H. No.19,576. A male, aged 33 years, R.B.2 classification and Blood Sedimentation Rate 29 mms./hour at empyema onset. Left artificial pneumothorax was induced and no positive pressure refills given at any time. Empyema ensued five months from induction and one month from adhesion section and is, therefore, attributed to that operation. Treatment was by frequent aspirations at first. When chest wall sinuses formed in 7½ months a stab drain was inserted for ten months. Massive albuminuria and a blood urea of 188 mgms. percent was a terminal event and death two weeks after he took his discharge against advice. The total duration of empyema was 17½ months.

A film taken just after adhesion section shows that the right lung field is clear but the transverse septum is prominent and elevated, and with the slight filling of right costo-phrenic angle this indicates former pleurisy. There is a left artificial pneumothorax present with a trace of fluid. There is still an adhesion apically between apex of lung and chest wall and a large apical vomica is present (arrows). This vomica is very clear in subsequent grid photographs and its distorted draining bronchus is visible (10.8.44).

9B. H.J.B. No.19,656. A male, aged 32 years, and R.B.2 classification at empyema onset. No Blood Sedimentation Rate records. Admitted with empyema for surgical treatment. History of left artificial pneumothorax in 1935 and adhesion section one year later. Superadded spontaneous pneumothorax in 1939 and rapid empyema onset. Therefore empyema ensued four years from induction and three years from adhesion section (also three years from a positive pressure refill). An intercostal drain was inserted in 1939 and the patient worked until admission in 1944. Treatment was by five stage thoracoplasty. Admitted 4.9.1944. Diagnosed pulmonary tuberculosis 1935

9B. H.J.B. No.19,656 (continued):-

and left artificial pneumothorax induced. 1936 left phrenic crush and adhesions cut. Within three months developed some effusion which cleared in a few months. He carried on with positive pressure refills.

1939, superadded spontaneous pneumothorax and mixed infection empyema of rapid onset (Staph. Aureus). He had an intercostal drain inserted soon afterwards in 1939 and continued to work until admission to Baguley Sanatorium. In July/1944 he coughed up blood and pus for one week. On admission Blood Sedimentation Rate 21 mms./hour. Vital Capacity 2,250.

15.9.44. First stage left thoracoplasty (Mr.G.Bryce).

The ribs were very close together and the thoracic cage deformed. Second rib (5½") was first resected and found a difficult task owing to rib tiling and the underlying fibrosis. Then rib 3 (2¼") was resected. Three inches of the first rib were now removed with difficulty. Apicolysis was impossible. An extrapleural fluid collection formed and the patient objected to aspiration.

25.9.44. Wound burst open in three places.

2.10.44. Wound open in mid.part and had a superior sinus. Only a slight serous discharge. No pain. No pyrexia. Sulphanilamide pulv. and flavine dressings.

27.10.44. Second stage left thoracoplasty (Mr.G.Bryce).

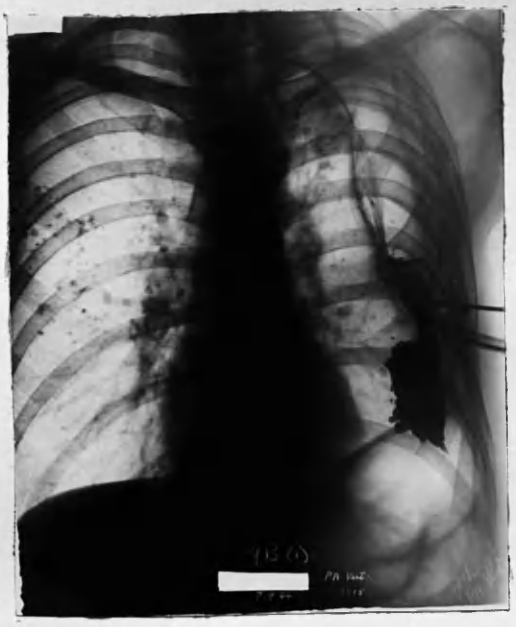
Vital capacity 2,000.

Old incision partially re-opened. Great difficulty in removing the angulated and firmly adherent ribs round the empyema cavity. Segments of third rib (2½"), fourth (6"), fifth (4½") and sixth (6") were resected paravertebrally. Wound closed in layers after insufflation with penicillin-sulphanilamide pulv.

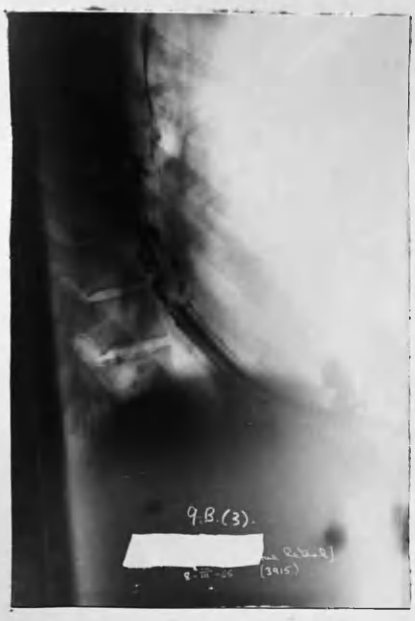
24.11.44. Third stage left thoracoplasty (Mr.G.Bryce). (An antero-lateral stage).

Vital capacity 1,850.

Empyema tube withdrawn. Sinus isolated with mastisol


9B(1)


9B.(2).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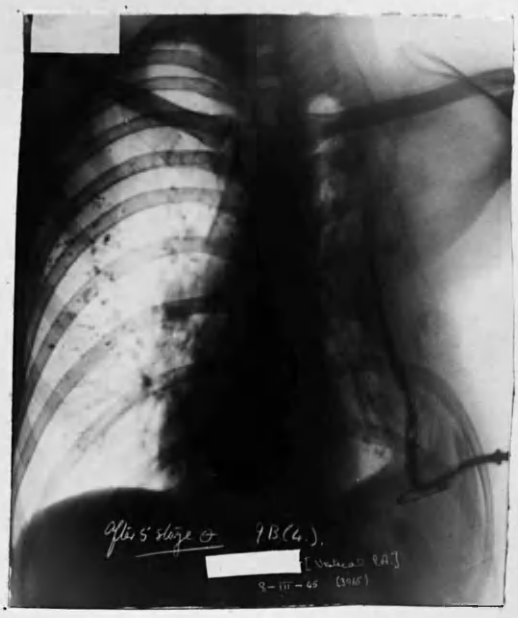
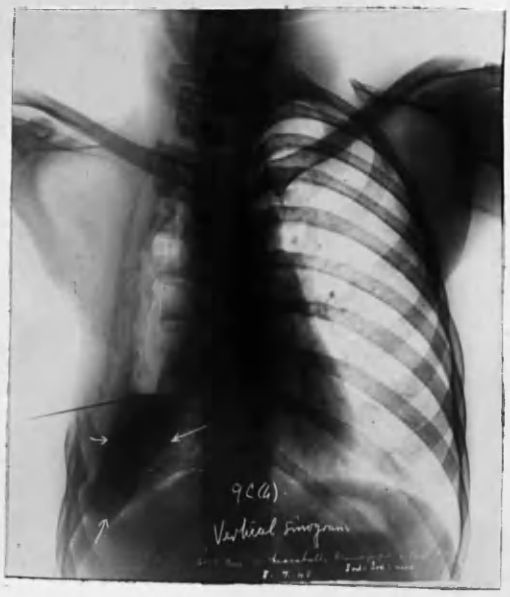
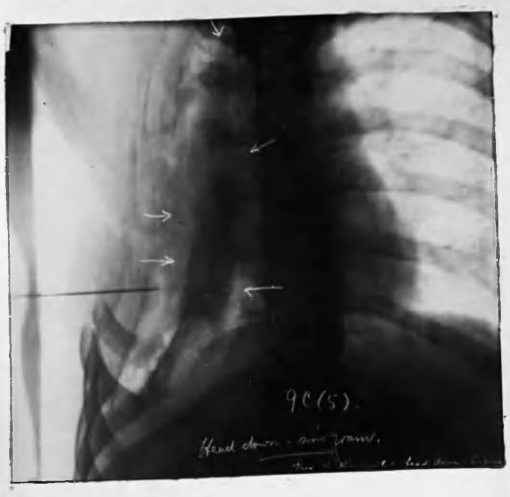

9B(4).

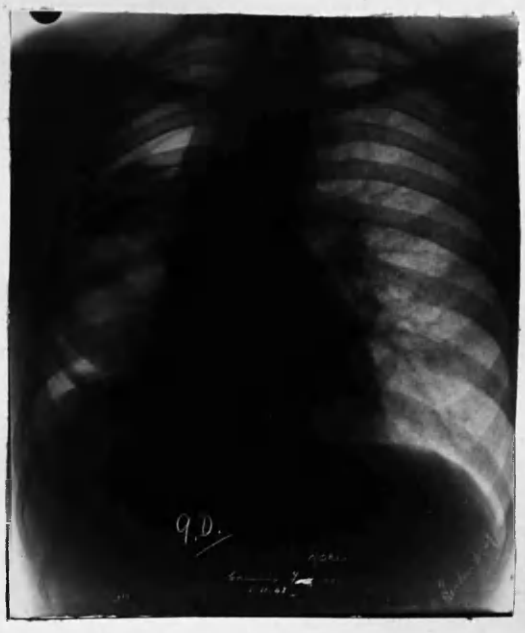

9C(1).
On Admission

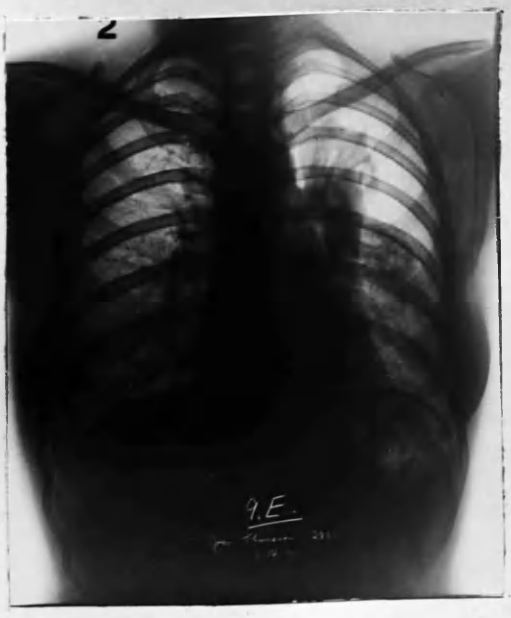

9.C.(2).
R. Lateral before
operation.

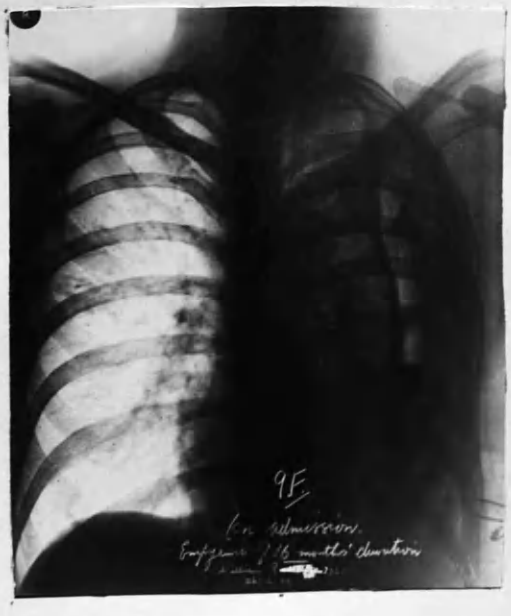

9C(3).
After 4 stage


9C(4).
Vertical Sinogram


9C(5).


9.D.


2
9.E.


9E

 H.J.B. No.19,656 (continued):-

dressing. Small incision in mid.axillary line dividing a few fibres of pectoralis muscle. Again there was a difficult and laborious rib resection over the empyema cavity. Resection done of ribs No.6 (1¾"), fifth (1½"), fourth (2") and third (¾"). Wound closed in layers and the self-retaining catheter re-inserted into the empyema cavity.

29.12.44. Fourth stage left thoracoplasty (Anterior) - (Mr. G. Bryce).
Vital capacity 1,850.

"U" shaped incision, almost peri-mammary, entering the old periscapular incision where there was a small septic collection present. Drainage tube removed and blood-stained pus sucked away. Skin flap dissected upwards and the old empyema sinus dissected outwith its epithelial lining right to the empyema cavity which was opened to allow digital exploration and found to be formed of two main pockets, one anterior, beneath the fifth, sixth and seventh ribs, and the second rather posterior, beneath the scapula. In this stage only the anterior prolongation of the empyema cavity was dealt with and thus portions of the following ribs were resected. Ribs fourth (3¼"), fifth (5"), sixth (7") and seventh (7"). Of those ribs the fourth, fifth and sixth were resected completely, right to the sternum. A fenestrated rubber tube was inserted in the most dependent part of the empyema cavity through the sixth intercostal space, anteriorly, and the old sinus was closed by interrupted stitches in two layers. Wound powdered (sulph. - penicillin pulv.) and closed at both sides of drainage tube. (It was observed that when the posterior pocket of the empyema was dealt with there would have to be a resection of the eighth rib as far forward as anterior axillary line and the seventh, eighth and ninth ribs as far back as their angles. There was regeneration of ribs in the upper axillary region which still held out the cavity but this was probably due to the unresected ends

9B. H.J.B. No.19,656 (continued):-

of the seventh, eighth and ninth ribs).

16.2.45. Fifth stage left thoracoplasty (Mr.G.Bryce).

Lower part of old periscapular incision re-opened. Seventh rib resected from transverse process forwards. Ribs eighth ($6\frac{1}{2}$") and ninth ($5\frac{1}{4}$") resected from their angles forwards. Small rubber tube for 48 hours.

9.3.45. Sputum - concentration test negative for tubercle bacilli.

16.4.45. Discharged home. General condition fairly good. Result - closure of empyema space, apart from a small sinus when discharged. The patient is at work on 5.4.48 but looks ashen in colour and of only fair general condition. Sinus healed. Duration of empyema six years.

Films show -

9B(1) 7.9.44. On admission - numerous calcified nodes in the right mid.zone. A pleurogram is present showing the extent of the left empyema space.

9B(2) 7.9.44. A left lateral pleurogram showing the extent of left empyema cavity. The arrow marks the upper limit of the space.

9B(3) and 9B(4) of 8.3.45 show the extent of the sinus and small residual cavity after a five stage thoracoplasty. The right side is unchanged.

9C. W.M. No.19,886. A female, aged 24 years, and R.B.2 classification at empyema onset. No Blood Sedimentation Rate records. Admitted with empyema which came on three years from right artificial pneumothorax induction but only six weeks from adhesion section. No record of positive pressure refills.

Treatment was by repeated aspirations and Azochloramide washouts at first and later a four stage thoracoplasty.

In hospital 12.3.1945 - 13.8.1945.

Weight on admission 7st. 6 lb.

Right artificial pneumothorax 1939. Adhesion section 1942.

90. W.M. No.19,886 (continued):-

Admitted here for surgical treatment. Pure tuberculous empyema.

23.3.45. First stage right thoracoplasty (Mr.G.Bryce).
Vital capacity 1,700.

Periscapular incision with division of trapezius and rhomboids. Resection of ribs third (5"), second (6½"), and first (3½") with their transverse processes. No apicolysis attempted. 20% tannic acid applied to rib beds. Wound closed in layers after powdering with penicillin-sulphonamide pulv.

Repeated aspiration between all the stages - consistently tubercle bacilli positive.

17.4.45. Second stage thoracoplasty (Mr.G.Bryce).
Vital capacity 1,400.

Periscapular incision enlarged downwards. Remaining fibres of trapezius and rhomboids divided. Resection of ribs fourth (7"), fifth (6"), and sixth (6½") from their transverse processes forwards.

Periosteal beds treated with 20% tannic acid. Sulph.-penicillin pulv. and wound closed in layers.

11.5.45. Third stage right thoracoplasty (Mr.G.Bryce).
Vital capacity 1,200.

Peri-mammary incision. Ribs resected, second (2½") as far as the cartilage, third (4"), fourth (3") and fifth (2") as far back as the previous sections. 20% tannic acid applied to periosteal beds. Wound closed in layers without drainage.

15.6.45. Fourth stage right thoracoplasty (Mr.G.Bryce).
Old paravertebral incision re-opened in its lower end and continued by another incision towards the spine. Fibres of latissimus divided. Resection of 3" of previously divided seventh rib. Then resection of ribs eighth (9¼"), ninth (5½") and tenth (4⅛") from transverse processes forwards. 20% tannic acid applied to periosteal beds. Wound closed in layers with no drain.

9C. W.M. No.19,886 (continued):-

13.8.45. Transferred to another sanatorium, still having aspirations. Not fit for further surgery.

(A review of the films of this case shows that there were three possibilities for this case if she had been fit - (a) extensive resection of the transverse processes and regenerated bone which was not very promising;

(b) treatment by Allison's method;

(c) either of the above, plus tube drainage).

She was left with a residual empyema requiring aspirations, and a spread to the left lung, and rapid fatal termination resulted. The total duration of empyema was three years ten months.

X-rays show -

9C(1) 6.3.45. On admission a right hydropneumothorax is present. The right upper lobe is cavitated and the vomica edges are like "ground-glass". Trachea over to right. There is scanty mottling left mid. zone.

9C(2) 21.3.45. This right lateral film shows pleural thickening and points the position of the right upper lobe vomica.

9C(3) 29.6.45. After a four stage right thoracoplasty a large empyema space, lined with thickened pleura, is clearly seen. Fluid is present. The left side is unchanged.

9C(4) and 9C(5) of 8.7.45 are vertical and head-down Sod. Iodide sinograms showing the extent of the residual cavity.

9D. C.F. No.19,293. A male, aged 25 years, R.B.2 classification and Blood Sedimentation Rate 25 mms./hour at onset of empyema. Empyema ensued sixteen months after right artificial pneumothorax induction and two months from adhesion section. No positive pressure refills given.

9D. C.F. No.19,293 (continued):-

Treatment was by vigorous aspirations and the lung began to expand. He was discharged against advice but treatment was continued outside hospital. A follow-up shows that on 22.3.48 he is extremely well with no sinuses, sputum tubercle bacilli minus, and he is quiescent. The empyema lasted four months.

Films show -

Taken after adhesion section - right artificial pneumothorax with no fluid present. The right upper lobe is opaque, "ground-glass" like and with no visible cavity. A few adhesions still remain between the right upper lobe and chest wall. Left lung is clear. (A film of 8.4.48 shows that the right lower lobe and right middle lobe have expanded to chest wall and opaque right upper lobe is swung up alongside the upper mediastinum. Trachea has come over to right and no vomica visible. Left lung field remains clear).

9E. J.T. No.18,134. A female, aged 23 years, R.B.2 classification and Blood Sedimentation Rate 31 mms./hour at empyema onset. Pyrexia of 99°F. at induction of left artificial pneumothorax. Empyema onset was five months from induction and two weeks from adhesion section due to a sudden spontaneous pneumothorax.

Treatment was by air replacement of the pus for one year, then aspirations and Azochloramide washouts for five months and formalin washouts for two months. The empyema lasted for two years and even then the pleurae are so thickened that there is probably some fluid in pockets.

A film after induction shows right lung field is clear. A left artificial pneumothorax is present and the lower lobe is apparently adherent to lateral chest wall. It shows some mottling and fine cavitation at its apex. Left upper lobe is cavitated and adherent to apex and mediastinum. The edges of the cavities are thick and like

9E. J.T. No.18,134 (continued):-

"ground-glass."

(A film of 24.11.41 after adhesion section shows ballooning cavities in the left upper lobe).

9F. W.R. No.19,439. A male, aged 47 years, and R.A.2 classification at empyema onset. No Blood Sedimentation Rate records. (Although R.A.2 classification when empyema ensued the case was later proved tubercle bacilli positive). Empyema ensued eight weeks after left artificial pneumothorax induction and two weeks after adhesion section at another institution. Admitted here with empyema he had an intercostal De Pezzer catheter introduced and frequent washouts. He died after the empyema had lasted forty months. Chest wall sinuses were present.

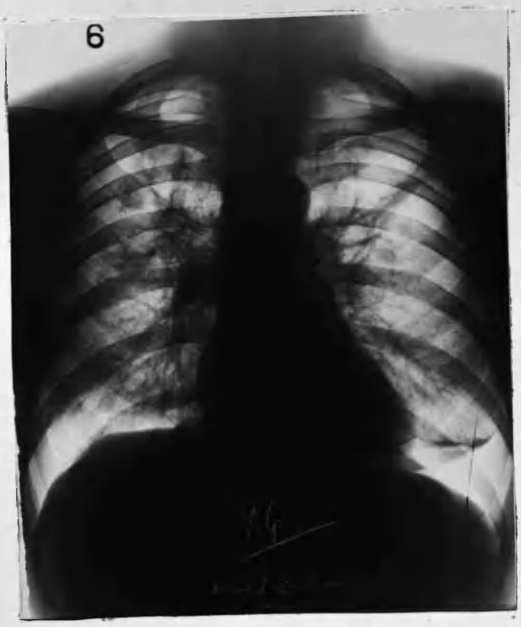
An X-ray taken on admission here shows scanty mottling with early cavitation right middle lobe. Left hydro-pneumothorax present. Left visceral and parietal pleurae are extensively thickened and this obscured the parenchymal condition.

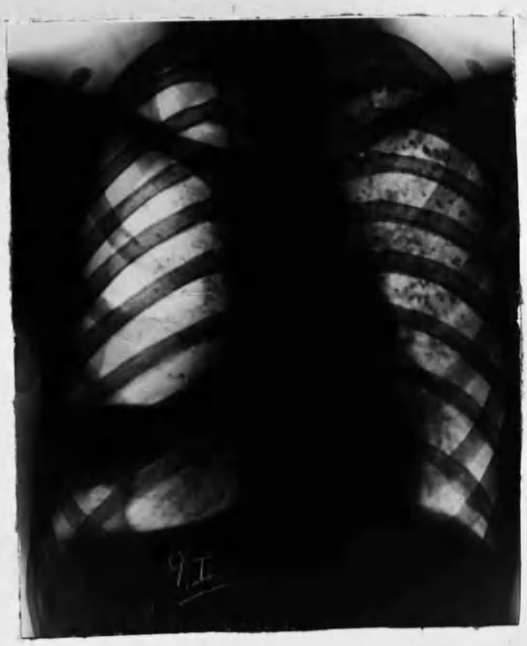
9G. D.J.L. A male, aged 28 years, R.B.2 classification and Blood Sedimentation Rate 17 mms./hour at empyema onset. No pyrexia at right artificial pneumothorax induction and no positive pressure refills or adhesion section.

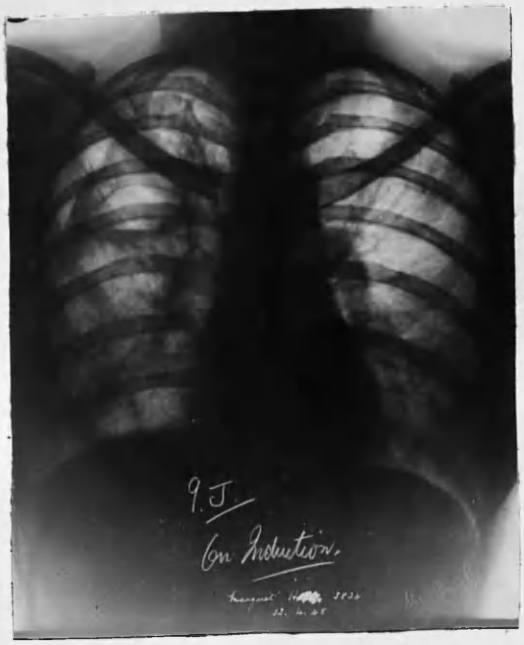
Films show -

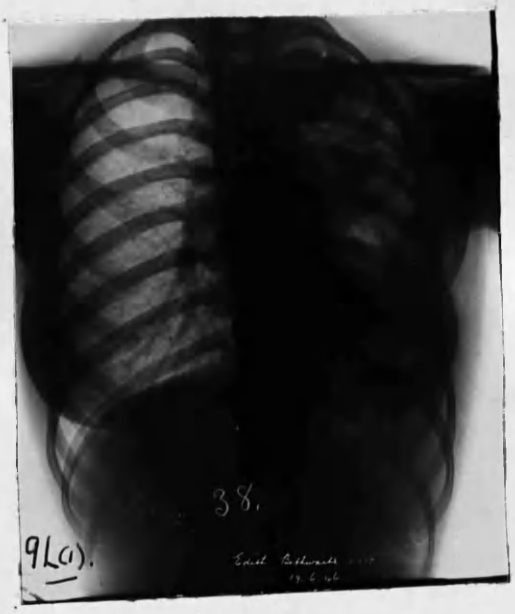
(21.7.41. Film shows a large tension type of right upper lobe vomica caused by the presence of a left artificial pneumothorax which had caused a mediastinal shift to the right).

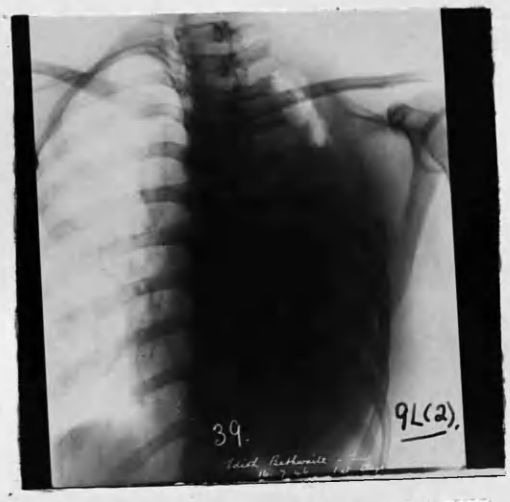
This film, after induction of right artificial pneumothorax, shows the mediastinum in its correct position. The right upper lobe vomica is smaller and is at the level of the anterior end of the second rib. There are right apical adhesions and right mid.lobe appears to be adherent to the chest wall. Left artificial



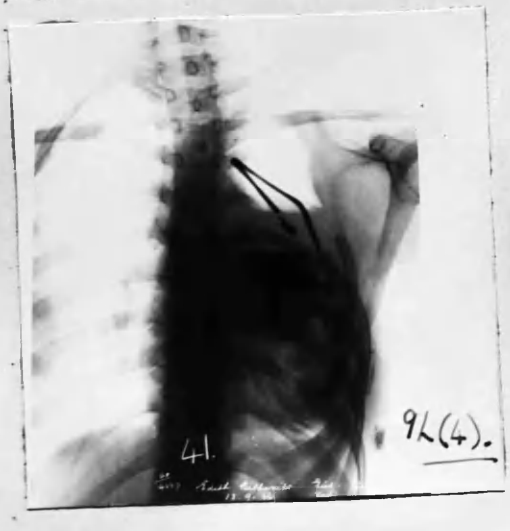

9.I.


9.J.
On Induction.


38.
9L(1).


39.
9L(2).


40.
9L(3).


41.
9L(4).

9H. M.M.L.N. No.17,719 (continued):-

Blackpool for a change of environment.

No films available.

Follow-up reveals that on 3.4.48 she is alive but still has ankle oedema and albuminuria. Chest sinus persists with thin discharge requiring daily dressing, and empyema cavity persists - duration 6 years 1 month.

9I. F.H.B. No.20,106. A male, aged 25 years, R.B.3 classification and Blood Sedimentation Rate 23 mms./hour at onset of empyema. No pyrexia at right artificial pneumothorax induction. The dossier shows that this was a contra-selective artificial pneumothorax with adhesions present. No adhesion section or positive pressure refills. There was a superadded spontaneous pneumothorax and empyema formation two months from induction, and repeated air-replacement was done for seventeen months when the patient took his own discharge in the middle of treatment. He was at home for eight years and returned with empyema necessitatis. Stab drainage was then done but he only lived one week. The total duration of empyema was 9½ years.

X-rays when the empyema had lasted for four years show a right hydro-pneumothorax is present. Its long-standing (four years) is shown by the gross pleural thickening which is evident. The right lower lobe has expanded laterally to chest wall and the diaphragm is flattened. There are numerous calcified foci throughout the left lung but no evidence of cavitation or recent active disease.

9J. M.H. No.19,522. A female, aged 23 years, R.B.2 classification and Blood Sedimentation Rate 20 mms./hour at onset of empyema. No pyrexia at induction of right artificial pneumothorax and no positive pressure refills. Empyema ensued 9½ months from induction but only seven days from partial adhesion section. The apex was adherent and was not touched; several small adhesions only were

9J. M.H. No.19,522 (continued):-

divided. Haemolytic streptococci were grown in the fluid and fulminating haemolytic septicaemia ensued. Treatment was by aspiration. Death ensued, the empyema having lasted four days.

A film shows a shallow right artificial pneumothorax is present and the right upper lobe has a large vomica with fluid level. The cavity walls are like "ground-glass" and numerous apical adhesions are visible. Left apex and first interspace show fine mottling and probable early cavitation.

9K. H.N. No.19,799. A male, aged 21 years, and R.B.3 classification at empyema onset. No Blood Sedimentation Rate records. No positive pressure refills given in a left artificial pneumothorax. Empyema developed 2¼ months from induction but only three weeks from a partial adhesion section done in another institution. He was admitted with a mixed infection empyema (Staph. Albus.) Repeated aspirations were done but cachexia progressed and death followed, the empyema having lasted six months. No films available.

9L. E.M.B. No.20,434.

Admitted 17.6.46. R.B.2 classification. Weight 7st.8¾lb. Diagnosed pulmonary tuberculosis in A.T.S. July/1943. Left artificial pneumothorax in a Military Hospital, February 1943 - March 1944.

Re-induced September/1945 but abandoned at once as she developed a pyo-pneumothorax and aspirations were necessary.

X-ray of 19.6.46 showed a lateral pocket of fluid on left side. A limited, localised empyema was shown to be present.

4.7.46. First stage thoracoplasty with apicolysis - (Mr.G.Bryce).

Periscapular incision with division of the periscapular muscles. Second rib resected first (7½") with costal cartilage. First rib resected completely (4½"),

9L. E.M.B. No.20,434 (continued):-

third rib resected with costal cartilage (9½").

Apicolysis. The apex of the lung was mobilised a great deal concentrically, extrafascially, without difficulty. Intercostal bundles divided. Resection of segments of fourth rib (3") and fifth rib (3"); these two with their periosteum as opposed to the three upper ones which were resected subperiosteally. Sulphathiazole-penicillin powder applied to wound. Closure in layers with no drain.

8.7.46. Temperature fluctuating. Left infraclavicular fossa tender. Pain in the wound area. Three ounces of blood-stained serum were removed from the extrapleural space. This gradually turned into an extrapleural space infection and she remained pyrexial.

18.7.46. Small rubber tube inserted into the second intercostal space anteriorly to drain the extrapleural space. Patient apyrexial by 15.8.46 but pus (two ounces) was daily aspirated. She had lost weight and general condition

13.9.46. Sinograms showed a large extrapleural space to be present. Her sputum had been converted to tubercle bacilli minus since her thoracoplasty but ironically the space became infected with coagulase positive Staph. Aureus for which she had local and general penicillin. She developed tuberculous meningitis and died on 22.1.47. (Apicolysis dangerous in face of pocket of fluid).

X-rays show -

No.38, 19.6.46. The right lung field appears clear. Trachea, heart and mediastinum are displaced to the left. Marked pleural thickening over entire left lung with at least two vomicae visible below the left clavicle, and possibly a pocket of pleural fluid at left mid.zone.

No.39, 16.7.46. A grid photograph showing the appearances after a first stage left thoracoplasty. Note the uniform opacity over the left lower lobe.

No.40, 13.9.46. A left lateral sinogram in the vertical position to show the lower extent of the extrapleural space infection.

9L. E.M.B. No.20,434 (continued):-

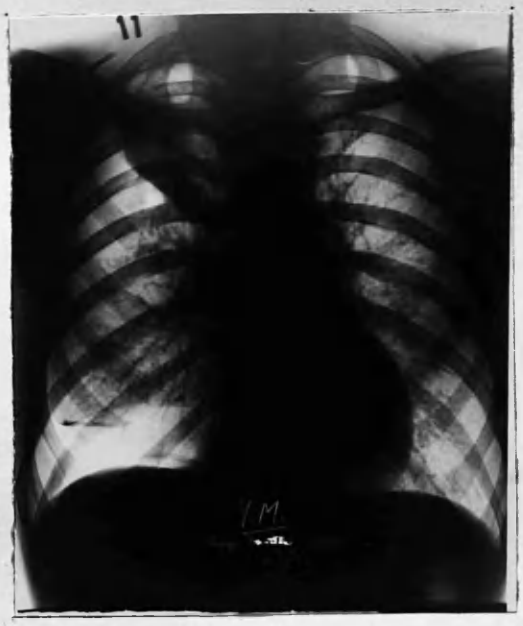
No.41. 13.9.46. A grid sinogram in the vertical P.A. position showing the lower limits of the empyema cavity.

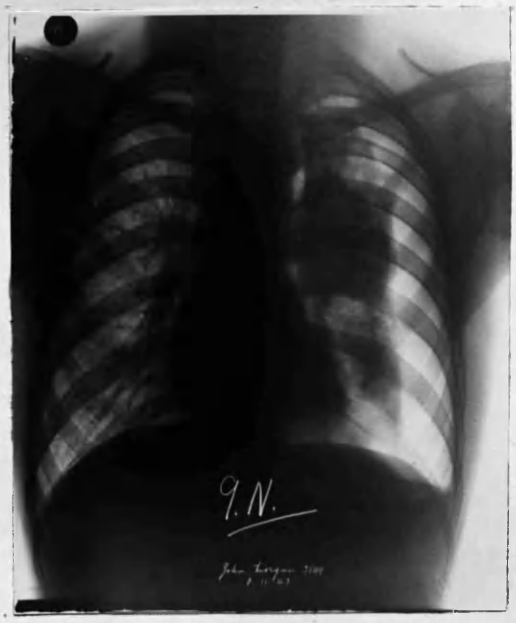
9M. G.F. No.18,051. A male, aged 39 years, R.B.3 classification and Blood Sedimentation Rate 29 mms./hour at empyema onset. No adhesion section done. No positive pressure refills given and no pyrexia at induction of right artificial pneumothorax. Non-purulent fluid formed three months and pus six months from induction. Treatment was by repeated aspirations. Progressive left carpal osteitis developed, cachexia and death, the empyema having lasted for five years.

A film taken after induction (21.7.41) shows that a right artificial pneumothorax is present. Right upper lobe is semi-collapsed and milky but shows no definite cavitation. It is widely adherent apico-mediastinally. The left lung field is clear. (A film before induction of this artificial pneumothorax shows an extensively mottled, pneumonic type of right upper lobe).

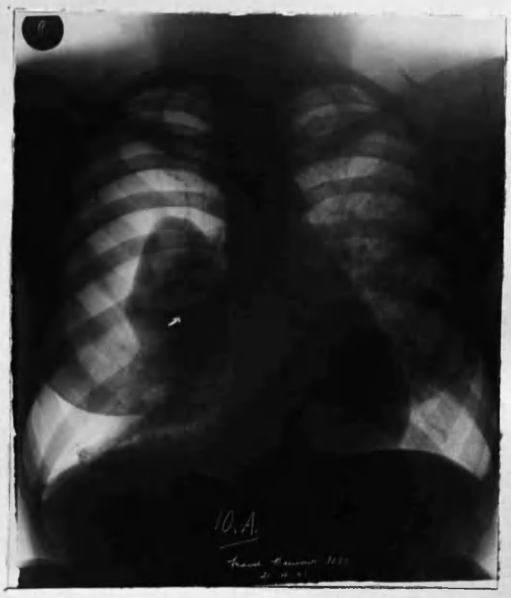
9N. J.M. No.19,157. A male, aged 18 years, R.B.2 classification and Blood Sedimentation Rate 29 mms./hour at onset of empyema. Pyrexia of 99.4°F. at induction of left artificial pneumothorax. No adhesion section done and no positive pressure refills given. Fluid occurred in one month and empyema followed in two months from induction. Treatment was by repeated aspirations and Azochloramide washouts at first. Then a stab drain was inserted for 1½ years and finally penicillin was used intrapleurally and systemically. This was a mixed infection empyema (Staph.Aureus and a non-haemolytic anaerobic streptococcus). Cachexia and albuminuria developed and finally a fatal brain abscess formed. The empyema lasted two years.

A film after induction (8.11.43) shows the right lung field is clear. A left artificial pneumothorax is present with a trace of fluid in left costo-phrenic angle. Left


11
I.M.


9.N.


9.P.
On admission


10.A.


10.B.
R. emphysema developed.


10.
Condition on admission

9N. J.M. No.19,157 (continued):-

upper lobe is opaque and has several peripheral cavities whose edges are like "ground-glass" and which are adherent to lateral chest wall and apex. A contra-selective artificial pneumothorax. (Pre-induction film showed extensive left upper lobe mottling and cavitation).

9.O. A.H.McM. No.19,639. A male, aged 24 years, R.B.2 classification and Blood Sedimentation Rate 33 mms./hour at empyema onset. No pyrexia at induction of left artificial pneumothorax and no positive pressure refills at any time. Empyema ensued 5½ months from induction and 1½ months from adhesion section (eight days after adhesion section there was a small spontaneous pneumothorax detected clinically). There were diphtheroid bacilli as well as tubercle bacilli in the pus.

Treatment was by repeated aspirations, Azochloramide washouts and finally penicillin. Death ensued in four months. No films are available.

9P. C.T. No.20,057. A male, aged 24 years, and R.B.2 classification at empyema onset. No Blood Sedimentation Rate records. This patient was admitted, extremely ill, with empyema. Left artificial pneumothorax was induced and adhesion section done in another hospital, and empyema ensued in one month and two weeks respectively from those procedures. Treatment was by repeated aspirations and antiseptic washouts and finally a monaldi catheter drain. Anterior chest wall sinuses formed. Death ensued, the empyema having lasted for 2½ years.

Post-mortem examination showed amyloid disease and rib necrosis round the sinuses.

A film, taken on admission, shows some scanty, hard mottling in the right mid. zone. A left hydro-pneumothorax is present, and while a small part of the left upper lobe is visible and aerated, there is no clue to the type of artificial pneumothorax which existed or the extent of parenchymal disease.

10A. M.B. No.18,730. A female, aged 32 years, R.B.3 (larynx) classification and Blood Sedimentation Rate 28 mms./hour at onset of empyema. No pyrexia at right artificial pneumothorax induction and no positive pressure refills at any time. Empyema ensued eighteen months after induction and eleven months after adhesion section. Unexpandable lung ensued, toxaemia, and death after 3 years 2 months of empyema.

Treatment was by frequent aspirations and a right phrenic nerve evulsion.

A film shows a right artificial pneumothorax is present with the merest trace of fluid in the costo-phrenic angle. Right upper lobe is collapsed and has a large cavity with milky, "ground-glass" like walls in its centre. There are several apical adhesions visible. The bronchus draining the cavity is seen to be swung downwards and kinked below the cavity (arrow) on the original film. This is at a point outwith bronchoscopic vision. There is a small vomica in the right lower lobe. The left mid.zone shows mottling and early cavitation (this increased later).

(A grid film of 14.7.46 shows the right upper lobe to be in a similar collapsed condition. The right lower lobe has expanded to chest wall and there is a dense fluid gathering opposite the lower lobe of scapula and up over the apex).

10B. L.B. A female, aged 18 years, R.B.3 classification, and Blood Sedimentation Rate 19 mms./hour at empyema onset. Pyrexia of 99°F. at induction of right artificial pneumothorax. No adhesion section or positive pressure refills. A cavity ballooned and ruptured and empyema ensued one month from induction. Unexpandable lung followed, cachexia and death in 2½ years finally. Treatment was by frequent aspirations and saline washouts and penicillin finally.

10B. L.B. (continued):-

An X-ray shows (11.10.44) bilateral artificial pneumothoraces are present. There is a trace of fluid in the right costo-phrenic angle. A large right apical vomica is present. This looks tense and its walls are beginning to assume a "ground-glass" look. The draining bronchus is visible and of uneven calibre. There is a small vomica and some mottling around, at apex of right lower lobe. Trachea and heart are over to left.
Left upper lobe appears opaque and collapsed.

10C. T.H.B. A male, aged 48 years, and R.B.2 classification at empyema onset. No Blood Sedimentation Rate records. This man had a left artificial pneumothorax and adhesion section in another institution. There was fluid after induction and he failed to attend for refills after fourteen months from his adhesion section. Empyema formed two years from induction and fourteen months from adhesion section. He was admitted here with empyema necessitatis (secondary organisms present).
A stab drain was introduced and continued for a year with saline washouts and penicillin. He was not in a fit condition for thoracoplasty. He died from toxaemia in three years eleven months from empyema onset.
An X-ray on admission (5.3.45) shows that the right lung field appears clear. A left hydro-pneumothorax is present with an opaque, collapsed left lung. A cavity (arrow) whose walls bulge out from the rest of the lung surface is seen in the left mid.zone. This has been a contra-selective left artificial pneumothorax and the apex is adherent to chest wall.

10D. I.E.W. No.19,302. A female, aged 27 years, R.B.2 classification and Blood Sedimentation Rate 32 mms./hour at onset of empyema. No pyrexia at right artificial pneumothorax induction and no positive pressure refills.
Empyema ensued five months from induction and six weeks from <u>partial</u> adhesion section. Treatment was by vigorous

10D. I.E.W. No.19,302 (continued):-

aspirations, and the instillation of blood and penicillin. The lung slowly expanded up to a point and then stuck with a narrow pleural space left. A left apical vomica precluded thoracoplasty. The patient returned home but a follow-up reveals that final obliteration of right pleural space occurred, the empyema having lasted for eight months.

An X-ray after induction shows that a contraselective right artificial pneumothorax is present. There is fine mottling in right lower lobe. Right upper lobe is semi-opaque and has one fairly large vomica and honeycombing above it. There are numerous apico-mediastinal adhesions present. The left upper and mid.zone show fine mottling with early cavitation first left interspace.

10E. E.F. No.19,084. A female, aged 32 years, R.B.3 classification, and Blood Sedimentation Rate 19 mms./hour at empyema onset. Pyrexia of 101°F. at induction of left artificial pneumothorax. Empyema followed fifteen months from induction, one month from partial adhesiotomy which left apico-mediastinal adhesions, and two weeks from a positive pressure refill of +2 +5 cms.water. Treatment was by left phrenic nerve evulsion and repeated aspirations at first. The lung almost re-expanded and the patient returned home with some fluid. A follow-up on 3.4.48 reveals that "there is probably no empyema left. She is in good general condition and has no sinuses or toxaemia." The empyema lasted three years. In view of the above report from her Tuberculosis Officer she will be classed as a cure.

An X-ray (1.9.43) taken on induction shows that the right lung and hilar markings are heavier than normal. There is mottling and fine honeycomb cavitation in the first right interspace. A shallow left artificial pneumothorax is present with apico-mediastinal adhesions. Left apex has extensive mottling extending from the hilum and a visible cavity with thin, opaque walls.

10E. E.F. No.19,084 (continued):-

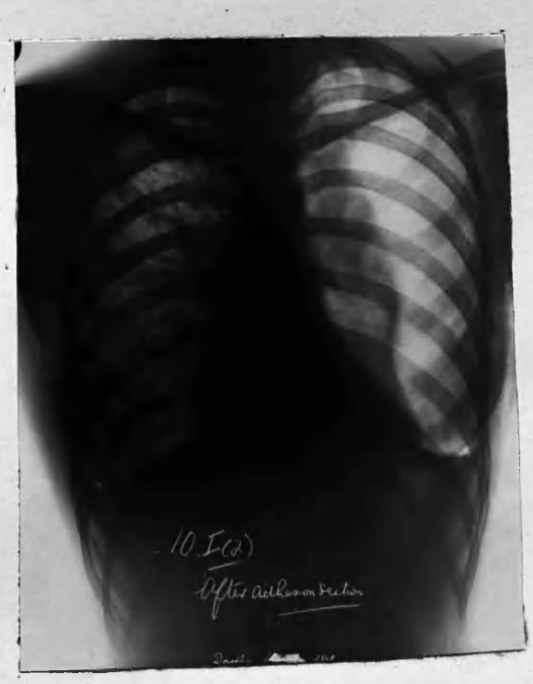
There is a tension type of cavity in left lower lobe.

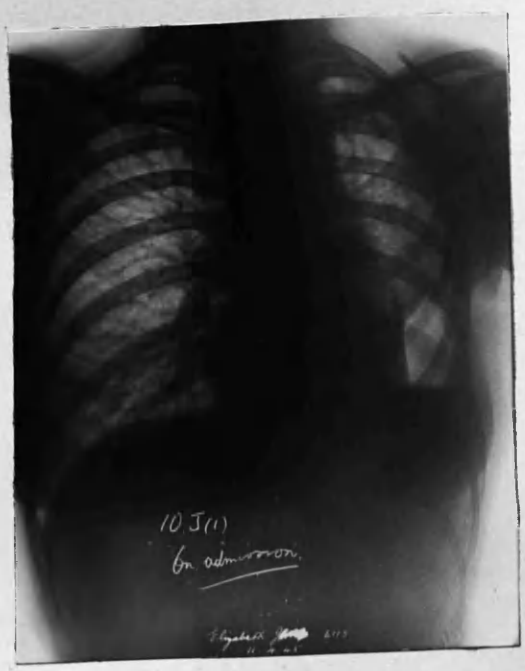
10F. W.D. 19,338. A male, aged 27 years, and R.B.2 classification at empyema onset. No Blood Sedimentation Rate records. There was no artificial pneumothorax in this case. Empyema is stated to have followed a left tuberculous broncho-pneumonia abroad. The patient was admitted with empyema having had air replacements for five months. Then he had a stab drain and Azochloramide washouts for sixteen months. A sinus developed round the tube in the left chest wall. He took his discharge against advice but died in one month from toxaemia, the empyema having lasted for two years six months.

A film, after air replacements, taken on admission shows no indication of whether or not a spontaneous pneumothorax was the cause of the left empyema. Right lung field is clear. A left hydro-pneumothorax is present with gross thickening of parietal and visceral pleura. No definite cavitation is seen in the underlying left lung.

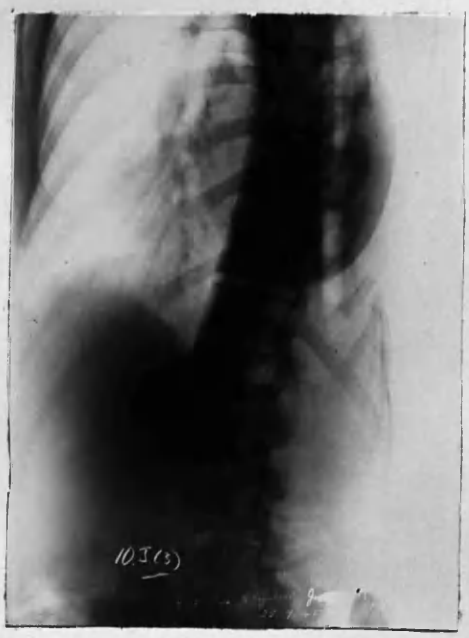
10G. J.D. No.20,002. A male, aged 38 years, R.B.2 classification, and Blood Sedimentation Rate 26 mms./hour at empyema onset. No pyrexia at induction of right artificial pneumothorax. No adhesion section or positive pressure refills. Empyema developed two months from induction. Repeated aspirations and Azochloramide washouts caused the lung to almost fully expand but a residual empyema remained. He was discharged before thoracoplasty but failed to return for operation. A follow-up reveals that on 3.4.48 he has no sinuses but poor general condition and his empyema is still present. To 3.4.48 it had lasted 2½ years and the outlook is poor. Films show -

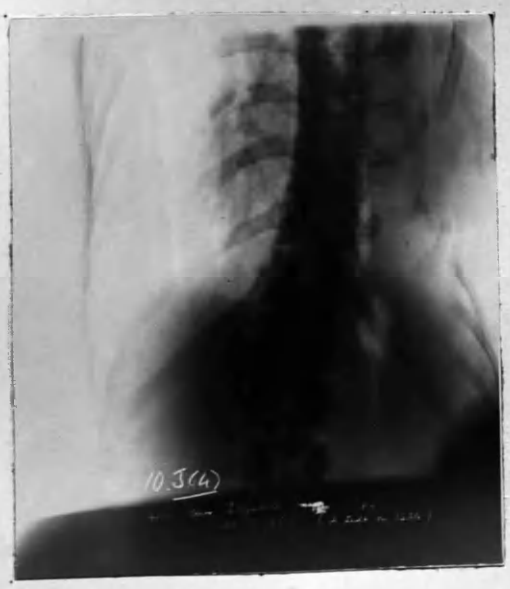
(A film of 11.6.45 before right artificial pneumothorax shows extensive soft mottling in the right upper and mid.zones with several early cavities visible. Left lung field is clear).

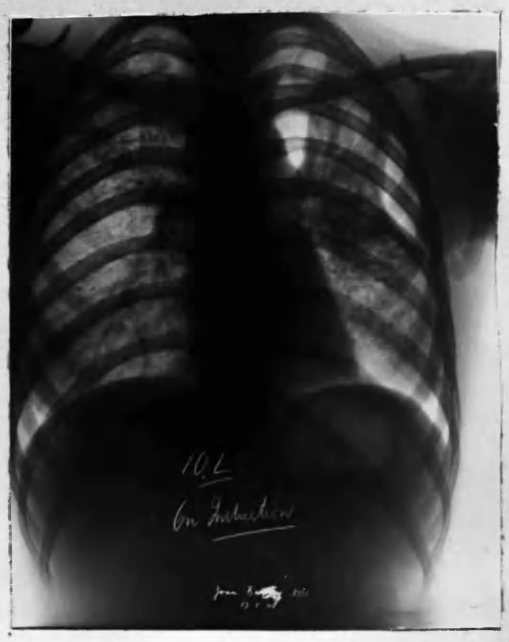

10.I(2)
After Aethamon Section


10.J(1)
On admission.


10.J.(2).


10.J(3)


10.J(4)


10.L
On Admission

10G. J.D. No.20,002 (continued):-

17.9.45. This film shows a contraselective right artificial pneumothorax present with numerous apico-mediastinal adhesions. Right lower lobe is adherent to diaphragm. A large right apical vomica is present and its draining bronchus (arrow) is tortuous and obviously kinked at one point. The surrounding walls are like "ground-glass" in appearance. Left lung field remains clear.

10H. J.W. No.20,112. A female, aged 18 years, and R.B.2 classification at empyema onset. No Blood Sedimentation Rate records. Empyema followed six months from left artificial pneumothorax induction. No adhesion section and no positive pressure refills. The patient was admitted with empyema and a grid film then shows left lung fields and the right lung markings are not visible. A left hydro-pneumothorax is present with gross parietal and visceral pleural thickening. A large left upper lobe vomica is visible. This has evidently been a contra-selective left artificial pneumothorax.

Subsequent films show cavitated disease in the right upper lobe.

Treatment was by repeated aspirations at first but chest wall sinuses formed. A stab drain was inserted and Azochloramide washouts given for four months. Toxaemia was marked, amyloidosis developed and she died one month after her discharge on compassionate grounds. The duration of empyema was ten months.

10I. D.S. No.19,625. A female, aged 22 years, R.B.2 classification, and Blood Sedimentation Rate 16 mms./hour at onset of empyema. No pyrexia at induction of left artificial pneumothorax. Empyema developed eight months from induction and two months from a partial adhesion section. Vigorous aspiration from the start, coupled with cannula suction of the debris and penicillin instillations, caused the lung to expand from below upwards. There was complete recovery and on 3.4.48 she is in good general

10I. D.S. No.19,625 (continued):-

condition and quiescent. (In this case penicillin made the pus thicker - necessitating cannula suction). Films show -

10I(1) 25.10.44. Right lung field appears clear apart from a small calcified nodule in the first right interspace. The right costo-phrenic angle is obliterated. A left artificial pneumothorax is present with several apical adhesions visible. Left upper lobe has a cavity whose walls are semi-opaque and have much soft mottling around.

10I(2) 28.2.45. Right lung field is unchanged. There has been recent adhesion section on the left side (surgical emphysema is visible). Left lung is more collapsed but still adherent apico-mediastinally. Entire left upper lobe is like "ground-glass" and still cavitated. A fluid level is visible in the left pleural space. (Empyema followed and the left lung eventually re-expanded completely).

10J. E.J. No.19,939. A female, aged 31 years, R.B.2 classification at empyema onset. No Blood Sedimentation Rate records available. At another institution a left artificial pneumothorax was induced in the absence of pyrexia. Empyema followed fourteen months from induction and two weeks from adhesion section. No positive pressure refills at any time. She was admitted with empyema for surgical treatment. First in Hospital 10.4.45 - 13.8.45. Admitted with left empyema for operative treatment. History of left artificial pneumothorax from August/1943 to October/1944. Then she had adhesion section and empyema in two weeks. R.B.2 case. The fluid was purulent and contained numerous tubercle bacilli.

10J. E.J. No.19,939 (continued):-

20.4.45. First stage thoracoplasty (Mr.G.Bryce).

Vital capacity 1,500.

Aspiration of 50 ccs. purulent fluid prior to operation. Division of muscles by a periscapular incision. Resection of ribs second (7"), third (8"), first (2¼"), fourth (1½") and fifth (2"). No apicolysis was attempted. The periosteal beds were treated with 20% tannic acid. Sulpho-penicillin powder applied to the wound which was closed in layers with no drain. There was repeated aspiration of the pleural fluid between the stages.

18.5.45. Second stage thoracoplasty (Mr.G.Bryce).

Vital capacity 1,100.

Old periscapular incision enlarged downwards. Exposure obtained through a gap between the trapezius and latissimus. Resection of 5½" of the already divided fourth rib and fifth rib (4½"). Then resection of ribs sixth (5"), seventh (4½") and eighth (3½") from the transverse processes forwards. 20% tannic acid to the rib beds and sulpho-penicillin powder to the wound which was closed in layers with a drain for 48 hours.

15.6.45. Third stage left thoracoplasty (Mr.G.Bryce).

Vital Capacity 900.

Low scapular incision prolonged downwards towards the spine. Resection of segments of the formerly divided following ribs:-

fifth (2¾"), sixth (3"), seventh (3"), eighth (3¾"). Then resection of the ninth rib (7"), tenth (5¾") and eleventh (3¾") from transverse processes forwards. Wound closed in layers with no drain after sulpho-penicillin powder.

29.7.45. Sodium Iodide sinogram showed a small residual empyema cavity with a fluid level in the lower part. No further operation was contemplated.

Discharged to Crossley Sanatorium 13.8.45. Sputum concentration test tubercle bacilli minus.

10J. E.J. No.19,939 (continued):-

17.6.46 - 28.6.46. Re-admitted to Baguley Sanatorium for Bronchoscopy, and bronchiectasis of left lower lobe diagnosed. Sputum remained tubercle bacilli minus. X-rays show -

10J(1) 11.4.45. On admission the right lung field is clear. Upper mediastinum and heart are displaced to left. A basal left artificial pneumothorax is present with fluid level. There is evidence of visceral and parietal pleural thickening. Left upper and mid.zones are close to chest wall (but not adherent as subsequent films show). Some cavitation of left upper lobe is evident.

10J(2) 6.7.45 is a left lateral, vertical sinogram using Sodium Iodide. It shows part of the extent of the post-operative cavity.

10J(3) and (4). 25.7.45. P.A. views with the right and left sides of the patient on the table, respectively, show how thickened parietal pleura has prevented chest wall collapsing and closing the empyema cavity properly.

By 3.4.48 a follow-up reveals that her small residual space has apparently healed. She is well with no sinuses, i.e., recovery from empyema.

10K. C.M.W. No.20,863. A female, R.B.2 classification, aged 19 years, at onset of empyema. No Blood Sedimentation Rate records at onset. Briefly this patient had left lower lobe lobectomy for bronchiectasis which was later proved to be tuberculous. Broncho-pleural fistula persisted. Pneumonectomy was done resulting in fistula, empyema, and declining general condition on 3.4.48. Had left lower lobectomy in Baguley Emergency Hospital in 1943 for bronchiectasis. Empyema followed and tubercle bacilli were recovered from the pus on one occasion only. She was discharged, with the empyema apparently cleared, to Blencathra Sanatorium. On 18.9.46 she returned with pus in the left pleural cavity

 C.M.W. No.20,863 (continued):-

and a tube was inserted on 19.9.46, and she went back to Blencathra on 25.10.46. The tube came out, pus pocketed and she became toxic and was returned to Baguley Sanatorium on 5.8.47 for further treatment. On review of the case it was decided that she was probably non-tuberculous and her sole tubercle bacilli positive finding three years previously was considered a probable error. She had no sputum. A small anterior sinus was present in the left chest wall at the level of the second rib anteriorly. On 8.9.47 a retrograde bronchogram was again obtained.

12.9.47. Pneumonectomy (Mr. G. Bryce).

General, intra-tracheal anaesthesia.

Old lobectomy incision re-opened with division of the trapezius, rhomboids and latissimus dorsi. The upper ribs were exposed and a long segment of the third rib resected and as much of rib 4 as possible. Then a segment of the fifth rib was resected. Each of those ribs was resected down to the transverse processes. The pleural cavity was entered through the periosteal bed of the fourth rib. The blowing of the pulmonary fistula seemed to come from the anterior aspect of thelung. The left upper lobe was blue and incompletely expanded and was firmly adherent to the chest wall. When the empyema cavity was examined it had at its postero-inferior angle some pus and blood contents. At this stage it was decided to perform a pneumonectomy. The left upper lobe was resected in spite of difficulty in identifying the hilar structures. Pulmonary artery, bronchus and veins were ligatured separately; the bronchial stump was secured with nylon mattress sutures and covered at the end with a pleural flap. A small portion of the left upper lobe, firmly adherent to the scapular region (in the vicinity of anterior sinus) had to be resected piecemeal and it was feared that some small portions

10K. C.M.W. No.20,863 (continued):-

of lung tissue were left adhering to the parietal pleura. All the mediastinal structures were clearly seen. At the end of the operation the thoracic gap was closed by approximating the ribs as much as possible. She had 80,000 units of intramuscular penicillin before the operation and the wound and empyema cavity were dusted with sulpho-penicillin pulv. A drainage tube was left in situ through the old, posterior sinus. Two pints of blood and two pints of saline were transfused during the operation.

She had a slow convalescence and was able to return to Blencathra Sanatorium on 21.11.47. The sinus was discharging profusely and her general condition would not allow her to undergo further operational treatment.

On 5.9.47 a laryngeal swab culture was positive for tubercle bacilli. This swab had been taken just before her operation but the result did not come back till several weeks later.

A follow-up of this case showed that her general condition was slowly deteriorating by June 1948.

10L. J.B. No.19,452. A female, aged 19 years, R.B.2 classification, and Blood Sedimentation Rate 10 mms./hour at empyema onset. Pyrexia of 100°F. at left artificial pneumothorax induction. Empyema onset fifteen months from induction and one week after a solitary positive pressure refill of 0 + 10 cms. water. No adhesiotomy was done.

Treatment was by repeated aspirations and later penicillin instillations which made the pus mucoid in character. This was a pure tuberculous empyema throughout. The lung apparently re-expanded after an empyema duration of two years nine months.

A follow-up on 3.4.48 shows no sinuses and no empyema but poor general condition and deterioration from her parenchymal disease. We must consider this a cure of the empyema.

10L. J.B. No.19,452 (continued):-

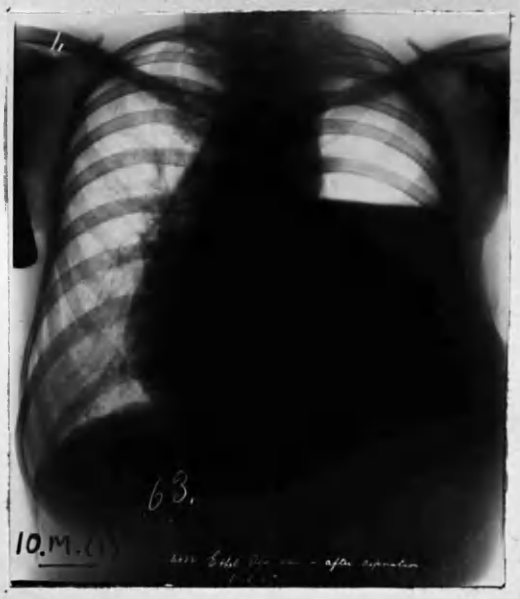
An X-ray shows (17.5.44). right apex and mid.zones have a profuse, soft mottling with probable early cavitation in first interspace. A contra-selective left artificial pneumothorax is present with no fluid. Left upper lobe is honeycombed and milky in appearance and widely adherent to upper and outer chest wall. Fine mottling is evident in left lower lobe.

10M. E.O. No.20,574. A female, aged 37 years, and R.B.3 classification at onset of empyema. No Blood Sedimentation Rate records. Pyrexial when left artificial pneumothorax was induced and empyema came on in two weeks. No adhesion section and no positive pressure refills. She was admitted with empyema to have an intercostal catheter inserted. This was done and Azochloramide washouts given after five months of regular aspirations. She returned to the hospital from which she came. A follow-up on 3.4.48 reveals that there has been a flare-up and extensive spread of disease in the right mid.zone. Collapsed left lung, empyema and sinus in left chest wall remain and she is deteriorating rapidly, the empyema having to date lasted two years eight months.

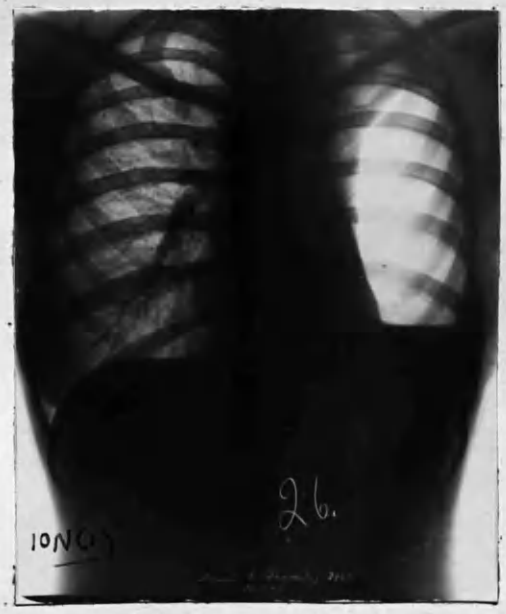
X-rays show - No.63, 1.1.47. On admission, lung markings prominent on right side, probably due to the gross shift of heart and mediastinum to the right. Large hydro-pneumothorax on left side. (This X-ray was taken after 1½ pints of fluid had already been removed). Left upper lobe opaque.

No.64. 28.4.47. Scanty mottling right apex, more prominent than on previous film. Heart and mediastinum now central in position and left lung has semi-expanded. Note the thickened visceral and parietal pleura on the left. Intercostal drain in left chest wall which is thickened around the tube. The appearances suggest a large vomica in the left upper lobe.

10N. S.D. No.20,443. A female, aged 24 years, R.B.2 classification and Blood Sedimentation Rate 30 mms./hour at


63.
10.M.(1)


64.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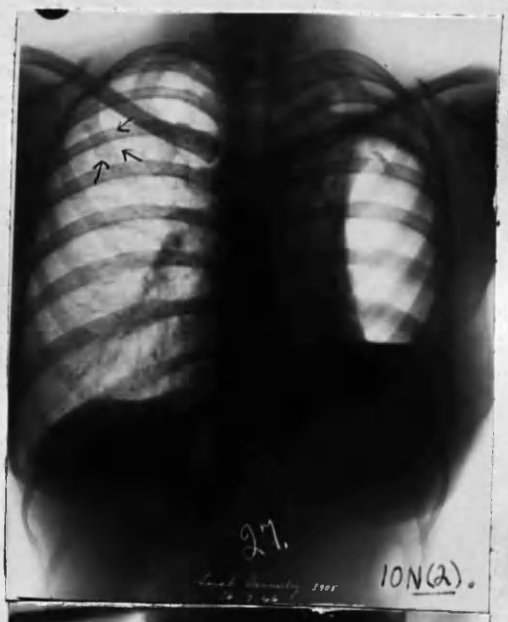
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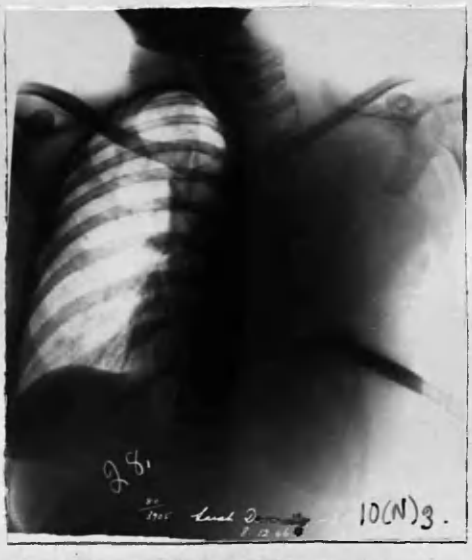
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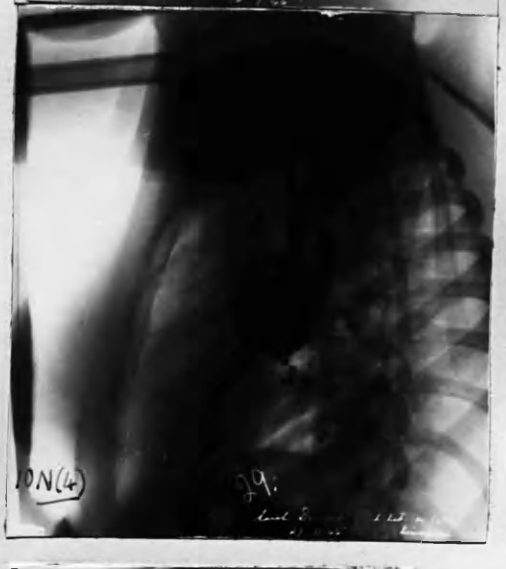
10(N)3.


10N(4)
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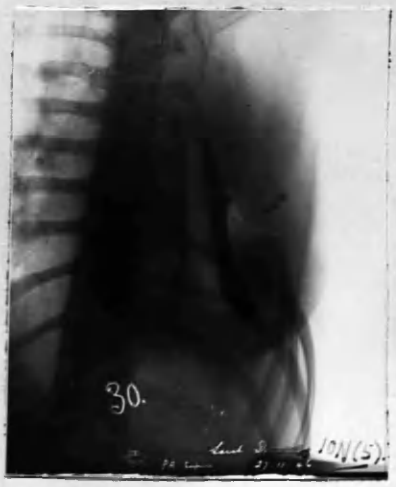

30.
10N(5).


31.
10N(6).

10N. S.D. No.20,443 (continued):-

onset of empyema. Admitted with an empyema which came on six years after induction of a left artificial pneumothorax.

In hospital 28.8.44 - 27.3.46 and transferred to Delamere Sanatorium. Returned to Baguley Sanatorium 18.7.46. Weight 8st. 12¾ lb. R.B.2.

A left artificial pneumothorax had been induced in 1940 in a sanatorium in North Wales. On her first admission here she had fluid at the left base and a left artificial pneumothorax present. Fluid was clear and yellow. No organisms were found and numerous lymphocytes were present. Occasional aspirations and refills were conducted until 9.3.45 when no more air was given. After 27.3.46 she was transferred to another sanatorium for a change in environment (weight 7st. 4½ lb.) and to prepare her for operative treatment. Re-admitted on 18.7.46 she weighed 8st. 12¾ lb. Blood Sedimentation Rate 21 mms./hour. 23.7.46 effusion was amber coloured. 30.7.46 intercostal stab drain prior to left thoracoplasty.

2.8.46. First stage left thoracoplasty (Mr.G.Bryce).

Low periscapular incision with division of only a few fibres of rhomboids, trapezius and latissimus dorsi. Although exposure was not satisfactory, the first upper ribs were resected without further enlarging the wound, thus sparing the periscapular muscles. Sub-periosteal resection of the following ribs, third rib (7"), second rib (7¾"), first rib (4"). The first and second ribs were resected with portions of their costal cartilages. The third rib was only resected to near its costal cartilage. No transverse processes were divided. The periosteal beds of the first and second ribs were divided and tied. The periosteal bed of the first rib was also divided in front. Mobilisation of the apex was attempted but abandoned in view of

 S.D. No.20,443 (continued):-

extreme thickness and fibrosis of the underlying pleura and lung tissue, practically uncollapsible and very firmly adherent to the paravertebral gutter and mediastinum. Sulphathiazole-penicillin powder sprinkled over the operative field. Wound closed in layers. No drain.

X-ray after this stage (4 days later) showed the left apical vomica still present and the empyematous cavity slightly smaller.

23.8.46. Second stage thoracoplasty.

Old periscapular incision re-opened. Care was taken not to enter the extrapleural space left after the last operation. The fourth rib was resected from the transverse processes towards the anterior axillary line. This was followed by resection of segments of the fifth and sixth ribs, long enough to allow bedding of the scapula. The transverse processes of those ribs were also resected. Sulpha-penicillin powder. No drain and closure in layers.

20.9.46. Third stage thoracoplasty.

Old periscapular incision re-opened. Resection of segments of the following ribs, seventh rib ($3\frac{1}{2}$") with transverse process, eighth (3") with transverse process, anterior segments of the sixth rib (2"), fifth rib (4"), and fourth rib ($5\frac{1}{2}$"). The pleural cavity was incised along the periosteal bed of the seventh rib and the empyematous cavity entered. This extended upwards towards the axilla, anteriorly to the cardiac area and posteriorly to the vertebral gutter; in this stage only the upper portion of this cavity was tackled, leaving the lower part for the next stage. The operation consisted in cutting the lateral wall along the axillary line up to its highest level until two hinge-like pleural flaps were obtained. A large gauze pack was left between the sunken parietal pleura and the skin, after sprinkling with sulphathiazole-penicillin

 S.D. No.20,443 (continued):-

powder. (The pack was to be removed in seven days). The anterior stab drainage was kept and connected to a suction machine. (Pleurograms were taken before stage 4).

6.12.46. Fourth stage thoracoplasty (Mr.G.Bryce).

Periscapular incision opened. No traces of muscles seen. Resection of 3" of paravertebral end of the ninth rib. Empyematous cavity re-opened. It was found to have three prolongations - one stretching anteriorly and upwards, one anterior and forwards behind the left breast and one posterior, towards the spine. The postero-paravertebral one was obliterated after the last operation. Now another prolongation had formed between the parietal pleura and the scapula. To obliterate this it was necessary to resect a great deal of the scapula, almost to the transverse spine, and even then it was found that the posterior aspect of this bone was overlapping the paravertebral aspect of the parietal pleura rather than falling downwards against the axillary region. This was corrected in part on sewing up, by stitching the periscapular flaps to the eighth intercostal muscle. A portion of the parietal pleura, together with bits of regenerated ribs, was resected (partial Schede's operation) leaving a thin flap of parietal pleura lying in the bottom of the cavity. (It was proposed at a fifth stage to resect the anterior segments of the ribs covering the anterior prolongation of the empyema and probably another partial Schede's operation. For the obliteration of the posterior pocket it would be necessary to resect the tenth rib). A rubber tube was inserted through the decostalised ninth space, in the scapular line, and the wound was closed with thread to the skin only. Albuminuria commenced after this stage and increased to 20 grammes daily. 4½ ounces of pus discharged daily from the tube.

On 2.1.47 oedema of the legs appeared and amyloid

 S.D. No.20,443 (continued):-

disease was diagnosed. She deteriorated rapidly and died on 13.2.47.

X-rays show -

No.26, 30.8.44. There is a fine, discreet mottling in the upper and mid.zones of the right lung. A left hydro-pneumothorax is present. The left lung is much collapsed but held out by adhesions to the chest wall below and above the clavicle. There is a large vomica in the left upper lobe and the lung around this is of an opaque, "ground-glass" appearance.

No.27. 24.7.46. The appearances of the right lung are stationary. There is possibly a small vomica in the first right interspace. The left lung has expanded slightly but has thickened parietal and visceral pleura. The left apical vomica persists, as does the fluid level in the pleural space. Trachea and heart have moved slightly to the left.

No.28, 8.12.46. This X-ray was taken in bed after the fourth stage of the left thoracoplasty. The right side has slightly more mottling in all zones. There is marked scoliosis with the spinal convexity to the left. An extensive left thoracoplasty is present with a large drainage tube in the empyema's residual cavity. Note the perpendicular slit of residual empyema cavity extending upwards above the head of the safety pin (which latter is in the dressings to hold the tube in position).

No.30, 27.11.46. Taken before the fourth stage of the left thoracoplasty this sinogram shows clearly the vertical extent of the empyema cavity which had to be closed.

Nos.29 and 31, 27.11.46. Those are sinograms in the "left lateral flat" and "left lateral vertical" positions to show the extent of the empyema residual space before the fourth stage thoracoplasty. The total duration of this empyema was seven months.

10.O.A.T. No.17,656. A male, aged 43 years, R.B.2 classification, and Blood Sedimentation Rate 26 mms./hour at onset of empyema.

First in hospital 9.1.36 - 24.8.36 when he took his discharge at his own request against advice. On admission then he had suffered from winter cough of many years' standing. Grade on admission R.B.2.

Re-admitted 15.4.40. R.B.2, weight 8st. 10 lb., Blood Sedimentation Rate 23 mms./hour.

9.6.40. Right artificial pneumothorax induced. Fluid appeared 9.7.40 and came and went. Refills continued.

2.8.40. Right phrenic crush.

7.4.41. Adhesiotomy.

Fluid increased and on 16.1.42 seven ounces of straw-coloured fluid were removed. This fluid became progressively thicker until it was frank pus on 25.9.42. The last refill given was on 30.1.42. Thereafter began a long series of regular aspirations and chest washouts with saline and later Azochloramide. The chest wall was indurated over the area where aspirations were done, at an early stage. The empyema became secondarily infected with coagulase positive Staph.Aureus.

24.8.43. De'Pezzer catheter inserted in conjunction with Eloesser flap operation of 3.8.43.

3.8.43. Eloesser's operation (flap drainage) right side (Mr.G.Bryce).

Premedication: Omnapon gr.1/3, Scopolamine gr.1/300, one hour before operation. Morphine gr.1/6, and Atropine gr.1/100 while on the table. Local infiltration (Procaine 1% and ½%).

"U" shaped incision below the scapula to axillary line. Flap of skin dissected and retracted upwards. Serratus magnus muscle divided. Seventh rib exposed and 2½" resected. Intercostal bundle divided and ligatured. Periosteal bed incised. Cavity explored and pus sucked away. Skin flap fixed inwards to parietal pleural edge.

10.O.A.T. No.17,656 (continued):-

Gauze pack inserted. Lower part of wound stitched and a small opening was left. Tuberculous caries episode: In May/1946 the fourth and fifth left vertebrae were found to be the seat of tuberculous caries, and he was placed in a spinal frame.

The empyema became a very narrow slit with a chronic discharge. Sputum remained tubercle bacilli positive to the end. On 12.12.47 he died of cerebral thrombosis, aged 49 years. Duration of empyema 5¼ years.

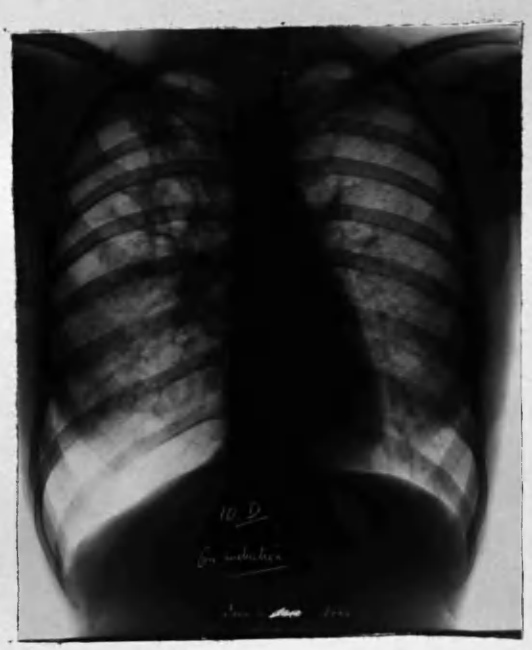
X-rays show -

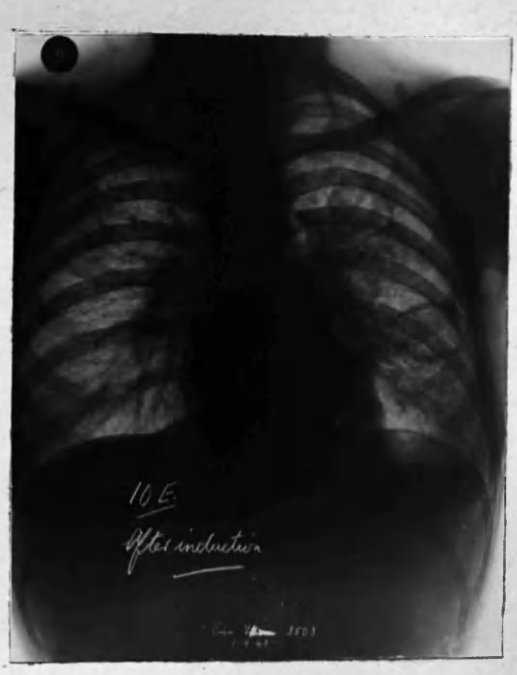
No.11, 15.4.40. Mottling all zones right lung. Upper half of right lung has several large cavities with fluid levels very near the lung periphery. Coarse mottling in all areas of the left lung but no definite cavitation.

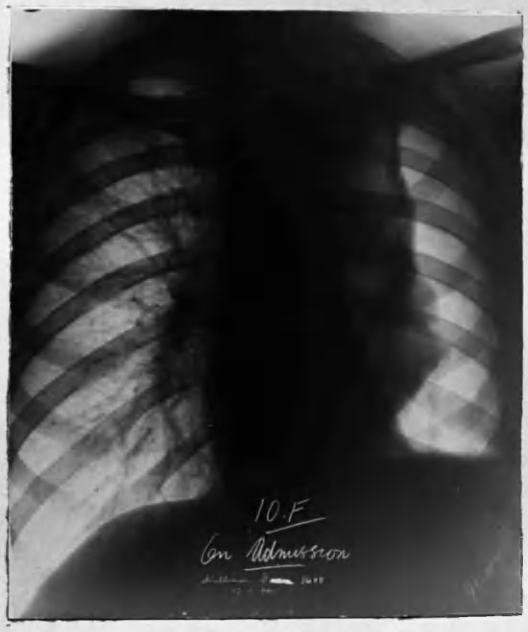
No.12, 22.7.40. Right artificial pneumothorax present with fluid level in costo-phrenic angle. Mottling visible in right lower lobe. Right upper lobe is the seat of one large vomica whose walls are opaque and like "ground-glass". Several right apico-mediastinal adhesions evident. Left side is unchanged.

No.13, 14.7.41. Right artificial pneumothorax present with expanded right lower lobe out to chest wall. Right hemi-diaphragm is elevated. Right upper lobe is still semi-collapsed and several cavities visible. Apico-mediastinal adhesions persist. Slight scoliosis developing. Not much change in left lung.

No.14, 18.3.42. Right lower lobe remains adherent to lower chest wall. Right hemi-diaphragm is less raised and somewhat flattened. Extreme collapse of right upper lobe which is opaque. Fairly large fluid collection evident. Left side is in status quo.


10.D
On induction


10.E
After induction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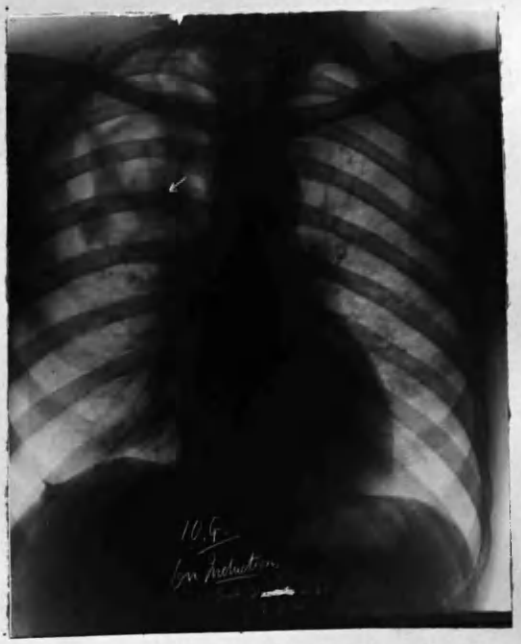
On Admission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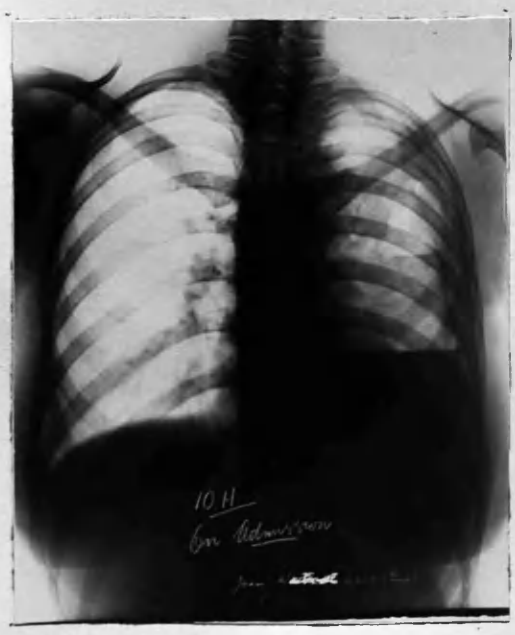
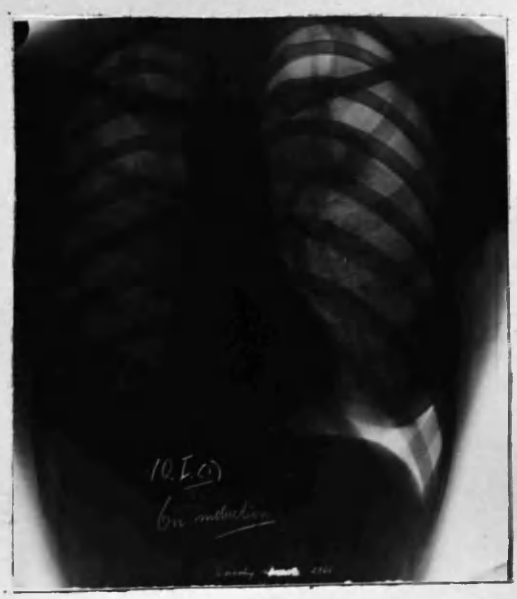

10.H
On Admission


10.I (?)
On induction


11.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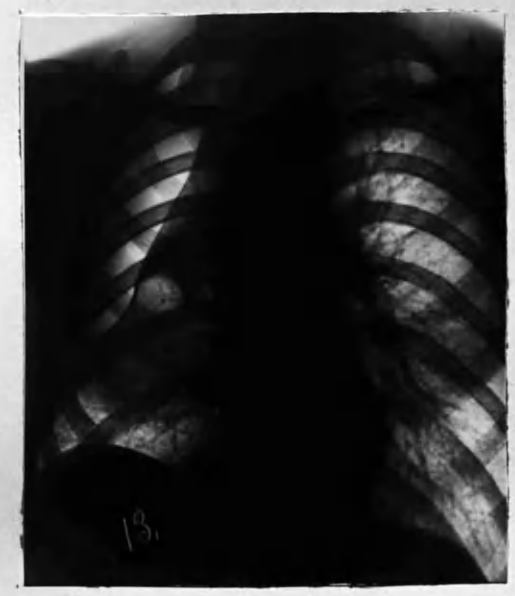
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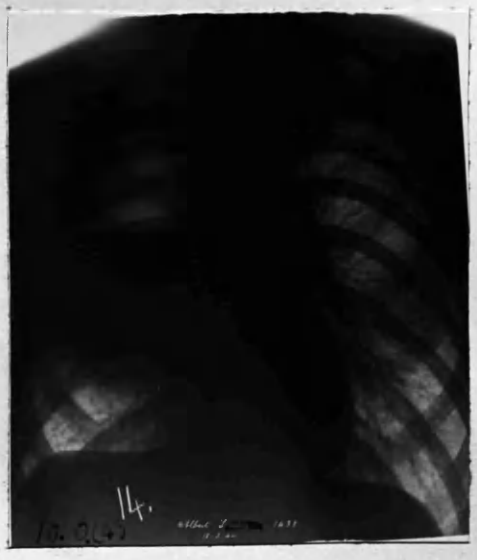
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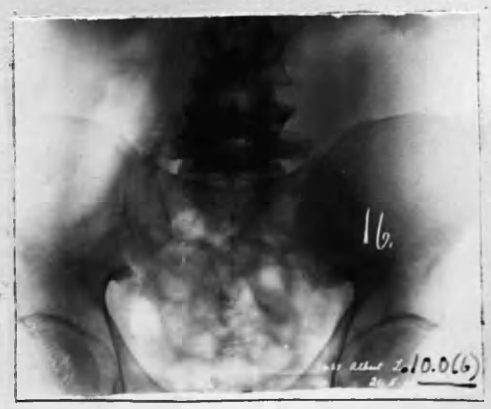

16.

10.O.A.T. No.17,656 (continued):-

No.15, 9.4.45. Right hydro-pneumothorax persists. The parietal and visceral pleurae are thickened. Cavities still visible in right upper lobe. Trachea over to right and scoliosis is more marked with tiling of ribs on the right side. The left side appearances are unchanged.

No.16, 21.5.46. Collapse of intervertebral disc and caries of adjacent bodies of third and fourth lumbar vertebrae.

10P. K.V. No.20,556. A female, aged 29 years, R.B.2 classification, and Blood Sedimentation Rate 15 mms./hour at onset of empyema. No pyrexia when left artificial pneumothorax was induced. Empyema ensued two years from induction and one year eight months from adhesion section. She had received refills to positive pressures for five months before empyema onset.

Treatment was by aspirations at first. When chest wall sinuses formed, bed rest alone and daily dressings were given. Cachexia progressed and death, the empyema having lasted seven months.

X-ray (No.17, 18.12.46) after empyema had existed some time shows scanty, hard, discreet mottling in right upper and mid.zones. More profuse and soft mottling in the right lower lobe. Honeycombing of extreme right apex. Scoliosis present and trachea over to left. Left lung is collapsed and airless. No definite cavities identified. Large pneumothorax space with a trace of fluid at the base on the left side. Greatly thickened parietal and visceral pleurae. (The safety-pin behind the heart is attached to dressings on the surface of the body).

This was a contraselective left artificial pneumothorax with apical adhesions.

10Q. E.W. No.19,641. A male, aged 18 years, and R.B.2 classification at empyema onset. No Blood Sedimentation

10Q. E.W. No.19,641 (continued):-

Rate records. Admitted with empyema which came on eight months from right artificial pneumothorax induction. No positive pressure refills or adhesion section. This empyema lasted three years one month before death ensued from albuminuria, cachexia, and finally tubercular meningitis.

Admitted 28.8.44. Weight 10st. 4¾ lb. with a right-sided empyema. Had previously been in another sanatorium from 12.11.43 to 8.4.44. Right artificial pneumothorax induced 19.11.43 and continued, his last refill being four weeks prior to admission to Baguley Sanatorium. Empyema onset was in early July/1944. Repeated aspirations undertaken and the lung appeared to be re-expanding. In April 1945 the fluid (previously tubercle bacilli plus only) showed a growth of B. Proteus and B. Pyocyaneus and Staph.Albus.

9.4.45. A right intercostal stab drain was inserted laterally. Saline washouts and penicillin therapy instituted.

27.11.45. For sinograms.

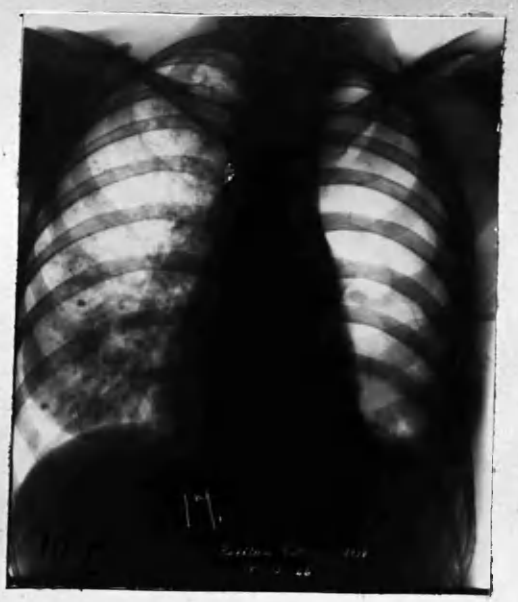
8.3.46. First stage thoracoplasty (Mr.G.Bryce).

Small incision below the scapula.

Subperiosteal resection of segments of ribs eleventh (3½"), tenth (5¼"), ninth (5¾") and eighth (7¼") with the transverse processes of all except the eleventh. Sulphonamide-penicillin powder applied to the wound.

26.3.46. Second stage thoracoplasty.

Low periscapular incision with only partial division of low fibres of trapezius and rhomboids. Resection of the seventh, sixth, fifth and fourth ribs as far forward as the anterior axillary line, near and above the draining tube, with their transverse processes. There was much fibrosis around this area due to underlying pleural thickening. Sulphon.-penicillin pulv. Closure in layers. Rubber drain for 24 hours.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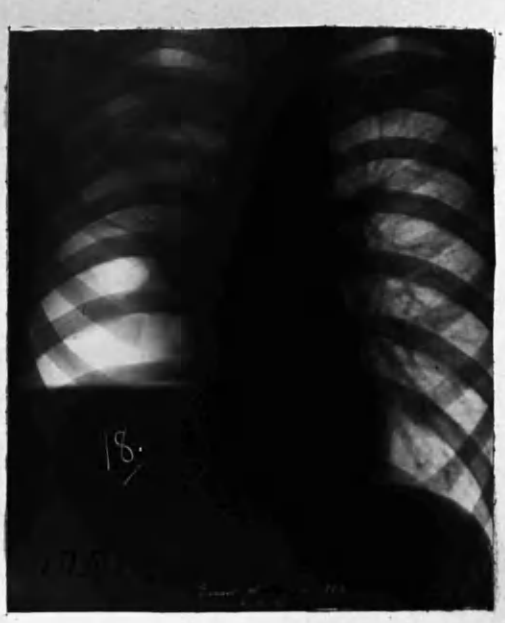
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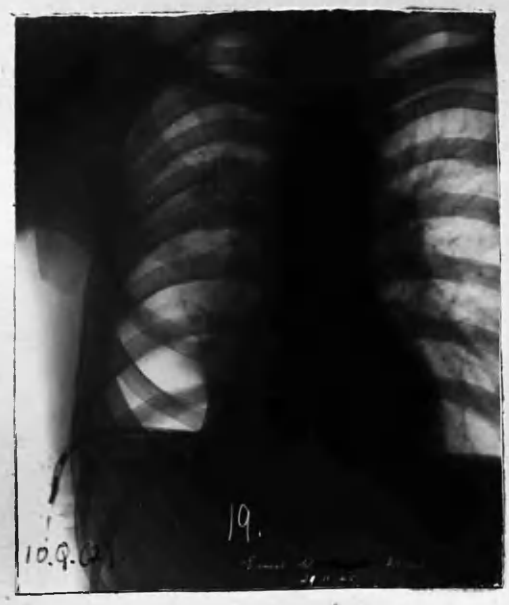
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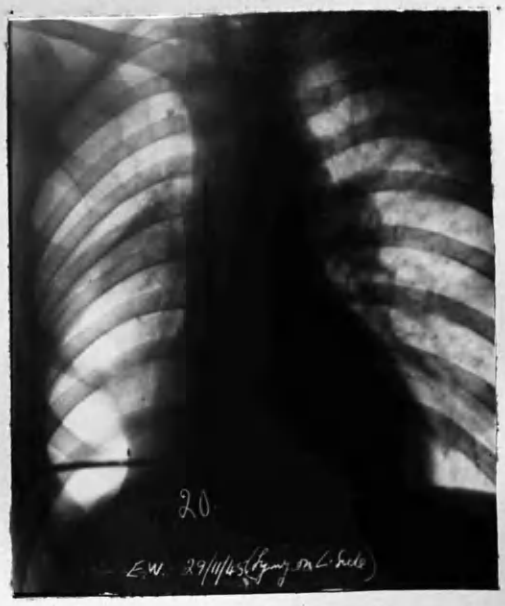
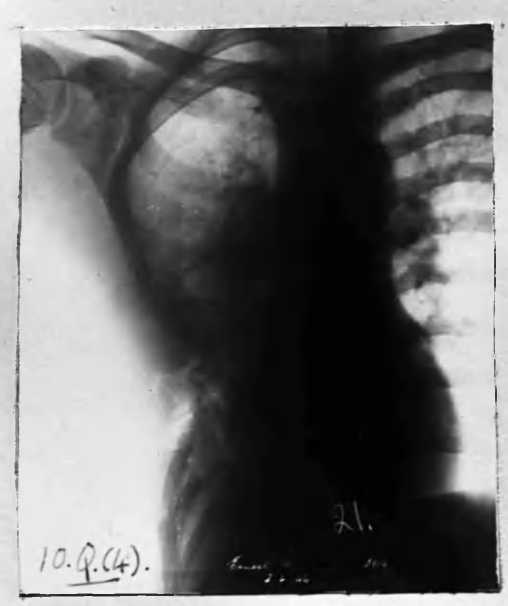

20
E.W. 29/11/43 Effusion in L. base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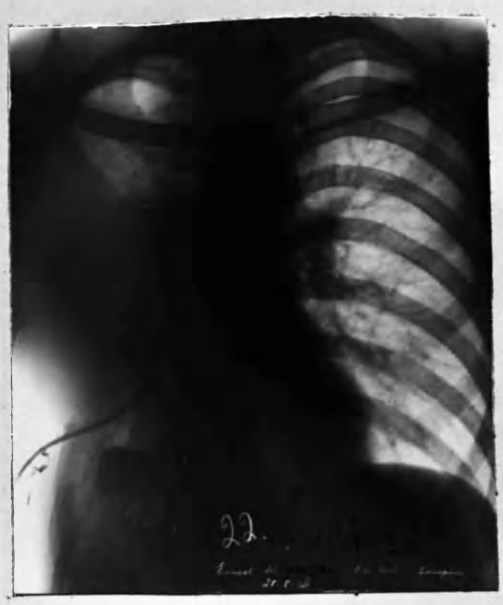
10.Q.(4).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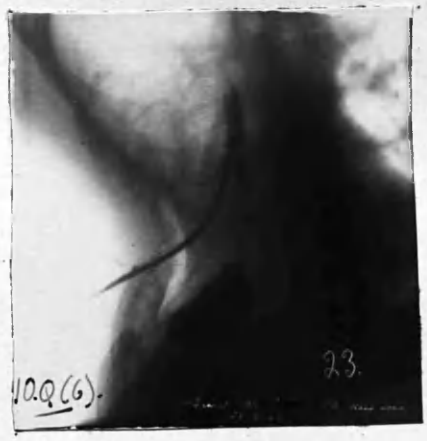
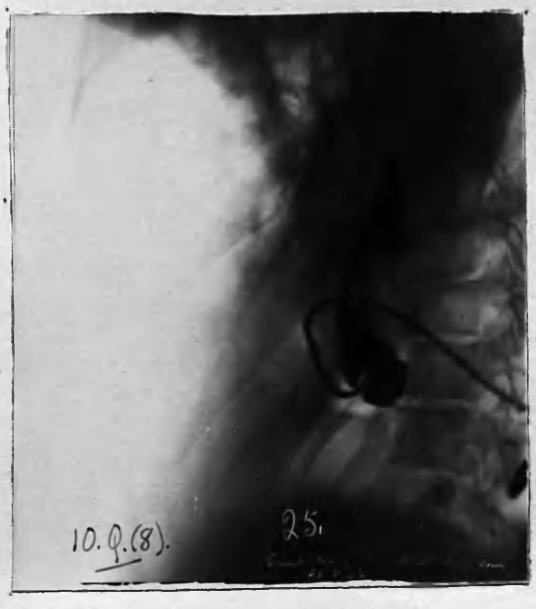

10.Q.(6).
23.


24
10.Q.(7).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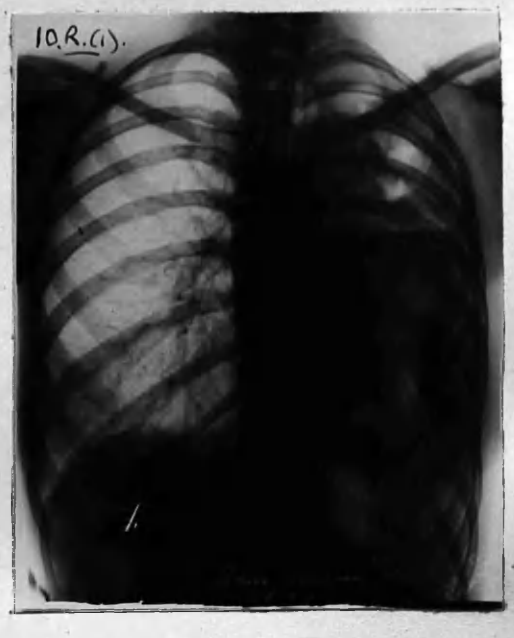
10.R.(1).
1.


10 R(2).
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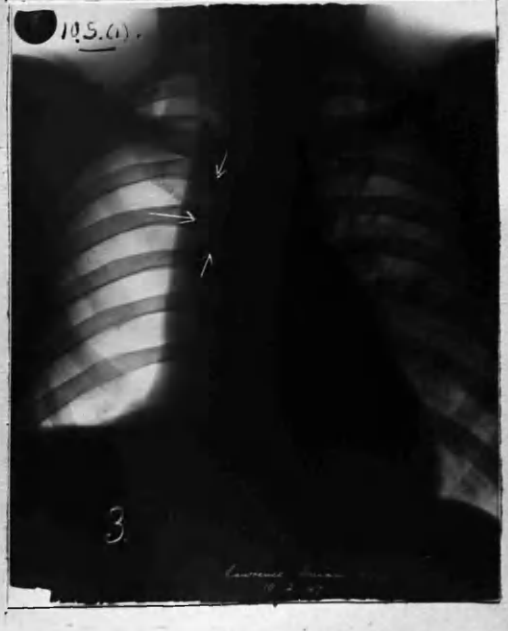

10.S.(1).
3.

10Q. E.W. No.19,641 (continued):-

26.4.46. Third stage thoracoplasty.

Vertical incision 2½" through existing tube sinus, anterior to latissimus dorsi, through the fibres of the serratus magnus. Two ribs were resected (regenerated ribs). Exploration showed a large residual paravertebral cavity extending higher than the finger could reach and, in addition, postero-inferior and antero-inferior pockets.

(a) Anterior pocket. Three ribs were resected over this, and the outer wall cut as a flap which was approximated to the visceral layer, with the intention of obliterating this pocket.

(b) The old paravertebral wound was enlarged forwards to the anterior-vertical line. Latissimus dorsi and serratus magnus were preserved but no plastic procedure was started here. The latissimus and serratus were marked by thread ligatures which encircled them, and the long ends buried and marked by a special skin stitch in the paravertebral wound. The empyema cavity was slightly packed with gauze and a long fine tube incorporated for the instillation of penicillin. The gauze (2 pieces) and tube were brought out of the anterior incision.

(c) At the next stage it was proposed to re-open the incision and attempt to preserve the latissimus and serratus muscles. Then the cavity would be widely opened at its inferior extremity and the extent of the paravertebral segment of the cavity determined. It was proposed to close the cavity by a Robert's operation or by a latissimus dorsi flap, bearing in mind that some further rib resection would probably be necessary.

31.5.46. Fourth stage thoracoplasty (Mr.G.Bryce).

Lower part of periscapular incision re-opened. Latissimus dorsi and serratus magnus

10Q. E.W. No.19,641 (continued):-

divided and used as muscle flap. The thread and catgut loops left during the last operation were of no practical use in identifying the above-mentioned muscles. The empyema cavity was re-opened, this time along the paravertebral pocket and transversely. No ribs were resected but regenerated ribs had to be freshly removed from the paravertebral flap. This flap, formed by parietal pleura, was raised by three incisions, a longitudinal, paravertebral one, and two small ones perpendicular to the spine. A Robert wedge was cut so the flap was hinged and allowed to fall loose, obliterating the paravertebral pocket. This flap was not stitched with catgut but was kept in position by the falling of the scapula. The lower and anterior parts of the empyema cavity were filled by the freeing of the parietal pleura around them but mainly by the muscular flap raised from the latissimus and serratus muscles, the pedicles of which received their blood supply from their scapular attachments. Gauze was applied to the operated area and covered by the skin, leaving a small rubber tube for irrigation and a rubber drain by a separate incision. The gauze was left in situ for a week and not reinserted. Irrigations were carried out by small amounts of Eusol.

20.7.46. Pleurograms showed two considerable residual pockets communicating with one another, (1) smaller paravertebral extending as high as the seventh rib (posterior end), the other larger (2) lower pocket in the lateral part of the chest.

Drainage was continued and it was thought that a further plastic procedure might be required later for the lower pocket. Albuminuria developed and the empyema sinus drained copious, evil-smelling pus. Eusol, saline and penicillin were all tried and painful daily wound dressings required to the end. A painful, lingering termination. 1.8.47. Death from tuberculous meningitis.

10Q. E.W. No.19,641 (continued):-

X-rays show -

No.18, 4.12.44. The right upper lobe is adherent to the chest wall and shows infiltration with probable cavitation. There is a basal hydro-pneumothorax, the right lower lobe being opaque, collapsed and airless.

The left lung markings are accentuated and there is scanty discreet mottling in all zones on the left side. Trachea central.

No.19, 29.11.45. Right upper lobe appearances I.S.Q. The right lower lobe appears to have expanded slightly but is still opaque. Marked thickening of parietal pleura over this area. There is an intercostal tube into the pleural cavity and lipiodol has been injected.

The appearances remain stationary on the left side.

No.20, 29.11.45. This is a pleurogram taken with the patient lying on the left side, and it shows the vertical extent of the empyema cavity.

No.21, 3.6.46. A partial thoracoplasty has been done on the right side and a drainage tube extends to the upper limits of the space.

No.22, 28.8.46. This is a P.A. vertical sinogram and shows the lower limits of the empyema space.

No.23, 28.8.46. This P.A. "head down" sinogram shows the upper limit of the empyema space.

Nos.24 and 25, 28.8.46. Those right lateral sinograms in the "vertical" and "head down" positions further demonstrate the extent of the residual empyema cavity.

10R. R.S. No.20,628. A female, aged 19 years, R.B.2 classification at empyema onset. No Blood Sedimentation Rate records. Admitted with empyema which came on 3½ months from left artificial pneumothorax induction. No

10R. R.S. No.20, 628 (continued):-

adhesion section and no positive pressure refills at any time. Treatment was by aspirations at first and then a stab drain and washouts. A large bronchogenic spread via left broncho-pleural fistula occurred and death in ten months from empyema onset.

X-rays (Nos.1 and 2) after empyema onset show -

No.1, 5.2.47. Grid photograph.

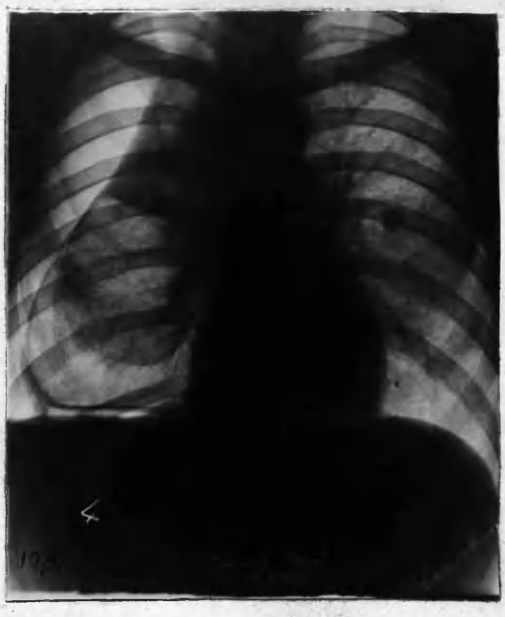
The heart, trachea and mediastinum are displaced to the left. Slight peaking of right diaphragm. Right lung appears clear. Pleural effusion in left artificial pneumothorax space. The left lung is extensively cavitated, especially in its upper half, and milky looking.

No.2, 13.6.47. The right lung in its lower half shows an extensive soft, infiltration. This extends into the upper zone. Intercostal De Pezzer catheter on left side and very little free fluid. The left lung is still semi-collapsed and there is evidence of thickened parietal and visceral pleura. Extensive left apical cavitation very obvious.

10S. L.Q. No.20,607. A male, aged 29 years, R.B.2 classification, and Blood Sedimentation Rate 16 mms./hour at onset of empyema which developed three weeks after right artificial pneumothorax induction in another hospital. No positive pressure refills and no adhesion section. Admitted with empyema, ill and dyspnoeic, he had repeated aspirations and instillations of blood and penicillin. Cachexia progressed and death in four months from onset of pus with failure of the lung to re-expand. Not fit for major surgery.

X-rays (3, 4 and 5) show -

No.3, 10.2.47. There is a spacious pyo-pneumothorax on the right side. Right lung is collapsed and of "ground-glass", opaque appearance. An apical adhesion is visible and an oval vomica the size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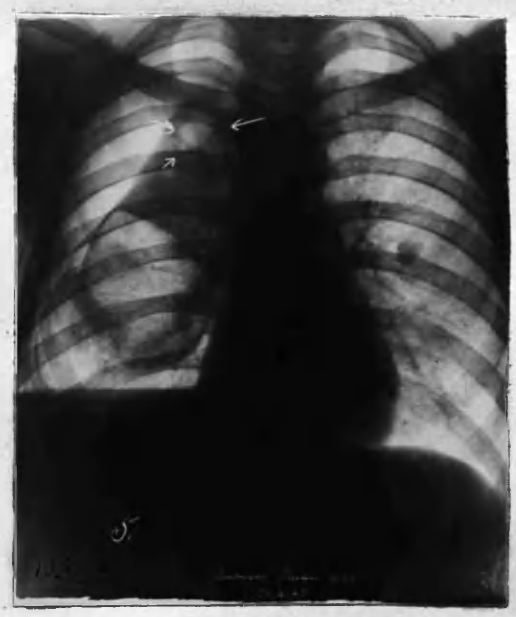
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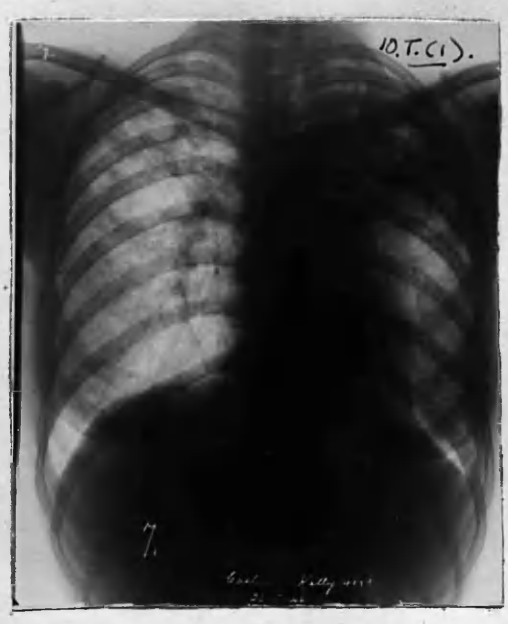
10.T.(1).
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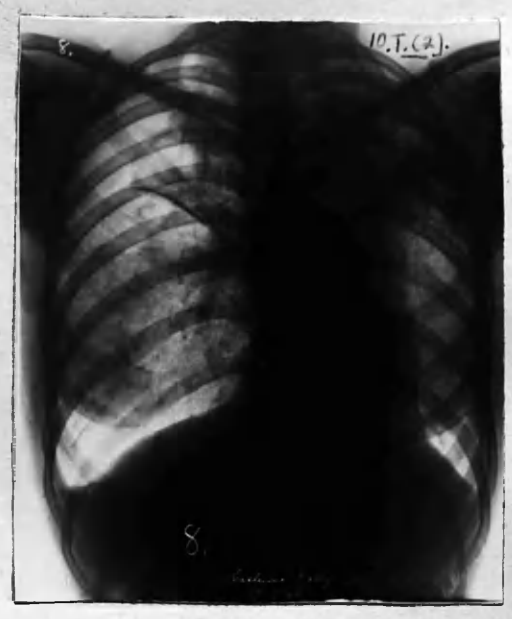
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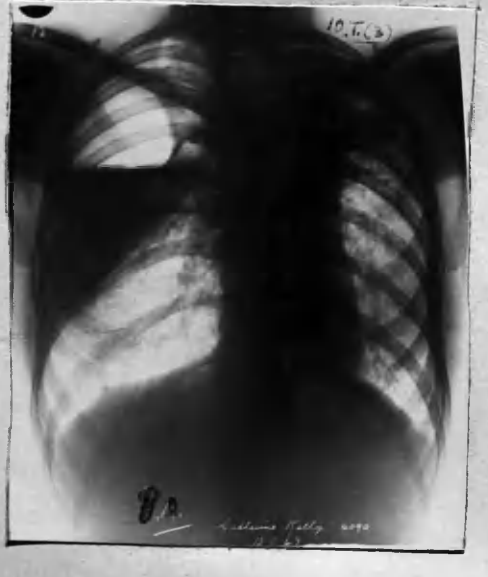

10.T.(3)
B.A.


10 T(4).

10S. L.Q. No.20,607 (continued):-

of a pigeon's egg (denoted by arrows).

Scanty hard disease on left side with one small round focus near the end of the third rib.

No.4, 31.3.47. Right artificial pneumothorax present with a basal layer of fluid. Evidence of early pleural thickening (both parietal and visceral). The right lung is now semi-expanded. Right upper lobe is opaque and "ground-glass" like, with apico-mediastinal adhesions visible and large central cavity obvious.

Left lung has a fine miliary type of mottling as has the right lower lobe.

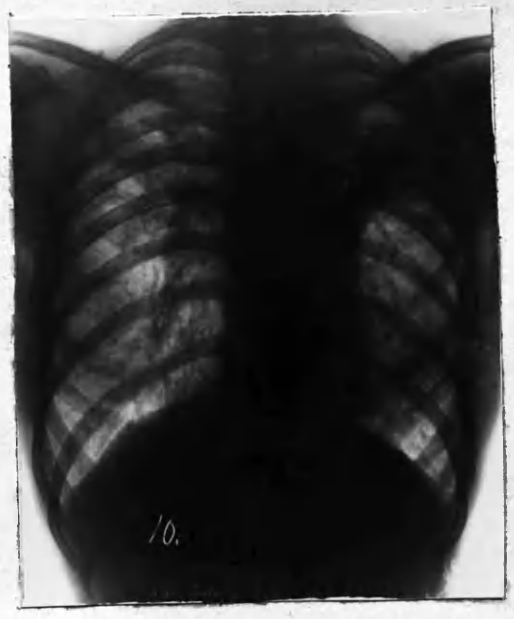
No.5, 28.4.47. Right lung slightly more expanded. There are still apico-mediastinal adhesions and appearances are otherwise as on the previous film. Trachea well over to right.

10T. C.K. No.19,891. A female, aged 25 years, R.B.2 classification, and Blood Sedimentation Rate 16 mms./hour at empyema onset.

Admitted 15.3.45. R.B.2. Left pleurisy three years before admission.

On admission, X-ray No.7, 22.5.46, shows coarse mottling all zones right lung but worse in upper zone. Large loculated vomica below and behind right clavicle in the peripheral border of the lung. Fairly dense infiltration in this area. Heart, trachea and mediastinum displaced to the left with evidence of thickened pleura over the left lung leading to peaking of left hemi-diaphragm and obliteration of left costo-phrenic angle. Several large cavities evident in left upper lobe.

A left artificial pneumothorax was induced on 10.6.45 but abandoned on 28.7.45 as contraselective. Clear fluid developed but the left lung re-expanded after repeated aspiration. Right artificial pneumothorax induced 21.6.46.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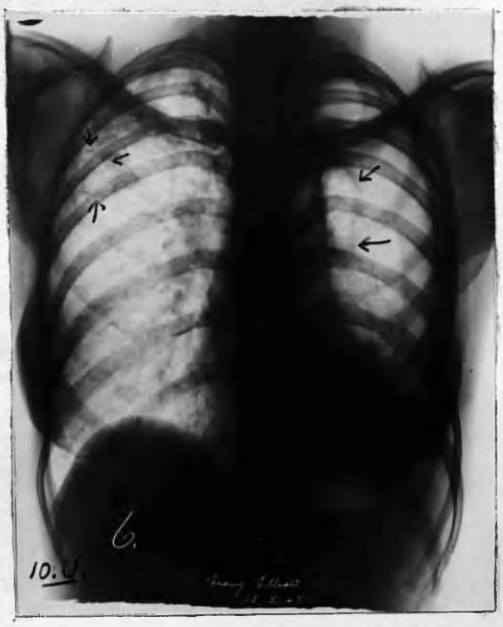
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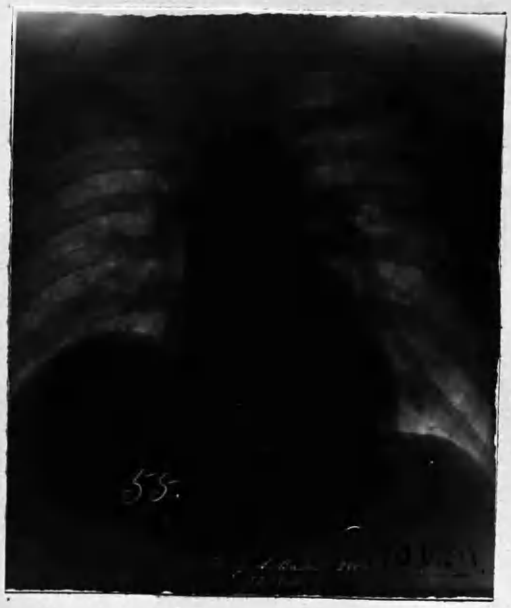
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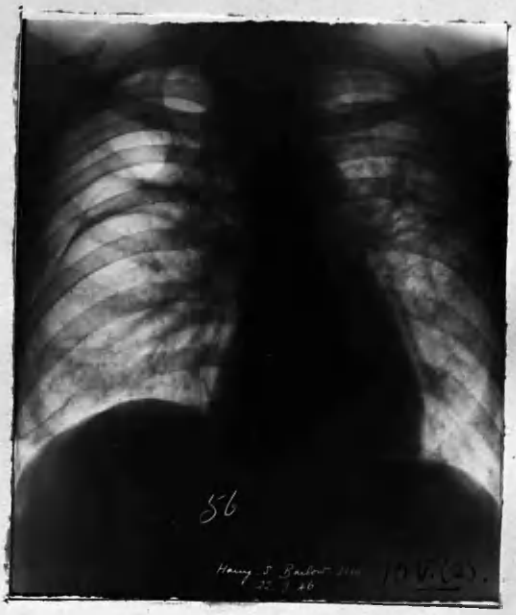
10.V.(1).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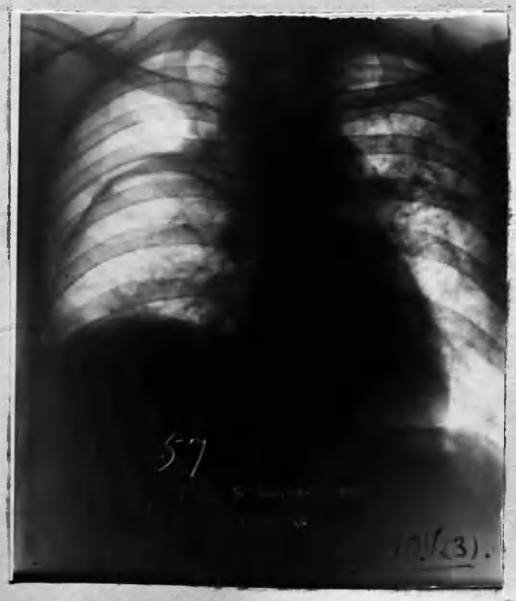
10.V.(2).


57
10.V.(3).


58.
10.V.(4).

10T. C.K. No.19,891 (continued):-

No.9A, 12.2.47. The right lung has expanded at the base but above this is a hydro-pneumothorax with fluid up to anterior end of second rib. The right upper lobe is collapsed and held out to the apical region by adhesions. A vomica is still visible in the right upper lobe which is still semi-opaque in places. Large vomica left upper lobe.

No.9B, 12.2.47. This lateral film shows the fluid layer and confirms the presence of right upper lobe vomica.

No.10, 26.11.47. The right lung is now completely re-expanded. There is pleural thickening over the upper and outer area. Note extensive mottling in all zones with apical cavitation. The left apical vomica is now much larger and there is a fresh spread in the left lower lobe.

(Death by extension of disease. Not empyema).

10U. M.E. No.20,755. A female, aged 23 years, R.B.2 classification. No Blood Sedimentation Rate record at empyema onset.

Admitted 12.5.47. R.B.3. Weight 6st. 2 lb. Thin and toxic and pyrexial. Diagnosed pulmonary tuberculosis in 1944. Had left artificial pneumothorax in June/1944, followed by adhesion section in June 1945 in a sanatorium in another county. Discharged with fluid in left chest in November/1945 and came to Manchester. Left artificial pneumothorax abandoned in September/1946 due to empyema. Had been awaiting admission since then. Aspirations until 26.7.47 when an intercostal stab drain was introduced and tube led to water seal at floor level. Daily Azochloramide washouts and breathing exercises. She was very dyspnoeic before her tube was introduced. Screening showed no expansion of the left lung. The pleural fluid was not infected with secondary organisms at any time. Death from cachexia on 11.10.47, the empyema having lasted thirteen months.

10U. M.E. No.20,755.(Continued):-

X-rays show -

No.6, 13.5.47. The right hemi-diaphragm is elevated. There is a coarse mottling throughout the right lung with honeycombing of the apex and a larger vomica at the periphery below the right clavicle. A left pneumothorax is present with a fluid level. The left lung is semi-collapsed and has thickened pleura over it. Appearances suggest a large vomica below the sternal end of the left clavicle. The heart and central mediastinum are tending to move to the left.

10V. H.S.B. No.19,580. A male, aged 37 years, R.B.3 classification and Blood Sedimentation Rate 32 mms./hour at onset of empyema.

Admitted 13.7.44. R.B.3. Weight 11st.5½ lb. Tuberculous laryngitis on admission. Illness commenced with slight cough in August/1942. In April/1944 he was X-rayed and diagnosed as pulmonary tuberculosis. Screening on admission showed bilateral disease with cavitation. 29.11.44. Left artificial pneumothorax induced but abandoned on 3.5.45. Contraselective in spite of adhesion section on 4.1.45. No fluid aspirated. 13.3.46. Right artificial pneumothorax induced. 5.8.46. Superadded spontaneous pneumothorax while on Bank Holiday Pass. Returned to hospital by ambulance. (Previous refill 600 ccs. 30.7.46, -14 -3, -8 ± 0). Temperature rose sharply. Screening showed marked collapse of the right lung whose upper and middle lobes completely disappeared against the mediastinum. Mediastinum grossly displaced to the left. There was a fluid level covering the right hemi-diaphragm. Air was removed from the chest and an exploratory aspiration showed greenish, turbid fluid. He was due to have a thoracoscopy but the spontaneous pneumothorax precluded it. An average of fifteen ounces of greenish fluid were aspirated every two days. On 11.9.46 the fluid was

 H.S.B. No.19,580 (continued):-

straw-coloured and opalescent, and the empyema could be dated from that date (six weeks after spontaneous pneumothorax).

Treatment was by repeated aspirations but the lung never expanded. On 30.4.47 he commenced a series of haemoptyses and died on 25.5.47. Fluid was tubercle bacilli positive and not a mixed infection. The pus lasted 8½ months.

X-rays show -

No.55, 17.12.45. Fine mottling all zones right lung with a vomica in the apex. Right hemi-diaphragm elevated.

Vomica, size of pigeon's egg, in second left interspace.

No.56, 22.7.46. Right artificial pneumothorax present with apical vomica still visible and at least six string-like adhesions holding out right upper lobe to chest wall.

Vomica on left side has increased in size and has a fluid level. Some thoracic spine scoliosis (concavity to right).

No.57, 7.8.46. Right artificial pneumothorax is still present and patent vomica in right upper lobe which is now semi-opaque at the base. One of the lower adhesions has parted, allowing collapse of the base of right upper lobe. Trace of fluid in right costo-phrenic angle. Left side in status quo.

No.58, 7.5.47. Hydro-pneumothorax present on right side. Upper lobe opaque and with a large vomica visible. Apical adhesions still obvious but thickened like the visceral and parietal pleurae. Trachea over to right. Increase of soft mottling in left mid.zone.

10W. B.E. A female, aged 21 years, R.B.2 classification, and Blood Sedimentation Rate 18 mms./hour at onset of empyema. Ten months after contraselective left artificial pneumothorax induction, empyema developed. No adhesion section and no positive pressure refills. Repeated aspirations made and finally intercostal drain and penicillin washouts. Tubercular meningitis and death after empyema had lasted six months. No X-rays available.

10X. K.McM. No.20,582. A female, aged 23 years, R.B.3 classification, and Blood Sedimentation Rate 10 mms./hour at empyema onset.

Admitted 9.1.47. Sputum positive.

Large vomica in apex of right lower lobe. Ill. The vomica was too large and old-standing to be affected by phrenic crush and pneumoperitoneum treatment.

Bronchoscopy on 28.2.47.

Right side. Right upper lobe openings and spur - normal.
Right middle lobe openings and spur -normal.
The right lower stem bronchus, and particularly the dorsal bronchus, were stenosed as a result of oedema of the mucous membrane, in particular the dorsal opening was very different from that on the left side.

Left side. Left upper lobe openings and spur - normal.
The dorsal opening and spur were clearly seen and quite different from the corresponding one on the right side. There was an escape of frothy fluid from the middle and dorsal openings, and a little non-offensive muco-pus was seen in the middle of the main bronchus. The basal bronchus and spur could not be brought into view.

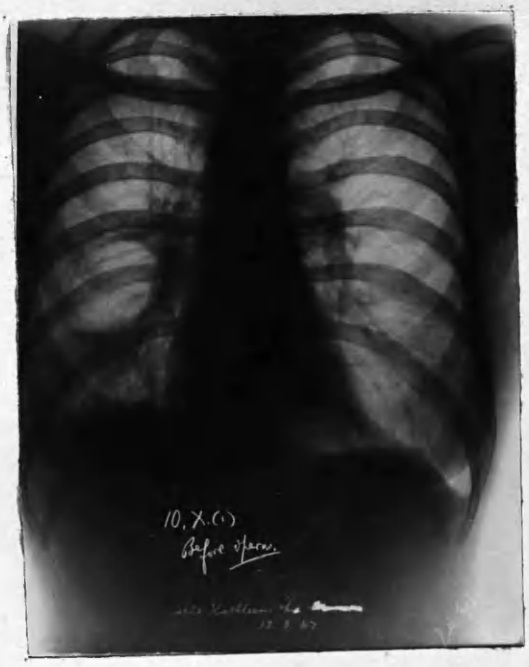
Summary: Bronchoscopic evidence of localised inflammatory lesion of the right lower lobe stem and dorsal bronchi.

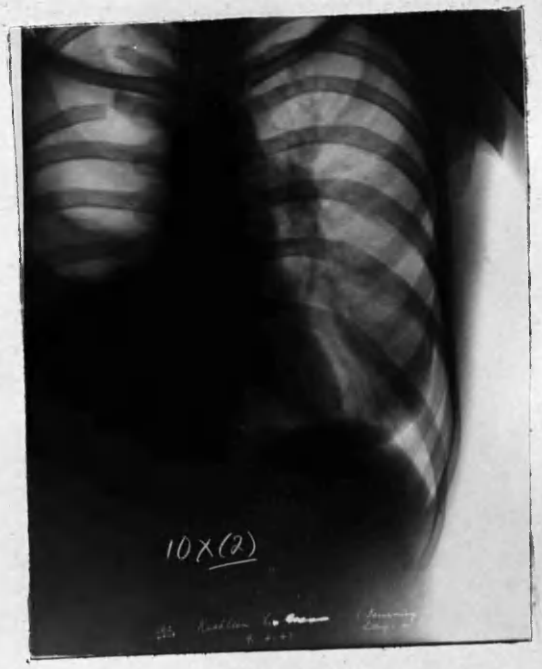
14.3.47. Pneumonectomy (Mr. G. Bryce).
General anaesthesia.

 K.McM. No.20,582 (continued):-

Blood transfusion during the operation. Routine exposure through the fifth space after removal of the fifth rib and 1" of the fourth and sixth near their transverse processes. Serratus magnus, latissimus and rhomboids divided. The lung was extensively adherent, particularly around the posterior aspect of the lower lobe. These adhesions were easily separated except in the area of the disease and at the base of the lung. It was intended to perform a right lower lobectomy but as the fissure was entered and the hilum approached, it became clear that the disease had encroached on the posterior segment of the upper lobe and only a pneumonectomy was feasible. The vessels of the hilum were of medium length. The pulmonary artery was injured in one of its branches and a certain amount of time was consumed in getting the vessels on this account. The bronchus was divided between clamps and sewn with three mattress sutures and ligatures. It was satisfactorily covered with a pleural flap. The adhesions of the affected segment of the lower lobe were left until the end of the operation and separation was affected in the extrapleural plane. As far as could be seen no tuberculous tissue was cut across and there was no fouling of the pleural cavity. The chest was closed without drainage. Sulphonamide-penicillin powder was sprinkled over all and 80,000 units of liquid penicillin instilled after closure. The specimen showed the expected cavity - 5 cm. in diameter. It contained some thin pus. Two branches of the dorsal segmental bronchus opened into the vomica near its upper end. The main bronchus appeared healthy but histologically tuberculous foci were present between the epithelium and the cartilage but they had not ulcerated the mucosa.

17.3.47. Twenty-one ounces of dark blood aspirated from chest and penicillin instilled. Thereafter she had


10, X. (1)
Before Starvation


10 X (2)

10X. K.McM. No.20,582 (continued):-

regular aspirations every two or three days and the fluid from blood became densely purulent by 5.4.47. On 14.4.47 she developed an urticarial rash due to penicillin.

15.4.47. Her pleural pus contained tubercle bacilli but no other organisms.

25.4.47. Right intercostal stab drainage.

14.5.47. Death from a massive haemoptysis.

Post-mortem No.9 describes the condition. She had a large broncho-pleural fistula and the margins of this area had ulcerated and eroded a branch of the pulmonary artery through which she had a secondary haemorrhage into the right pleural cavity. This, then, is an example of tuberculous empyema following pneumonectomy. The main bronchus was bronchoscopically clear and at operation was normal to the naked eye. It had on histological examination tubercles beneath its epithelium and this area was cut across at operation.

X-rays show -

10X(1) 13.3.47. Taken before left pneumonectomy this shows a large, tense-looking, right lower lobe vomica with fluid level. There is much opacity around and distortion of the draining bronchus. Left lung field is clear.

10X(2) 9.4.47. The right lung has been removed and a fluid level is seen in the pleural cavity. Several fibrin ridges are visible. Left lung field remains clear.

This represents the end of the series of cases under review up to the end of 1947. The following cases are included to show special points in treatment or management. Additional cases of tuberculous empyema treated by surgery are included. They were admitted from other regions for operative treatment and the survivors still attend for review.

No.1. M.I., aged 32 years, 19,198. R.B.2 on admission.

She had a left artificial pneumothorax induced in December/1942, left phrenic crush on 24.9.43, partial adhesion section 31.12.43. Green fluid formed and cleared with several aspirations. On her return home, with refills by an outside authority, an empyema developed on 26.2.44 with clinical broncho-pleural fistula (pus tubercle bacilli positive only). (This artificial pneumothorax after adhesion section was in Group IV previously described, with fluid appearing under six months).

After several aspirations left anterior stab drainage was done, preparatory to thoracoplasty, on 14.7.44.
9.2.45. Intended first stage thoracoplasty could not be performed as some residual pus in the empyema space flooded the respiratory passages during anaesthesia induction. It was deemed inadvisable to proceed but instead another intercostal, malecot tube was inserted through the sixth intercostal space in the axillary line.
16.2.45. Vital capacity 1,200. First stage left thoracoplasty (antero-lateral) by Mr.G. Bryce.

General anaesthesia.

Incision along the second rib, anteriorly. Upper ribs exposed through a split through the pectoralis major muscle. The second rib was resected first ($6\frac{1}{4}$") from its cartilage backwards. The third rib was resected from its cartilage as well (6"). The resection of the first rib was somewhat difficult due to underlying fibrosis, limited exposure and proximity of the axillary and subclavian vessels. Nevertheless about $2\frac{3}{4}$" of the first rib was resected from its cartilage. During the resection of the third rib some pus escaped through the pleuro-cutaneous fistula into the operative area. Sulphanilamide powder was applied and the wound sutured in layers. A drainage tube was left through the old sinus.
X-ray 28.2.45. Right lung shows fresh mottling near the hilar region suggestive of acute congestion from aspiration of pus and blood.

M.I. (continued):-

16.3.45. Second stage left thoracoplasty by Mr.G. Bryce.

General anaesthesia.

Upper periscapular incision with division of trapezius and rhomboids. Resection of the remaining segments of the following ribs:-

first rib (1½"), second (3"), third (4½"), followed by resection of 6¼" of the fourth rib and 1½" of the fifth rib, without transverse processes. No apicolysis attempted. 20% tannic acid applied to the periosteal beds. Sulpho-penicillin pulv. applied and wound closed in layers.

2.4.45. General condition was only fair and there was a hissing sound from the broncho-pleural fistula. No pyrexia, tube discharging two ounces of pus daily but thoracoplasty wound healed.

13.4.45. Vital capacity 1,050. Third stage left thoracoplasty by Mr.G. Bryce.

General anaesthesia.

Periscapular incision continued downwards. Remaining fibres of trapezius and rhomboids divided as well as a few fibres of the latissimus dorsi. Resection of small segments of the following already cut ribs:-

fourth rib (½"), fifth rib (5¼"). Resection also of the following ribs, sixth rib (5"), seventh rib (5"), eighth rib (3½") from their transverse processes forwards. 20% tannic acid and sulpho-penicillin powder applied to bony ends, periosteal beds and wound. Closure in layers. After the operation for a few days there was a troublesome cough and abundant frothy sputum and râles over the right lung. Sputum persistently negative for tubercle bacilli by this time.

15.5.45. Vital capacity 950. Fourth stage left thoracoplasty by Mr.G.Bryce.

General anaesthesia - pentothal, gas and oxygen. The drainage tube was removed. The old incision was partly re-opened and extended anteriorly. 1½" of the fifth, 2½" of the 6th, 2" of the seventh, 2" of the eighth, and 4" of the ninth ribs were removed. The anterior wall of the pleural

M.I. (continued):-

cavity was removed. 20% tannic acid was applied to the periosteal beds, excess being removed. The wound was dusted with penicillin sulphonamide powder. Remaining pleural cavity space was packed with thick gauze roll which was brought out through the anterior end of wound and secured. Wound was closed in layers. Bleeding was brisk throughout the operation and a plasma drip necessary. Moderate amount of breathlessness evident after the operation.

21.5.45. Gauze pack removed and a rubber tube inserted.

26.11.45. Discharged home with sinus requiring daily dressings. Blood Sedimentation Rate 10 mms./hour. Only fair general condition.

By July/1947 the pus from the left chest sinus was still tubercle bacilli positive. She required daily dressings. She was of fair general condition and had dyspnoea on exertion but could manage light housework.

By 3.4.48 the empyema had persisted for four years two months.

No.2. Mona M. 20,001.

A diabetic, controlled throughout by insulin. Admitted with right empyema following spontaneous pneumothorax January/1945. On admission an X-ray (20.6.45) showed a widely adherent right upper lobe containing a large vomica. A basal right hydro-pneumothorax was present. The left lung field was clear. Several aspirations of pus containing tubercle bacilli only were done and saline washouts performed.

8.7.45. Stab drainage low down in the anterior axillary line. Penicillin was instilled daily after saline washouts.

3.8.45. First stage right thoracoplasty by Mr. G. Bryce.

Local anaesthesia.

Periscapular incision. Division of trapezius and rhomboid muscles. Resection of third rib ($7\frac{1}{2}$"), second rib (7") and first rib ($2\frac{1}{2}$"). Mobilisation of the apex was easy in this case. The three first periosteal and intercostal muscles were divided. The apicolysis was concentrical and the entire upper lobe was liberated as far down as the azygos vein.

No.2. Mona M (continued):-

Then a further resection was made of the fourth rib (3½"), and fifth rib (2½"). Sulpho-penicillin pulv. applied to the wound which was closed in layers, leaving no drain. The week following this stage two aspirations of blood-stained extrapleural fluid were necessary.

10.9.45. Vital capacity 1,300. Second stage right thoraco-plasty.

Old incision re-opened. Resection of ribs sixth (4½"), seventh (4"), eighth (3½") without their transverse processes. Then further resection of already divided fourth rib (2½") and fifth rib (2½"). The rib resection was sufficient to allow of the scapula embedding. A corrugated rubber drain was left in the lower end of the wound for 36 hours.

12.10.45. Vital capacity 1,000 c.c. Third stage right thoracoplasty by Mr. G. Bryce.

Old periscapular incision re-opened and enlarged towards the spine. The existing anterior tube opening was enlarged. The empyema cavity was found extending to a lower level than the tube in the axillary region and also upwards, paravertebrally, as high as the seventh transverse process. Decostalisation done by resection of portions of ribs sixth (3⅛"), seventh (4"), eighth (6"), ninth (5"), tenth (4½") and eleventh (3¼"). The empyema cavity was then opened posteriorly and the tube inserted in the postero-inferior angle. The decostalised tissues were then stripped in the extrapleural plane from the upper extremity of the cavity. They were divided, thus mobilising this portion of the chest wall while preserving the visceral pleura. A twenty-four hours' drain was put into the antero-inferior angle of the wound. The latissimus dorsi, though narrow by previous stages, was preserved in its continuity. Sulpho-penicillin pulv. to wound and closure in layers.

23.1.46. X-ray shows good general collapse of right lung. The upper lobe looks bronchiectatic. A lateral opacity just above the diaphragm may indicate some residual fluid. The

No.2. Mona M. (continued):-

wound was healed. She was, mentally, not quite normal when discharged home.

No.3. Theresa G. No.18,596. R.B.2 on admission.

Onset of illness January/1939 with chronic cough, laryngitis and loss of weight. Left artificial pneumothorax in December/1939 followed by empyema in one month (outside area). For three years had closed intercostal drainage and frequent washouts.

Admitted Baguley Sanatorium 22.6.42. Bad dental caries present, corneal ulcers of both eyes, and left chest had a discharging sinus with surrounding unhealthy skin. Operation was postponed until dental sepsis was cleared up and the skin around the sinus was more healthy.

21.8.42. First stage left thoracoplasty by Mr.G.Bryce.

Empyema sinus excluded from operation area by sticking a Batiste towel with Mastisol. Short paravertebral incision. Resection of upper four ribs with portions of second and third transverse processes. First rib removed from its tubercle to the cartilage. Periosteal beds treated with 20% tannic acid. No drain.

18.9.42. Second stage left thoracoplasty by Mr. G. Bryce.

Periscapular incision continued downwards. Resection of additional lengths of third and fourth ribs, and also portions of fifth, sixth and seventh ribs, with corresponding transverse processes. 20% tannic acid to rib beds. No drain.

23.10.42. Third stage left thoracoplasty by Mr. G. Bryce.

Peri-mammary incision. Resection of stumps of second ($2\frac{1}{4}$"), third (3") and fourth ($4\frac{1}{2}$") ribs. Mobilisation of second and third costal cartilages. No drain.

2.12.42. Pyrexial for several days and there is a profuse discharge of pus from her sinus.

18.12.42. Fourth stage left thoracoplasty by Mr. G. Bryce.

Sinus opened up with diathermy needle and removal of regeneration ring of bone. The cavity was found to be obliterated to the extent of the previous rib resections but

No.3. Theresa G. (continued):-

there remained the paravertebral and anterior prolongations (shown by a sinogram of 12.10.42). Resection of para-vertebral segments of five ribs overlying the posterior position of the cavity. Corresponding flap of outer wall of cavity mobilised by dividing it in an upward and forward direction and also upwards and backwards along its anterior oblique attachment. The flap fell inwards. It was not hinged as in the complete Robert's technique. The anterior position of the cavity was thus left roofed over by three ribs which were marked down for resection later. A new anterior, intercostal stab drain was established at the foremost angle of the cavity. A sub-cutaneous pack was placed outside the outer wall of the empyema cavity so as to approximate it to the visceral wall. Wound partially closed, leaving the end of the pack protruding.

23.12.42. Offensive discharge from wound. Profuse discharge from tube. Pack removed.

27.5.43. Wound still ulcerated and unhealed. Residual empyema cavity persisting. Patient discharged home and to return for further operation when wound had healed but she deteriorated and died in the meantime.

<u>No.4. John A.R. 18,322.</u>

Admitted 10.11.41. Grade R.B.2. History of being in the water for several hours at Dunkirk evacuation (May/1940). September/1940 onset of pleurisy left base.

Left artificial pneumothorax induced 3.2.42, left phrenic crush 30.6.42, and thoracoscopy without division of adhesions 25.8.42.

Empyema onset 1.11.42 and thereafter regular aspirations and Azochloramide washouts. The empyema became secondarily infected with Staph. Aureus. On 21.5.43 a stab drain was inserted in the fifth left interspace in the anterior axillary line. The lung expanded up to a point only and the parietal and visceral pleurae were greatly thickened.

13.2.45. First stage thoracoplasty (left) by Mr. G. Bryce.

Partial upper periscapular incision. Division of upper fibres of the trapezius and rhomboids without dividing

No.4. John A.R. (continued):-

the levator scapulae. Resection of ribs, third (4½"), second (5") and first (3"). The ribs were found to be triangular in shape and very close together. Resection was difficult owing to extensive fibrosis and rigidity of the tissues below and between the ribs. No apicolysis was possible. Resection was then done of ribs fourth (3¼") and fifth (1¾"). Sulphanilamide pulv. was applied and the wound closed in layers with no drain.

9.3.45. Second stage left thoracoplasty (paravertebral) by Mr. G. Bryce.

General anaesthesia.

Periscapular incision continued downwards. Much bleeding present. No division of muscles. Rib resection difficult owing to extensive underlying fibrosis and materially welded condition of ribs, leaving practically no intercostal spaces around the operative area. Resection of a few inches of ribs fifth, sixth, seventh and eighth with their transverse processes. 20% tannic acid to rib beds. Sulpho-penicillin powder applied and the wound was closed in layers with no drain.

9.4.45. Third stage left thoracoplasty.

Old periscapular incision re-opened and enlarged downwards by a small fresh one towards the spine. The scapula was materially welded to the underlying chest wall and its separation was laborious: cut ends of the third, fourth and fifth ribs were seen underneath the angle of the scapula to be firmly welded into one bony mass. Portions of ribs resected were sixth (2"), seventh (1½"), and eighth (2"). The parietal pleura appeared to be very tough and thick. Resection of vertebral portions of ribs ninth (3½") and tenth (3") with part of their transverse processes. 20% tannic acid applied to rib beds and sulpho-penicillin pulv. applied to wound which was closed in layers with no drain.

26.10.45. Schede's Operation by Mr. G. Bryce.

General anaesthesia.

Operation on the Schede principle. Removal of outer border

No.4. John A.R. (continued):-

of empyema cavity throughout its extent from the level of the superior angle of the scapula to about the eleventh rib in the paravertebral line. The cavity was completely unroofed but it was felt that the scapula might form a new 'tent-like' roof to the upper portion of the cavity. In that case it was noted that it might be necessary later to remove the entire vertebral border of the scapula throughout its length. Closure in layers, leaving a corrugated rubber drain in the most dependent area. There was much shock after this operation.

14.4.46. Sinograms revealed a compressed, elongated cavity extending from the fifth rib behind to the costo-phrenic gutter below.

By 25.4.48 the sinus and residual empyema cavity persisted. His condition was "fair" only and he was thin and sallow. The right lung was clear on X-ray but he still had occasional sputum, i.e., final result was indifferent and the outlook poor.

To 3.4.48 the empyema had lasted five years five months.

No.5.K.D. 20,833. (See page 122).

No.6. Doreen R. 20,861.

A female case, aged 23 years, R.B.2 classification on 31.7.47. Diagnosed pulmonary tuberculosis May/1946. Bilateral apical cavitary disease, worse on left side.

Right artificial pneumothorax induced 22.8.47. The idea was to prepare her for a future left thoracoplasty.

Adhesions held out the right apical vomica to chest wall. On 22.10.47 a right Jacobeus operation was done. The right lung was pink in colour with the three lobes perfectly separated. The upper lobe was held up by two main adhesions, both postero-lateral, one against the third rib, the other against the second. Both were completely divided. As soon as it was freed from its parietal attachments, the entire lung was seen to collapse against the hilum. No bleeding occurred.

No.6. Doreen R. (Continued):-

11.11.47. Twenty-two ounces of fluid aspirated - turbid, tubercle bacilli positive.

13.11.47. Dyspnoea troublesome. Pleural pressures +4 +6. 400 c.cs. air removed with some relief. Dyspnoea soon returned and gas analysis revealed O_2 8%, CO_2 6%, i.e., a fistula of moderate dimensions.

Tube inserted 17.11.47 and continuous suction applied. The air could be heard whistling in the fistula when the tube was inserted. The lung failed to re-expand. Her condition rapidly deteriorated and she died on 6.1.48. Post-mortem refused.

In this case gas analysis indicated the size of the fistula, confirmed the clinical findings and indicated a poor prognosis.

The condition of the opposite lung precluded any form of surgical treatment.

No.7. Catherine J. 20,634.

A female case, aged 22 years, R.B.2 on admission. Admitted to another hospital 18.4.46 and tuberculosis diagnosed. Admitted here 29.1.47 with a right artificial pneumothorax already induced.

On admission an X-ray showed a right artificial pneumothorax with limited apico-mediastinal adhesions and fluid absent from the intrapleural space. There was mottling with slight cavitation at the left apex.

Left artificial pneumothorax induced 25.2.47 and partial adhesiotomy 19.5.47. The main adhesion consisted of a tough, triangular shaped membrane inserted against the third intercostal space posteriorly and holding up the posterior aspect of the left upper lobe. Although it was seen to run backwards towards the costo-vertebral canal and not to have a way through it, it was partially divided by enucleation as far back as possible down to a point where the lung itself was adherent to chest wall. It appeared doubtful if the apical cavity would close by the relaxation thus obtained.

No.7. Catherine J. (continued):-

Clinical signs of spontaneous pneumothorax on 26.5.47. Dyspnoea and intrapleural pressures of -5 + 15, 600 c.cs. air removed with relief of symptoms. 29.5.47 much fluid present (cloudy yellow at first). This was turbid and purulent by 4.6.47 and contained no tubercle bacilli; coagulase positive Staph. Aureus and Diphtheroid bacilli were seen on smear. Aspirations and Azochloramide washouts were repeated and then large doses of intrapleural and intramuscular penicillin given. By 22.9.47 her general condition had improved, Blood Sedimentation Rate 18 mms./hour and the effusion was like jelly and could not be aspirated. By 15.1.48 the left pleural opacity had largely cleared and the lung had almost completely re-expanded with gross pleural thickening. On 25.3.48 she was discharged - laryngeal swab repeatedly negative.

This is a case of empyema following partial division of an adhesion. Secondary infection, which may have been introduced from the exterior during one of her frequent needlings, was cleared up with Azochloramide washouts and finally penicillin. The latter caused the empyema fluid to become "Jelliform" with subsequent pleural thickening and almost complete lung re-expansion.

No.8. Alan A.P.

A male case, R.B.2 on admission, aged 19 years. On admission 13.11.47 there was a small right apical vomica and some mottling in left upper lobe with early cavitation. Sputum was tubercle bacilli positive. Right artificial pneumothorax was induced 27.11.47 and a right adhesion section done on 15.12.47 for apical adhesions. At operation two main adhesions were seen and both divided completely.

13.1.48. Clinical spontaneous pneumothorax. Pain, dyspnoea, and later stair-like elevation of temperature. Lung collapsed.

15.1.48. Eighteen ounces turbid fluid removed from chest, tubercle bacilli positive.

16.1.48. Treatment with Nisin commenced. Daily aspirations

No.8. Alan A.P. (Continued):-

and 10 c.cs. Nisin daily. The fluid by 28.2.48 was thick pus and had gram positive cocci present.

By 3.3.48 three small sinuses formed in right chest wall. Those rapidly coalesced and formed a large, evil-looking sinus. The fluid continued to yield large numbers of tubercle bacilli in spite of Nisin therapy, and the lung failed to expand.

Toxaemia and pyrexia were marked and he died on 12.4.48. A post-mortem (No.24) was done.

On 5.3.48 pleural fluid tubercle bacilli positive and secondary organisms, Staphs., still present in small numbers. From 21.3.48 the dose of intrapleural Nisin had been 1×10^6 units/day and he had Nisin powder (1×10^6) units to his sinuses and Nisin cream (1×10^6) units to the skin around. On 11.3.48 red blood cells 4,740,000/cmm. and white blood cells 12,750 cmm.(Polymorphs 79% and Lymphocytes 17% and Monocytes 4%). The interesting factor here is the failure of Nisin to control the tubercle bacilli or the secondary infection. The post-mortem revealed with what rapidity an entire collapsed lung can become carnified and incapable of expansion, i.e., three months exactly.

No.9. Lily B. No.21,101, aged 17 years.

Admitted to another hospital on 10.1.48 with pain over the right lower ribs of recent onset. Treated by a few aspirations at irregular intervals for purulent effusion following spontaneous pneumothorax. Admitted to Baguley Sanatorium 2.2.48 with the right lung collapsed against the mediastinum which was displaced to the left. The left lung field was clear. She was dyspnoeic, thin and in poor general condition, and had a pyrexia of 102 to 103°F. throughout her period in hospital. There was no sputum at any time. Gas analysis revealed CO_2 14%, O_2 5%. Vigorous aspirations were commenced every three days and the pus yielded tubercle bacilli only and no secondary organisms at any time. The lung quickly re-expanded to half its volume and resolutely refused to expand further. Negative intrapleural pressures

No.9. Lily B. (continued):-

could be maintained between aspirations showing no large broncho-pleural communication. From the outset 10 c.cs. P.A.S. were injected every three days. This had no effect on the bacteriological content of the pus or on its amount or nature. She deteriorated slowly and developed tuberculous meningitis from which she died on 21.5.48 (see post-mortem No.26).

The interesting feature of this case is the minimal, peripheral area of tuberculous disease in the right lung which leaked and caused an empyema. It might even have been associated with a primary focus as no evidence of previous primary disease was seen radiologically or at post-mortem. Even in this minimal parenchymal case, P.A.S. was of no use given intrapleurally. Intrapleural negative pressures and gas analysis revealed that there was no gross broncho-pleural fistula, yet with vigorous aspirations after the empyema had lasted one month, the lung only semi-expanded. Post-mortem showed that the visceral pleura was so firm and like a rind round the lung that complete expansion was a physical impossibility, although the underlying lung was not carnified or pneumonic and was diseased only in one small area at apex of right lower lobe. She was too ill for surgical intervention and, moreover, deteriorated from the outset.

No.10. Henry W.G.M., 20640.

First admitted 12.5.44. Tubercle bacilli positive. Left artificial pneumothorax 19.5.44 for disease with cavitation left upper zone. Contraselective artificial pneumothorax. Left phrenic crush 3.8.44.

17.11.44. Thoracoscopy and Adhesion Section. Adhesions were only partially severed at the apex.

19.2.45. Empyema present three months after adhesion section. Aspiration of large volumes of pus at two or three weekly intervals, tubercle bacilli only.

15.8.45. Sinus formed through a needle track and secondary

No.10. Henry W.G.M. (Continued):-

infection of empyema cavity with gram positive cocci occurred.

21.8.45. Intercostal stab drain through the sinus area. Thereafter there were daily Azochloramide washouts. By 29.1.46 there were four ounces of pus daily from tube. The left lung had almost completely expanded but had stopped short of full expansion and the pleural layers were very thick. A pleurogram showed that the pleural space extended along the posterior chest wall from top to bottom. He was not fit for operative treatment owing to his long toxaemia. There was still a cavity at apex of left lung but the right lung field remained clear.

Intensive penicillin therapy had no permanent effect on the secondary infection.

9.5.47. First stage left thoracoplasty by Mr. G. Bryce.

General anaesthesia.

Periscapular incision. Trapezius and rhomboids divided in their totality. The scapula was divided first along a line which comprises removal of 2/3rds. of the bone and most of its paravertebral border. The muscles were divided partly by diathermy and partly with scissors. The exposure obtained was excellent. The ribs did not appear fused in spite of the chronicity of the underlying empyema. First and second ribs resected completely, first rib (3¾"), second rib (7¼"), together with their costal cartilages. No apicolysis was attempted. Wound closed in layers with nylon. No drain left.

6.6.47. Second stage left thoracoplasty by Mr. G. Bryce.

Limited perimammary incision. Subperiosteal resection of anterior portions of third rib (4"), fourth rib (5") and fifth rib (5½"). During resection of the fourth rib the pleura was punctured and had to be stitched. Closure in layers after powdering with sulpho.penicillin pulv. Rubber tube drain left. Peri-scapular incision was re-opened. Division of trapezius and rhomboids along Brock's line. There was some bleeding. Subperiosteal resection of remains of

No.10. Henry W.G.M. (Continued):-

third rib ($6\frac{1}{4}$"), fourth rib (9") and fifth rib (8"). Closure in layers after powdering.

27.6.47. Third stage left thoracoplasty by Mr. G. Bryce.

Periscapular incision re-opened. Resection of sixth rib ($7\frac{1}{2}$") and seventh rib ($6\frac{1}{2}$"). Trapezius divided along Brock's line to raise a muscle flap. Empyema cavity opened along the periosteal bed of the sixth rib. The cavity was seen to extend up to the level of the fifth transverse process, downwards to level of the eighth rib and anteriorly to mid.axillary line with a small pocket beneath the scapula. The parietal pleura was cut at right angles to the first cut and then towards the scapula, i.e.,

The main point about the operation was the stitching of the spinal border of the trapezius to the parietal pleura, leaving part of the muscle embedded into the cavity. This was aimed to abolish the unavoidable tent that is left if trapezius is sutured to trapezius. A large rubber tube was inserted through the old sinus and the wound powdered and closed in layers.

The patient had a severe pyrexial upset after this operation and large doses of penicillin were given through the tube and systemically. He refused to have a final stage to deal with the sinus.

By 5.8.47 he had three discharging sinuses along the thoracoplasty wound and those went in to the remains of empyema cavity.

22.1.48. 10 c.cs.(g.2) P.A.S. instilled every other day. By 1.6.48 the fluid still contains tubercle bacilli, Staph.Aureus and a coliform organism. Pyrexial and deteriorating. P.A.S. has had no effect on general condition or sinuses or bacteriology of the pus (one ounce pus daily). He has albuminuria 1 - 2 gm.daily. White blood cells remain at 7,650/cmms.

POST-MORTEM CASES.

No.1. Thomas C. 20,910, aged 55 years.

Larynx free. Illness began August/1936.

Hospital 26.11.36 - 11.1.37. 28.7.38 - 24.10.38.

2.10.39 - 19.11.39 (left against advice).

15.9.47 - 17.9.47 - died. R.B.3. No X-ray possible then.

Post-mortem 17.9.47. Right pleural cavity obliterated with adhesions. Right lung - generalised fibro-caseous disease. Scattered small vomicae. One large apical vomica adherent at periphery to chest wall. Walls of this vomica sloughing.

Left pleural cavity - numerous adhesions but less than on right side.

Left lung - apical vomica with epithelial lining, cyst-like.

Remarks: Trachea, main bronchi and main branch bronchi were examined carefully. Much tenacious muco pus present. No visible ulceration in any part that could be seen with a bronchoscope. Some reddening and swelling of the mucosa in the bronchus that drained the right apical vomica. Complete stenosis of left apical bronchus and vomica was held open by surrounding fibrosis and cyst-like cavity. Stenosis was at extreme limit of bronchoscopy. Histology of right main bronchus revealed no evidence of tuberculous involvement.

No.2. Fred B.A. 20704, aged 60 years.

Illness began February/1946. Hospital 17.4.47 - 2.10.47. R.B.2. Larynx free.

Post-mortem 2.10.47. Right pleural cavity - extensive dense adhesions with gross thickening of visceral and to a lesser extent of the parietal pleura in right upper lobe and part of middle lobe. No free fluid. Right lung - huge cavity at right apex, 3" x 1½". No caseous material, thickened fibrin wall. Surrounded by area of broncho-pneumonia, almost certainly tuberculous.

No.2. Fred B.A. (Continued):-

Left pleural cavity - extensive adhesions, particularly at apex and to the diaphragm.

Left lung - area of tuberculous broncho-pneumonia at the apex. No cavitation.

Remarks: The trachea, bronchi, and their main subdivisions were followed up in both lungs. No areas of abnormality discovered. Histological examination of main bronchi revealed no tuberculous disease.

(This man had also a left pyogenic pyelonephritis).

No.3. William O. 18,178, aged 36 years.

Illness began February/1941. Larynx free.

Hospital 21.7.41 - 27.4.42. 21.1.46 - 9.10.47.

R.B.3 classification.

Second stage right thoracoplasty completed 9.9.46.

Revision thoracoplasty 20.6.47. Sputum remained positive. Followed by extrapleural space infection and death.

Post-mortem 10.10.47. Right pleural cavity - extensive adhesions present at apex but lower lobe separated fairly easily.

Right lung and lymphatics - residual cavity identified in right apex. Broncho-pleural fistula identified through open scar. Large extrapleural space infected.

Right lower lobe normal.

Left pleural cavity - adhesions present but easily separated.

Left lung and lymphatics - scattered nodules throughout left lung with two caseous areas approximately $\frac{1}{2}$ cm. in diameter.

Remarks: The tracheo-bronchial tree was dissected (left lung). No evidence of stenosis or ulceration of the passages. Bronchi dissected well past the area of bronchoscopic vision.

Histologically there were early tuberculous lesions in the mucosa of the right main bronchus.

No.4. Alwyn B. 20,921, aged 44 years.

Hospital 29.9.47 - 13.10.47. R.B.3. classification.
Illness began January/1946. Larynx involved.
Post-mortem 14.10.47. Right pleural cavity - extensive adhesions present. Lungs could not be shelled out.
Right lung - extensive cavitation.
Left pleural cavity - extensive adhesions and lung had to be cut out.
Left lung - widespread cavitation.
The trachea and main bronchi were removed and opened. On posterior wall of trachea about 2" above main carina was an ulcer the size of a threepenny piece. Sectioned after photographing. The pallor of the ulcer contrasted with the general congestion of the rest of the tracheal mucosa. The remainder of the bronchial tree as far as the bifurcation of the main bronchi, showed some congestion but no definite ulceration. Small bronchi beyond bronchoscopic vision were congested.

"Report T.47/968. Sections of trachea show tuberculous ulceration. Many typical caseating tuberculous foci are present.

No.5. Wm. H.B.S. No.20,889, aged 43 years.

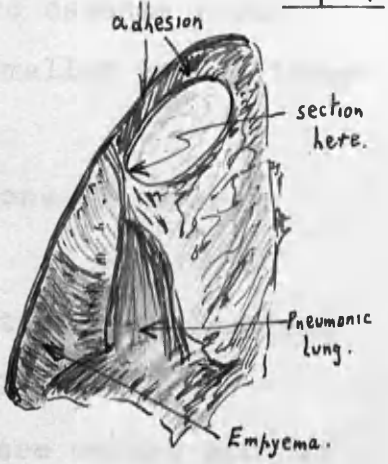
Illness began in 1942 probably. In hospital 4.9.47 - 14.10.47. No X-ray. R.B.3. classification. Larynx free.
Post-mortem 15.10.47. Right pleural cavity - basal empyema present. Many apical adhesions.
Right lung - large apical vomica part of whose wall was held to chest wall by adhesions. The inferior periphery of vomica, which was superficial, was next to the pleural space containing the empyema and un-guarded by adhesions (section made). Much tuberculous disease of right middle lobe.
Left pleural cavity - many adhesions. No free fluid.
Left lung - emphysematous. Several areas of pneumonic

No.5. Wm. H.B.S. (Continued):-

type of tuberculous disease. Hypostatic pneumonia at base.

Remarks: Trachea and bronchi examined. Obvious tuberculous bronchitis at junction of right apical, right pectoral and right main upper lobe bronchus. (Specimen for section). Right apical bronchus led directly into large right apical vomica. Left pectoral bronchus had some tuberculous bronchitis and drained an area of tuberculous pneumonia. (Specimen kept).

DIAGRAM.



No.6. Basil K. 20,915, aged 59 years.

Illness diagnosed January/1947 but pleurisy 30 years ago. In Hospital 25.9.47 - 21.10.47. R.B.2 classification. Larynx free. Died of coronary thrombosis. Post-mortem 22.10.47. Right pleural cavity - dense adhesions between upper lobe of lung and chest wall. Lower and middle lobes free from adhesions. (An artificial pneumothorax could have been induced).

Right lung - emphysematous and congested. Cavity in upper lobe. Lower and middle lobes free of disease.

Left pleural cavity - free of adhesions.

Left lung - congested, emphysematous lung - minimal tuberculosis.

Remarks: Trachea was healthy. Right upper lobe bronchus was the seat of tuberculous bronchitis. A partial, fibrous, stenosis was present in right sub-apical bronchus $\frac{1}{2}$ ins. prox. to entrance to cavity. (Photograph previously reproduced in section on tracheo-bronchial tuberculosis, page 63, and sections). Between stenosis and cavity ($\frac{1}{2}$ ins.) bronchial wall was softened with tuberculous bronchitis.

Patient died of coronary thrombosis confirmed at the post-mortem.

No.7. M.S.

Post-mortem 6.11.47. Right pleural cavity - free from adhesions except at apex where there was a stout band in the apico-mediastinal position. No free fluid and lower and mid. zones of lung easily shelled out.

Right lung - nodular from extensive caseous areas. Large vomica apex and mid. zones. Smaller vomica lower lobe in posterior position.

Left pleural cavity - dense adhesions all over, including diaphragmatic surface.

Left lung - large vomica apex. Rest of lung involved with caseous areas and several small cavities.

Remarks: The trachea and bronchi were opened up. At the limits of bronchoscopic vision some bronchitis was present in right lower lobe bronchus and around the origin of the lingula bronchus. Those bronchi draining the main vomicae were involved in the bronchitic process in their distal parts only, i.e., softening, swelling and reddening of the mucosa (A and B). No fibrous or other narrowings seen. Pus + + oozing from the main bronchial orifices. Note bronchitis most marked in right lower lobe bronchus.

No.8. F.L., aged 45 years. Larynx free.

Fairly early case of pulmonary tuberculosis. Left artificial pneumothorax established for two weeks. Sudden death while washing in the morning.

Post-mortem 20.11.47.

Well-nourished and well-built man. Petechiae left shoulder and arm.

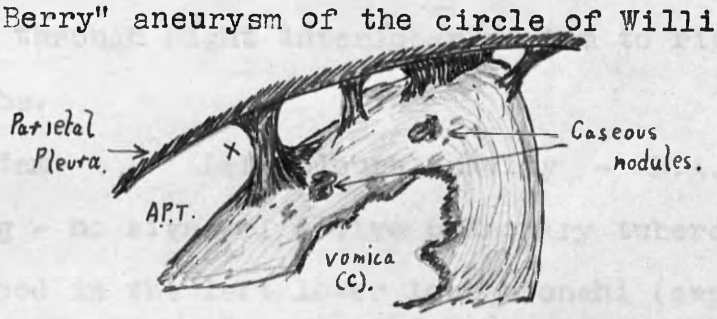
Right pleural cavity - a few apical adhesions. Lung was mainly free.

Right lung - bronchi N.A.D. Had fibro-calcareous nodules at apex. No visible vomicae. Entire lung deeply congested.

Left pleural cavity - no free fluid. Artificial

No.8. F.L. (Continued):-

pneumothorax present but lung was not entirely collapsed. No sign of spontaneous pneumothorax. Some adhesions at apex of lung subsequently found to be over a cavity (C) the size of a plover's egg. Left lung - petechial congestion on surface of lung. Cavity near apex and some calcareous, pea size nodules. Rest of lung deeply congested. The bronchus draining the cavity had inflamed and oedematous mucosa. Cranial cavity. Death due to subarachnoid haemorrhage from a "Berry" aneurysm of the circle of Willis.



Remarks: Trachea and bronchi within bronchoscopic vision were macroscopically normal. Only the bronchus draining the cavity was the seat of bronchitis. On artificial pneumothorax side one cuttable adhesion looked fleshy. This and right apical adhesions were removed for section with part of underlying lung. (See diagram above).

Section of left main bronchus just distal to main carina taken.

See report T.47/1088 of 5.12.47.

Microscopical examination:-

(1) Left main bronchus - a chronic inflammatory cellular infiltration is present beneath the epithelial lining but there is no structure to indicate tuberculosis.

(2) Left apical cuttable adhesion. Tuberculosis is plainly seen in the adjacent lung tissue. The adhesion contains an infiltration of small round cells and occasional plasma cells with no structure to indicate tuberculosis and no lung tissue.

No.8. F.L. (Continued):-

(3) Right apex. The thickened pleura covering the apex is composed of hyaline fibrous tissue and does not show any active tuberculous process.

No.9. Kathleen McM, aged 24 years.

On admission had a large vomica at apex of right lower lobe. Left lung appeared clear on X-ray. Right pneumonectomy 14.3.47. Empyema developed (mixed infection) and broncho-pleural fistula. Patient was intubated (25.4.47). On 15.5.47, after going steadily downhill, she had a sudden haemoptysis. Vomica extended through right interlobar septum to right upper lobe.

Post-mortem: Left pleural cavity - N.A.D.
Left lung - no signs of active pulmonary tuberculosis. Fresh blood in the left lower lobe bronchi (aspirated by breathing). Left lung was emphysematous.
Abdominal contents - N.A.D.
Right wall of mediastinum had a broncho-pleural fistula at site of pneumonectomy stump. There was some surrounding ulceration on side of mediastinum and inferior to the fistula there was an eroded pulmonary artery. The right empyema cavity was full of clotted blood. The whole side of the mediastinum showing fistula and eroded artery was removed and mounted. Later a portion of the right bronchus was taken from the mounted specimen and sectioned. At operation bronchus draining cavity was the seat of tuberculous bronchitis. Also right main bronchus was normal bronchoscopically and at operation, but the portion cut (T.47/248) showed "tuberculous foci between the epithelium and the cartilage but they have not ulcerated the mucosa."

No.10. Doris H, aged 18 years. Larynx free.

Pyrexial illness. Very few signs lungs. Too ill for X-ray when admitted. Emaciated. Gross oedema feet and legs. Died 9.5.47.

No.10. Doris H. (Continued):-

Post-mortem: Right pleural cavity - scattered tubercles on pleura. No adhesions.

Right lung - early tuberculous lesions throughout lung. Some breaking down to form small cavities.

Left pleural cavity - scattered tubercles on pleura.

Left lung - scattered, small, tuberculous lesions.

Heart - old rheumatic vegetations on mitral valve.

Liver and spleen had surface tubercles.

Small intestine had numerous tuberculous ulcers.

There was a bilateral, tuberculous, pyosalpinx.

No signs of tuberculous tracheitis or bronchitis.

The lung lesions were small and fairly recent. The main trouble here, which was probably the first lesion, was abdominal tuberculosis.

Histological examination of main bronchi showed no evidence of tuberculosis.

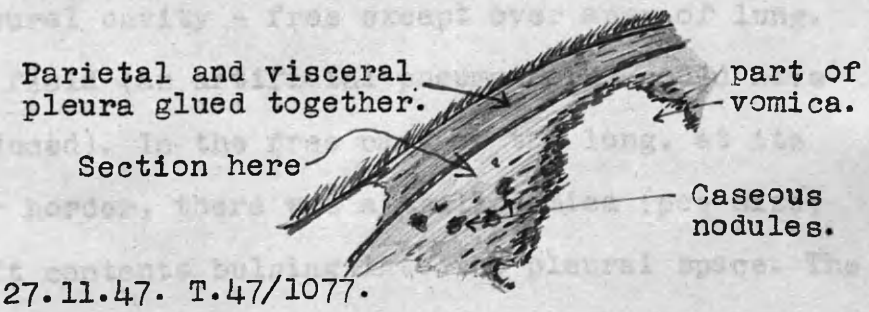
No.11. Arnold W., aged 66 years.

Post-mortem 18.11.47. Several enlarged glands at bifurcation of trachea.

Right pleural cavity - free in lower two-thirds. Adherent in upper one third.

Right lung - lower and middle lobes emphysematous. Medium sized vomica right apex.

Left pleural cavity - free in lower part but adherent over the apex. Both lungs were removed extrapleurally and a slice of right lung at the apex was taken.



"Report 27.11.47. T.47/1077.

Pleural surface. The section taken through the portion indicated on the diagram shows tuberculous areas, some proliferative and some caseous."

Left lung - lower lobe emphysematous. Small vomica at apex and pea size areas of firm caseation.

No.11. Arnold W. (Continued):-

Abdomen - free purulent fluid. Small bowel the seat of old-standing enteritis. Several hard, chalky glands at root of mesentery. Small bowel had atresia in two places. There was a Meckel's diverticulum 1½ feet from the caecum, with a small leaking ulcer at its base and loops of bowel glued around it. The cause of death was a low grade peritonitis and the pulmonary tuberculosis was probably not sufficiently extensive to kill him. Trachea and main bronchi examined. Only at the extreme limit of bronchoscopic vision was there evidence of bronchitis - swollen and red mucous membrane. From there distally there was evidence of bronchitis, especially at the smaller carinae. No stenosis seen. Section of left main bronchus taken just distal to main carina.

"Report 27.11.47. T.47/1077.

A small early tuberculous focus in the mucosa of the section examined."

No.12. E.S., aged 28 years.

Post-mortem 16.11.47. Glands at hilum enlarged.

Right pleural cavity - completely adherent all over. Lung could not be removed between pleural layers.

Right lung - total disorganisation of upper lobe of lung. The remainder was a foul-smelling, cheesy mass. No free lung tissue.

Left pleural cavity - free except over apex of lung. No free fluid (an artificial pneumothorax could have been induced). In the free part of the lung, at its anterior border, there was a small vomica (pea size) with soft contents bulging into the pleural space. The wall was thin and translucent. Several cheesy, hard nodules bulged into pleural space on left lung.

Remarks: Right lung was completely disorganised. All the remaining bronchi were softened and the seat of extensive bronchitis. Posterior wall of trachea

No.12. E.S. (Continued):-

was extensively ulcerated. Left main bronchus and left apical bronchus had reddened mucosa. No macroscopical ulceration and no visible stenosis. Specimen of left main bronchus removed ½ ins. distal to main carina.

"Report 27.11.47. T.47/1078.

The postion of bronchus examined shows no indication of tuberculosis".

Signs of chronic bronchitis present. Loss of ciliae. Thickening and organisation of submucous layer and marked hypertrophy of mucous glands. Two tags of adhesion over left apical cavity (mostly parietal pleura) were removed for section.

"Report 27.11.47. T.47/1078.

The adhesions are composed of active proliferating fibrous tissue but show no specific structure of tuberculosis."

No.13. Arra W., aged 22 years. (M.& B.1270 case).

Larynx free. Right artificial pneumothorax 29.11.46. Right adhesiotomy 7.2.47. Right phrenic crush 5.5.47. Pneumoperitoneum 2.5.47.

Intercostal drain 16.7.47. Death after 20 g. M.& B.1270 intrapleurally.

Post-mortem 2.1.48. Well nourished female. No icterus visible. Intercostal drainage opening right side chest.

Right pleural cavity - empyema cavity present from apex to base. No free pus present. Visceral and parietal pleura were thickened, yellow and caseous looking. No sign of broncho-pleural fistula. No foul smell and the walls were "sweeter" than the usual empyema cavity.

Right lung - lung could not have expanded owing to firm crust of thickened, visceral pleura. Cavity at apex was compressed and there was evidence of healing

No.13. Arra W. (Continued):-

in the tuberculous foci in right upper lobe.

Lower lobe apparently healthy.

Left pleural cavity - no adhesions. No free fluid.

Left lung - small cavity at apex and mid. zone.

Several hard, pea-like, calcareous foci in lower lobe, some on the pleural surface (one for section).

Small areas of active pulmonary tuberculosis in left lung.

Heart - slight petechiae in atrial walls. Some icterus of endocardium.

Liver - uniformly yellow. Liver not shrunken. Acute yellow atrophy (specimens for section).

Pancreas - N.A.D.

Spleen - not enlarged. No unusual colour.

Stomach - dark blood in lumen. Petechial haemorrhages in submucosa.

Small and large intestines - putty coloured stools in lumen. Slight petechiae in submucosa.

Kidneys - pale, slight suggestion of icteric tint.

Small haemorrhage right renal pelvis.

Suprarenal bodies, ureters and bladder - N.A.D.

Bone marrow - hyperplastic reaction.

Brain - removed and fixed for examination of fifth nerve nucleus later.

Remarks: No evidence of macroscopic bronchitis in trachea or bronchi. Sections of both right and left main bronchi taken just distal to the carina.

Section of surface of right lung, i.e., pleura and wedge of lower lobe and lung tissue taken for histology.

Results:

Right bronchus - no tuberculous infiltration seen.

Left bronchus - no evidence of tuberculosis.

Liver - acute yellow atrophy. A few remnants of liver cells left. The main part of the liver had extensive fatty degeneration.

No.13. Arra W. (Continued):-

The section of empyema crust with wedge of healthy right lower lobe is interesting. It shows several zones.

Hosts of polymorphs and cellular debris in outer area.
Fibrinous material with groups of polymorphs and plasma cells.

Deepest layer is thickened visceral pleura. Layers of fibroblasts and new b.v.s. organised.

See page 112.

Then comes layer of compressed and organised lung. Does not look as if it would ever re-expand. It has clumps of dark pigment.

Below this lung alveoli are collapsed and walls thickened. In the few air spaces left are histiocytes crammed with the black pigment. Coming or going to pleura?)
? Significance.)

Then finally as we go towards hilum more healthy lung.

No evidence of tuberculosis here.

No.14. T.L., aged 55 years. Larynx free.

Ill for eight months with acute haemolytic anaemia and increased fragility of red cells. ? Acquired acholuric jaundice. Investigated at Manchester Royal Infirmary and sent to Baguley Sanatorium because of sputum positive for tubercle bacilli. Had weekly transfusions of packed red cells but succumbed from his anaemia on 3.1.48.

Post-mortem 4.1.48. Well nourished male. Jaundiced. Right pleural cavity was free except for one apical adhesion and some small apico-mediastinal adhesions. No free fluid. An artificial pneumothorax could have been induced.

Right lung had several hard apparently healed, fibrotic nodules in its upper lobe. The apical adhesion and a wedge of adjacent lung were removed for section. Left pleural cavity was adherent all over, especially at apex.

Left lung had an apical vomica the size of a pigeon's egg. The roof of this vomica in one part lacked

No.14. T.L. (Continued):-

pulmonary tissue and consisted of dense fibrous tissue adherent to the apical pleura.

Walls of cavity fibrotic and relatively free from necrotic tissue. Very little secretion in the vomica or in the bronchi of either lung. Two bronchi from left apical bronchus drained left apical vomica. No macroscopic bronchitis in them. No evidence of tracheitis, or bronchitis in either lung. Histological examination of the main bronchi showed no evidence of tuberculous bronchitis.

Several pea size, black lymph glands clustered in front of ascending aorta and at bifurcation of trachea.

Liver - not unduly large.

Spleen - enlarged and red.

Large intestine had advanced diverticulosis.

Bone marrow - hyperplastic.

The kidneys were interesting. Each had a double pelvis and a double ureter down to the bladder. Round each kidney transversely, in its mid.zone between the two pelves, was a furrow or groove. An aberrant left renal arterial branch ran into kidney substance on the anterior aspect of the organ.

Suprarenals - each was studded throughout its substance by irregular, calcareous nodules. The suprarenals were not destroyed by a caseous mass like one usually finds in Addison's disease.

No.15. M.P.

Admitted with post-cricoid carcinoma and large tumour mass right side neck. Also tuberculosis of each apex, sputum positive and tuberculous laryngitis.

Post-mortem 13.1.48.

Post-cricoid carcinoma ensheathing and invading the larynx and base of epiglottis. Laryngeal lumen narrowed and distorted. Tumour-like tissue projecting into lumen of larynx at base of epiglottis. (Previous biopsy

No.15. M.P. (Continued):-

confirmed that this was a carcinoma). Large, adherent tumour mass in right side of neck above clavicle, not apparently infiltrating lung apex.

Lungs emphysematous and oedematous in lower lobes. Adherent at each apex only but an artificial pneumothorax could have been induced. Each upper lobe was nodular to the feel.

Right upper lobe had a vomica the diameter of a halfpenny.

Left upper lobe had small vomica the size of an acorn.

All air passages full of pus.

Marked congestion and inflammation of the trachea and several small ulcers, and all main and secondary bronchi were the seat of bronchitis.

Section of left main bronchus just distal to carina removed for examination (i.e., healthier lung of the two) with adherent small gland.

This man's tuberculosis did not kill him directly. His partial laryngeal obstruction prevented him from coughing up all his sputum and this accounted for the large quantity of purulent sputum in the air passages.

No.16. Lily B., aged 38 years. Larynx case (treated with Nisin). Also diabetes mellitus and albuminuria + +.

A long-standing case - 4 years.

X-ray appearances and clinical signs showed extensive disease right upper and mid.zones.

Post-mortem January/1948. Oedema of legs and thighs.

A gland on anterior aspect of right main bronchus was the size of a walnut and hard and chalk-like. It was adherent to the outer coverings of the right main bronchus.

Right pleural space densely adherent all over. Lung could be freed only in the endothoracic fascial plain over lower and mid.zones but not in upper zone where lung was adherent densely to ribs and intercostal

No.16. Lily B. (Continued):-

tissues. Vomica in right upper zone was close to ribs. Specimen taken from this zone, including intercostal muscles and endothoracic fascia, fused on lung. No artificial pneumothorax could have been done here.

Right lung - large vomica upper lobe. Tuberculous areas in mid.zone and a few in lower zone. Emphysematous areas in lower lobe between the tuberculous foci. Lower lobes frothy and oedematous.

Left pleural cavity - no adhesions except at extreme apex. No free fluid. An artificial pneumothorax could easily have been induced.

(Endothoracic fascial plain stripped easily - specimen for section).

Left lung - emphysematous and frothy. Pea size calcareous area at extreme apex under adhesions. Bronchi:- macroscopic evidence of bronchitis in bronchi draining right upper lobe vomica and in distal part of right main bronchus. This could possibly have been seen with bronchoscope. The mucosa was roughened, oedematous and inflamed. No macroscopic evidence of bronchitis in left lung bronchi.

(Section of left main bronchus taken for section just distal to main carina - tuberculous bronchitis present).

The posterior wall of the trachea, three or four inches above carina, had two or three small ulcers similar to those in post-mortem case No.4.

No.17. Elizabeth H., aged 37 years.

Old-standing bilateral pulmonary tuberculosis. Larynx involved. Thin, wasted subject. Pressure sore on right hip.

Post-mortem 3.12.47. Bean-sized paratracheal and hilar glands - firm.

Right pleural cavity - free except at the apex. An artificial pneumothorax could have been induced.

No.17. Elizabeth H. (Continued):-

Right lung - apical vomica present. There were several draining bronchi and those had bronchitis in their distal parts only, close to the cavity. Swollen, oedematous mucosa visible.

Left pleural cavity - adherent in all aspects. The lung was removed in the extrapleural plane. Several small, shotty, yellowish calcareous nodules present in the extrapleural tissues over the lower lobe of the lung. (Section. "Report T.47/1134. This has the structure of an organising caseous tubercle').

Left lung - lung contracted. Trachea and heart pulled to the left. Extensive vomica of entire left apex. Left lower lobe was the seat of caseous infiltration. Some dilatation of left lower lobe bronchi.

Bronchitis in all the peripheral bronchi just beyond bronchoscopic vision. There was an ulcer which could be seen with the bronchoscope opposite the origin of the lower lobe bronchus.

Abdomen - the spleen was adherent to the liver. In the tissues between the lower surface of the left diaphragm and the left lobe of the liver were several pea size chalky nodules. No other abdominal lymphatic involvement seen.

Remarks: All the evidence here pointed to a peripheral spread of tuberculosis from the diseased left lung which was closely adherent to chest wall, i.e., spread to chest wall and to below the left diaphragm which is rare.

No.18. Agnes M., aged 18 years. Larynx involved.

Post-mortem 27.1.48. Chronic left tuberculous otitis media.

Several enlarged lymph glands present above the suprasternal notch.

Right pleural cavity - free apart from a few adhesions, thin apico-mediastinally and at one point on the

No.18. Agnes M. (Continued):-

lateral aspect of the upper lobe to the chest wall. An artificial pneumothorax could have been induced. No free fluid.

Right lung - firm, caseous, tuberculous nodules throughout the lung, especially in the upper lobe where a vomica was forming. Lung heavy. The firm, caseous subpleural areas on the free lung surface gave a marbled appearance over wide areas up to one or two inches square and at the very point on the anterior axillary line where an artificial pneumothorax needle would have been introduced. (Some of those nodules for section. Macroscopically there was no visible pleural reaction to this caseous infiltration.)

Left pleural cavity - densely adherent all over. The lung had to be removed extrapleurally. Even the endothoracic fascia had fibrosed and fused over the apex.

Left lung - soft, caseous, diseased areas in all zones. Several cavities in apex and left lower lobe. Lung heavy.

Pericardium - copious green effusion. Heart small and flabby.

Liver - enlarged to umbilicus. Amyloid disease.

Spleen - soft. Not enlarged.

Kidneys - small and soft.

Remarks: The trachea and all the bronchi were intensely congested and red and full of thin pus. (This even oozed from her left ear on manipulating the lungs). Many small, whitish ulcers, the size of a pin head were grouped together on the mucosa of the left main bronchus and lower trachea. (Sections of each bronchus were taken).

"Report. T.48/127. 12.2.48.

One of the sections of bronchus shows an early tuberculous proliferating lesion in the mucosa. Both

No.18. Agnes M. (Continued):-

sections of lung and pleura show extensive caseating tuberculous foci in the lung, extending almost to the pleural surface."

No.19. K.S., aged 25 years. Laryngeal case.

Long-standing left artificial pneumothorax with no fluid. Last X-ray 11.6.47, and he was too ill for further radiography. At that time left lung had expanded in its mid. portion only. No free fluid then.

Post-mortem 28.1.48. Thin, emaciated subject. Liver large and the seat of amyloid disease (section taken).

Large collection of pale green fluid in the abdomen. Spleen large and firm. Kidneys - N.A.D.

Right pleural cavity - no free fluid. Slender adhesions, friable, between the right upper lobe and chest wall. An artificial pneumothorax could have been done.

Right lung - shotty caseous nodules in lower and middle lobes which were the seat of broncho-pneumonia. Some of those nodules were sub-pleural in position. Fibro-caseous areas in upper lobe and a fairly extensive cavitary system.

Heart, trachea and mediastinum were well displaced to the left. Upper dome of left hemi-diaphragm raised.

Left pleural cavity - the lung surface was thinly adherent to the parietal pleura in most areas but was easily separated from it except at the extreme apex (wedge of parietal visceral pleura and lung taken - see report "A"). No free fluid and no evidence of any empyema.

Left lung - this was shrunken and of a dull slate-blue colour. The left upper lobe was extensively cavitated. Old-standing chronic, cavities with thick walls. There was a large vomica in the left lower lobe with necrotic walls and evil-smelling contents.

No.19. K.S. (Continued):-

The lung looked airless and carnified (surface wedge for section - see report "B").

(It looked as though the left lung had been approximated to the chest wall, not by its own expansion, but by expansion of the opposite lung coupled with a mediastinal shift to the left).

Trachea and bronchi - a large (marbled on section) mass of firm fibrocaseous glands was situated paratracheally. One gland hard and fibro-caseous, the size of a pigeon's egg was closely attached to the anterior wall of the left, lower lobe bronchus below its origin from left main bronchus. The pressure of this had narrowed the lumen of the left lower lobe bronchus but had not produced complete obstruction. No endo-bronchial ulceration. This gland could not from its nature have resolved, and therefore the compression was permanent and may, in fact, have been more severe before glandular contraction occurred. Slight bronchiectasis in left lower lobe distal bronchi. The mucosa of the trachea and main bronchi (within the limits of bronchoscopic vision) was injected and there were several pin-head ulcers present (therefore no section was necessary). The mucosa of all the left lower lobe bronchi below the obstruction was plum-coloured and deeply congested. Those bronchi draining right upper lobe and left upper lobe cavities were also intensely inflamed and the mucosa ulcerated.

"Report. T.48/48, 12.2.48.

Liver shows extensive amyloid degeneration."

Report "A". One portion of the lung shows active caseating tuberculosis.

Report "B". The other shows older healing foci with extensive fibrosis around the caseous areas.

No.20. B.M., aged 27 years.

Thin patient. Two healed sinuses in left chest wall at posterior axillary line.

Post-mortem 29.1.48. Enlarged paratracheal glands.

Right pleural cavity - copious clear pleural effusion. Some tags of plastic lymph on diaphragmatic surface of right lung. A few, friable apical adhesions.

Right lung - subpleural cavity with cross section the size of a penny on lateral aspect of right lower lobe. Visceral pleura over it was thin like tissue paper (? cause of recent pleural effusion). This vomica was of very fresh origin. The apex had scattered fibrosis and nodules and a vomica. X-ray before death did not reveal this vomica.

Left pleural cavity - parietal and visceral layers densely adherent in lower third of lung. (A) Empyema space above this was patent, (B) but narrow and had wide communication with two cavities (C) - one of them very large. The extrapleural space tissue was white and sclerotic at the site of the two healed chest wall sinuses. This endothoracic fascia was also organised at the extreme apex.

Left lung - lung surface slate blue. Lung widely cavitated in apex and mid.zones. The empyema lining was of a yellow material like thick, sodden cardboard and peeled off readily from the walls. It was obviously not organised.

Pericardium - some free fluid.

Liver - large and nutmeg.

Remarks: Trachea and main bronchi were injected and the mucosa swollen (much purulent material had been coughed up during life from her empyema cavity). The origin of left upper lobe bronchus was partially stenosed by a fibrous thickening in <u>whose centre was a cartilage ring.</u> This could have been seen by the bronchoscope. All peripheral bronchi were injected and inflamed.

No.20. B.M. (Continued:-

(Sections of stenosed bronchus and parietal and visceral left pleurae taken).

"Report T.48/130. 3.2.48.

Both pleura and bronchus show granulation tissue in which giant cells of tuberculous type are present."

(Slide of pleura shows the phenomena of non-organisation of peripheral layers).

No.21. J.J., aged 26 years. Laryngeal case.

Post-mortem: Body extremely emaciated.

Mediastinal lymphatics - several large, firm glands paratracheally and around left main bronchus.

Right pleural cavity - adhesions very dense at the periphery only, where outer wall of apical vomica was fused with chest wall.

Right lung - vomica at apex of lower lobe.

Extreme right upper lobe apical, large cavity.

Below this much fibrosis and hard lung tissue which was honeycombed.

Right lower lobe emphysematous.

Left pleural cavity - dense adhesions everywhere and even the endothoracic fascia was fibrous and obliterated, especially in left mid.zone.

Left lung - slate blue and atelectatic. Carnified and fleshy over large areas in lower lobe. Giant vomica left upper lobe and smaller one in left lower lobe.

Liver - amyloid.

Remarks: Trachea was dotted with small ulcers in its posterior wall as were the main bronchi. No marked redness of mucosa of trachea or main bronchi. There was marked redness and congestion of all small bronchi, beyond bronchoscopic vision, draining cavities.

N.B. Semi-stenosis of left main bronchus by a gland lying on inferior aspect of left main bronchus and elongated along it between bronchus and pericardium.

Bronchiectasis in left lower lobe (lining of bronch-

No.21. J.J. (Continued):-

iectatic cavities was roughened and septic looking).

Note. This girl had a left artificial pneumothorax on 7.5.46. Cavity in left upper lobe ballooned and many adhesions were seen. Some atelectasis of left upper lobe appeared and fluid formed (clear greenish and tubercle bacilli minus).

Thoracoscopy 20.8.46 showed indivisible adhesions. Lung semi-expanded and heart and mediastinum moved to the left. Fluid vanished by 12.2.47. Lung re-expanded from the bottom.

Study of the X-rays shows the definite narrowing of left upper lobe bronchus in series of films, although the gland is not visible, apart from rest of mediastinal structures.

(This is second example of collapse of whole lung and subsequent carnification from combination of (a) glandular pressure on a bronchus and (b) giant pulmonary cavities).

No.22. Mrs. L.W., aged 33 years.

Laryngeal case. Cough troublesome.

Admitted 4.3.48 with only seven months history of illness. Died 21.3.48.

Post-mortem 22.3.48. Large and firm right paratracheal glands and firm glands below right bronchus.

Right pleural cavity - no adhesions over lower lobe and no free fluid. Right lung adherent to chest wall from transverse fissure up over the apex. The adhesions were easily separated with the fingers and an artificial pneumothorax would have been possible here and on the left side.

Right lung - apical vomica with much surrounding caseation. Lower and middle lobes studded with small caseous nodules - many of them subpleural in position.

Left pleural cavity - no free fluid. Several thin, easily separated adhesions.

No.22. Mrs. L.W. (Continued):-

Left lung - caseous area in the apex which was broken down in the centre. As on right side the rest of the lung was peppered with small caseous nodules. The lingula was a free lobe. Pericardium had some greenish fluid. Examination of the bronchi showed generalised tracheo-bronchitis. The mucosa was plum-coloured and peppered with pin-head greyish ulcers. One large, deep ulcer, the size of a half-penny, was situated on the posterior wall of the trachea just above the main carina. Although deep it did not apparently communicate with any lymph gland in the post-tracheal wall.

No histological section was necessary here to confirm the diagnosis of tuberculous tracheo-bronchitis.

No.23. M.K., aged 32 years. Laryngeal case.

Illness characterised by intense dyspnoea and asthmatic attacks and terminal cyanosis for which O_2 was necessary.

A thin and wasted subject.

Post-mortem: Mediastinal lymphatics - large, firm, black glands in the paratracheal position and also in front of right main bronchus to which they were firmly adherent and below the left main bronchus.

Both lungs were removed per the endothoracic fascial plane.

Right pleural cavity - extensive adhesions with complete apical symphysis. No fluid.

Right lung - large apical vomica. Scattered caseo-pneumonic lung around this (portion removed for section at periphery). Right lower lobe and right middle lobe scattered with caseous nodules.

Left pleural cavity - adherent in all zones.

Left lung - large apical cavity and extensive firm caseous nodules throughout the entire lung.

Bronchi - trachea appeared normal and no ulcers were visible. Distal parts of both main bronchi, especially

No.23. M.K. (Continued):-

on inferior aspects and posterior walls, were inflamed but not ulcerated (specimen of left main bronchus for histology - early tuberculous bronchitis present).

All peripheral bronchi full of pus and injected, especially the bronchi draining the apical cavities. Left lower bronchi showed some caseous bronchitis in segments where they were involved in caseous areas. Note the association of asthmatic attacks and enlarged and adherent tracheo-bronchial glands.

No.24. A.P., aged 21 years. One month after right adhesion section a sudden spontaneous pneumothorax with clinical rupture of a cavity wall and empyema onset. Treatment by Nisin. Thin subject.

Large sinus right lower chest wall where several smaller sinuses had coalesced.

Right pleural cavity contained much evil-smelling pus. Right lung - collapsed in its three lobes. Carnified and bluish in all areas and extensively invaded with caseous tissue. This lung could not have re-expanded. One such area over a collapsed vomica had a fistula, the size of a lentil, into empyema space. Diaphragm, parietal and visceral pleura, covered with an evil, yellowish curd. (Wedge of carnified lung with visceral pleura for section).

Left pleural cavity - no fluid, free from adhesions except at apex.

Left lung - emphysematous and frothy lower lobes. Cavity and surrounding caseous disease at the apex. Lower lobe and lingula had scattered pea size caseous foci.

There was a spread of tuberculous nodules to the peritoneum and ligaments on the under surface of right hemi-diaphragm.

Kidneys and liver - N.A.D.

Spleen - soft, pale and large.

No.24. A.P. (Continued):-

Trachea and all main bronchi were merely congested from the copious volume of sputum expectorated daily and filling the air passages. (Histology showed that there was chronic bronchitis but no tuberculous bronchitis).

Post-mortem: No macroscopic ulceration.

Only the bronchus (apical) draining left upper lobe and outwith bronchoscopic vision was softened and ulcerated (specimen of this for section - involved with tuberculous bronchitis).

Several enlarged hilar glands but no demonstrable main bronchial occlusion.

(This right lung represents the end result of a vicious circle, i.e., ballooning right upper lobe vomica from kinking of a diseased draining bronchus. Tension of this obstructs diseased bronchioles and fine bronchi leading to collapse round main vomica and then rapid caseous pneumonitis of the area. Next step is spontaneous pneumothorax and empyema and collapse of entire lung, aided by devitalisation from hot cautery. Pneumonitis spreads - fibrin on visceral pleura forms a curd holding lung down. Chronic inflammation forms and carnification and lung cannot now expand and empyema is chronic).

No.25. M.P., aged 39 years. Tuberculous laryngitis case.

Post-mortem 3.5.48. Right pleural cavity - free apart from one thin apical adhesion.

Right lung - emphysematous. Small vomica middle lobe (area around it for section, i.e., where small bronchi possibly are).

Fibro-caseous areas right upper lobe.

Left pleural cavity - densely adherent all zones.

Heart pulled over to left.

Left lung - upper half of lung was the seat of one giant vomica. Left lower lobe fibro-caseous, dense and bluish.

No.25. M.P. (Continued):-

Liver - amyloid.

Kidneys - left had inflamed pelvis and several discreet small, chalky foci in cortex (one for section).

Trachea and bronchi were all intensely congested. Trachea had pin point small ulcers on posterior surface. Some bronchiectasis left lower lobe.

No.26. L.B., aged 17 years.

Post-mortem 21.5.48. Right sided empyema following spontaneous pneumothorax. Imminent sinus formation right chest.

Right pleural cavity - empyema present. Yellow, dry curded deposit of fibrin and pus on visceral and parietal pleurae. This firmly bound down the right lung. Very little pus.

Right lung - semi-collapsed and not adherent in any part; firm in texture but full of frothy material and not carnified, i.e., it could have re-aerated had it not been for the thickened visceral pleura. The only disease seen was a small area of caseous nodules below the pleura at the apex of the right lower lobe. Presumably the spontaneous pneumothorax occurred here, but no fistula was evident. (Specimen for section).

Left pleural cavity - free from adhesions. No fluid.

Left lung - no evidence of tuberculous disease.

A few moderately enlarged, tuberculous glands around right hilum.

Heart - blood-stained pericardial effusion and visceral and parietal pericardium coated with plastic lymph. (Specimen for section revealed tuberculous pericarditis present).

Abdomen - local plastic peritonitis below the right hemi-diaphragm glueing that muscle closely to the liver.

C.N.S. Tuberculous meningitis. Gelatinous material

No.26. L.B. (Continued):-

around the optic chiasma. No other evidence of miliary spread.

Trachea and bronchi showed no signs of macroscopic bronchitis and could be followed well into the lung substance. No histological evidence of tuberculous bronchitis in right main bronchus. (Specimen of right main bronchus for section).

No.27. F.H., aged 50 years.

Post-mortem 24.5.48.

A thin subject. Oedema of ankles and loins.

Mediastinal glands - small, black and firm glands paratracheal in position.

Right pleural cavity - adherent in all zones.

Endothoracic fascia was fibrous at the apex.

Right lung - apical vomica present. Small caseous areas of tuberculous broncho-pneumonia scattered in the upper half of the lung.

Left pleural cavity - adherent at the apex (limited adhesions). Also adherent over lingula and diaphragmatic surfaces. A limited artificial pneumothorax could have been induced.

Left lung - large lower lobe vomica. Caseous focus, size of a thrush's egg, at apex.

Kidneys - pale and fatty looking. Casts in renal pelvis. Organs not enlarged and capsule stripped easily.

Trachea and main bronchus had chronic bronchitis, and histology showed tuberculous involvement of the mucosae. Bronchiectasis and ulcerated mucosa in left lower lobe bronchi. Intense congestion and ulceration of draining bronchi of right upper lobe vomica.

No.28. J.W.C., aged 55 years.

Post-mortem 23.5.48.

A thin, emaciated subject.

Right pleural cavity extensively adherent throughout.

Right lung - large cavity with ragged walls occupying

No.28. J.W.C. (Continued):-

almost the whole of the right upper lobe. Rest of lung showed broncho-pneumonic consolidation.

Left pleural cavity - extensively adherent throughout.

Left lung - large cavity with ragged walls similar to right lung. Remainder of left lung shows extensive broncho-pneumonic consolidation.

Pericardium - four ounces of straw-coloured fluid in pericardium.

Trachea and bronchi showed marked purulent bronchitis. No gross ulceration.

Death was due to tuberculous broncho-pneumonia complicating chronic phthisis.