SPONTANEOUS PNEUMOTHORAX.

A Study of 100 Cases of the Benign or
Idiopathic type, with a review of the Literature.

by

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I. **INTRODUCTION.**

In September of the year 1819 Laennec published his immortal work on the Diseases of the Lungs and Heart, "De L'Auscultation Médiate", and in the Third Book there is a chapter entitled as translated by Forbes (1827) "Of Air in the Cavity of the Chest, or Pneumothorax." The introductory paragraph of this particular chapter reads as follows:

"Occasionally we find aeriform fluids in the cavity of the pleura. These are sometimes without smell, more commonly fetid, and of a fetor resembling that of sulphurated hydrogen gas. These fluids are sometimes in such quantity as very forcibly to compress the lung, and to distend the thoracic parietes in a very sensible manner .... Although this affection cannot be said to be of excessive rarity, it has hitherto been but little noticed by medical men."

This Thesis is a study of one hundred cases of Spontaneous Pneumothorax admitted to the Royal Infirmary in the period 1932 to 1950. I was given personal responsibility for seventeen of these cases.

My /
My interest in the subject of air in the pleural cavity, or pneumothorax, was first aroused as a House Physician in the Royal Infirmary, Edinburgh in 1944. A patient (Case 72) was admitted with a history of illness of three days' duration and, in spite of an apparently free airway, he appeared to be dying of suffocation. Though he lived for about twenty minutes after admission, he died before I was able to get the assistance of a more experienced Physician in dealing with the problem. His death was shown at autopsy to be due to a bilateral spontaneous pneumothorax with rupture of emphysematous bullae at the apex of each lung. To an inexperienced House Physician the bilateral resonant percussion notes and faint breath sounds, masked by stridor, in an unconscious patient, did not immediately suggest the diagnosis, but since that time the experience so gained has saved more than one patient from laparotomy, who had been wrongly diagnosed as an acute abdominal emergency.

The opportunity came my way of observing more of these cases of spontaneous pneumothorax and, on being appointed Clinical Tutor in 1946, after my release from Military Service, to the Wards of the Royal Infirmary, Edinburgh, under the charge of Dr. A. Fergus Hewat, I began to collect my notes on cases of Pneumothorax /
Pneumothorax that I had seen, and to study and compare them. I extended the scope of my investigations further, and through the kindness of the Physicians to the Royal Infirmary, I was allowed to study and to follow up examples of this condition in subjects who had been in their Wards as patients.

While the earlier work of this Thesis was in progress, an Editorial leading article was published in the British Medical Journal (1948) in which it was stated:

"The mechanism responsible for simple spontaneous pneumothorax .......... has not been thoroughly investigated though isolated cases showing cystic, and bullous emphysematous changes of various sorts have been reported, and valvular mechanisms demonstrated histologically."

With this encouragement I was stimulated in a task which like most researches entailed a large amount of work that was often tedious and was not without its periods of disappointment and frustration.

No series of cases comparable in number with my material has been described from Scotland before, and only one, (Perry 1939) in England. In the Continental and American literature there are a few series of over fifty cases, and reference is made to these later in this Thesis.
HISTORICAL REFERENCES.

In the year 1803 Itard, in his Thesis, first used the term Pneumothorax to describe the presence of air in the pleural cavity. He reported three cases from his own experience and referred to a further two published by Bayle and Selle. In each case the diagnosis was made at autopsy and phthisis and pleurisy were also present (Laennec, 1819).

In earlier times it was recognised by surgeons and anatomists, that air was occasionally found in the cavity of the chest, often in association with fluid, and was encountered in "performing the operation of empyema" (Laennec 1819), and in the Sixteenth Century on opening the diaphragm, Vesalius (1543) observed the lungs of an animal collapse and was able to cause them to re-expand by a "pipe made from reed", inserted into the trachea through which he introduced air.

At a still earlier date Celsus (c 30 A.D.) described the collapse of the lungs which took place when the diaphragm was opened, and it has been stated that the operation was probably carried out on condemned criminals, as a method of execution by the Greeks /
Greeks in even earlier times." (Prinzmetal and Kountz 1935).

In 1819 Laennec described the clinical features of pneumothorax and its recognition in the living subject, and discussed the differential diagnosis of the underlying causes in the lung from his own wide experience in clinical and pathological studies of diseases of the chest. The last type which he described forms the subject of this Thesis, and Laennec's own description of it is translated thus by Forbes (1827) on page 494.

"Finally an aeriform fluid may be formed in the cavity of the chest, without there being any solution of continuity, any other effusion, or any perceptible change of structure whatever. I have often perceived the escape of an in-odorous gas, in opening the thorax, while there was no perceptible affection of the pleura. Sometimes indeed, this membrane appeared to be drier than natural; and I remember one case in which it was in some places almost as dry as parchment."
TYPES OF PNEUMOTHORAX.

The lungs in health are normally kept in close apposition to the chest wall by the negative pressure which exists in the intrapleural sac. (Heynsius, 1882; Aron, 1891; Christie, 1934). This pressure varies dynamically with respiration about a mean of 5 cm. of water less than that of the atmosphere. (Wirz, 1923; Rohrer, 1925; Christie, 1934; Christie and McIntosh, 1934; Pain, 1940), and is sometimes referred to as "Donderische Drück", after Donders (1853), who repeated an experiment which had been carried out earlier by Carson (1820). In this experiment it was shown that the intratracheal pressure with the post mortem lungs in the position of full inspiration was greater than that obtained when the lungs were in the collapsed state. In life, therefore, a negative pressure must exist to keep the lungs in apposition with the chest wall, and any condition which allows air to enter this potential space between the two layers of the pleura will make this pressure less negative, that is, raise it towards that of the atmosphere. The elastic lungs will then collapse towards the hilum.

There /
There are two main routes whereby air may enter the pleural space. They are:

(1) Perforations of the thoracic wall and parietal pleura, when air will then enter the space directly from without. Penetrating chest wounds are examples.

(2) Perforation of the visceral pleura, where connection with atmospheric air is then made through the bronchial airways direct, or indirectly through the mediastinum, (Macklin, 1937) in which the pressure is also negative. (Meltzer, 1892; Jehn and Nissen, 1927; Jessup, 1931; Prinzmetal and Kountz, 1935).

We can thus divide pneumothorax into the following main groups:

(1) Pneumothorax occurring as a result of trauma to the chest wall from without.

(2) Pneumothorax occurring spontaneously as a result of underlying disease in the chest.

In addition to these two groups there is a third variety which is now recognised, and this may be called:

(3) Pneumothorax occurring spontaneously in the apparently healthy person, or Benign Spontaneous Pneumothorax.

This /
This Thesis is based on a study of One Hundred Cases in this last category, in whom there was no clinical or radiological evidence of underlying disease of the lungs. The criteria required for admission to this group are amplified in a later section of the introduction to this Thesis.

**BENIGN SPONTANEOUS PNEUMOTHORAX.**

Spontaneous pneumothorax occurring in individuals who show no evidence of disease or Benign Pneumothorax was first given recognition as a clinical entity in this country by West (1884) who communicated a collection of twenty-one cases to the Clinical Society of London. West gives as the earliest recorded case in his series one which was published by McDowell (1856) in the Dublin Hospital Gazette, and includes in his collection the case recorded by Church (1876) in the Edinburgh Medical Journal. Hall (1887) added a case of his own and three others not included by West. In the course of my studies on this subject I came across another case recorded at the same time as McDowell's (1856) by Banks (1856) which is not included in either collection.

It /
It was not until the year 1932, however, that the first series of over fifty cases personally collected was published by Kjaergaard (1932) who recorded a series of fifty-one cases from hospitals in Denmark over a period of twenty years, and followed up their progress. Since that time numerous individual cases and smaller groups have been recorded in the literature until Perry (1939) recorded eighty-five cases seen at the London Hospital between the years 1924 and 1937, and was able to follow-up seventy-five of these patients for periods varying from two to fifteen years. More recent contributions to the literature on this subject have come from America, in many cases in special groups or sections of the population. Ornstein and Lercher (1942) and Hyde and Hyde (1948), record cases occurring in the general American population, Blackford (1939), in College Students, Schneider and Reissman (1945), in Recruits for the American Services, Heath (1946), in Flying Personnel, and Leach (1945), in the American Armed Forces. More recently, Rottenberg and Golden (1949) have published briefly a series of ninety-seven cases occurring in the general population in America, between 1930 and 1947.
One other notable contribution to the literature of a specialised nature should be mentioned at this point, that of Brock's (1948), seventy-one personal cases of chronic and recurrent pneumothorax communicated to the Thoracic Society in February 1948, with a description of the mechanism of production, in many instances observed in the living subject by direct visualisation of the lesion through the thoracoscope.

Kjaergaard (1932) has a comprehensive view of the literature up to that date in his monumental study on this subject. Perry (1939) brought forward some further original observations on Benign Spontaneous Pneumothorax and reviewed the writings on the subject up to 1938. No series of cases comparable with these has been published in Great Britain up to 1949.

DEFINITION.

Various names have been given to this type of pneumothorax in contributions to the literature on the subject, such as "BENIGN SPONTANEOUS PNEUMOTHORAX", "SIMPLE SPONTANEOUS PNEUMOTHORAX", "IDIOPATHIC PNEUMOTHORAX", "INNOCENT PNEUMOTHORAX", "PNEUMOTHORAX DE CONSCRIPTS", "PNEUMOTHORAX SIMPLEX", and SPONTANEOUS PNEUMOTHORAX in the APPARENTLY HEALTHY."
11.

Probably the best definition of "Spontaneous Pneumothorax in the Apparently Healthy", is that given by Kjaergaard (1932), who defines it thus:--

"The clinical entity 'Spontaneous Pneumothorax in the Apparently Healthy' covers every case of Spontaneous Pneumothorax that appears without any demonstrable cause in healthy persons, in whom tuberculosis cannot be demonstrated - neither by examination of the sputum, by auscultation, nor by X-ray examination - and in whom the lesion is not accompanied by fever or by pleural effusion. This category comprises also the cases in which there is a slight rise of temperature in the first week of illness and cases in which the X-ray examination reveals the presence of an insignificant exudate too small to be made out on auscultation."

In the present series, no patient has shown any evidence of active underlying lung disease except for radiological signs of "emphysema" and occasional "bronchitis", as recorded in the Radiologist's Report, and it is well recognised that the diagnosis of emphysema is often not confirmed at post-mortem examination (Cabot, 1931; Davidson, 1936; Raelsen, 1938; /
1938; Perry, 1939; Paine, 1940; Christie, 1944). One patient in my material had a history of having had tuberculosis six years before in the other lung, but no active disease was present at the time of onset of his pneumothorax, nor has there been any recrudescence of his tuberculosis in the four year follow-up period: both Kjaergaard (1932) and Perry (1939) include similar cases in their material.

Subjects who have suffered trauma, perforating wounds or fractures of the chest wall are excluded. Likewise those cases of pneumothorax complicating gross pathology in the chest, such as lung abscess, bronchiectasis, empyema, bronchial carcinoma, advanced pneumoconiosis, acute oesophagitis, carcinoma of the oesophagus, and mediastinal emphysema alone without a demonstrable pneumothorax, are not included.

The other largest single group - those in whom there was acute or chronic active tuberculosis - are also excluded since they fail to fulfil the criteria required by Kjaergaard's definition.

Case records exemplifying all these conditions I have mentioned have been examined in the course of this research but fall outwith the scope of this Thesis.

With /
With the improvement of radiological technique in the last two decades and the advances which have been made in the surgery of the thorax it is now much easier to recognise the presence of localised emphysematous bullae and blebs in the living subject, and since an inspection of the radiographs reproduced in Kjaergaard's (1932) monograph shows the presence of such bullae, I have included cases in which there was radiological evidence of one or other. Two cases are included in which blood was aspirated from the pleural cavity (Kjaergaard includes two similar cases) and two who were sufferers from asthma, but in all other respects the cases are unselected.

The material of this Thesis is founded on the records of one hundred patients seen at the Royal Infirmary, Edinburgh, from April 1932 to March 1950 and on a study of the literature that has been published on this subject within the last decade. With the exception of six patients seen by myself as out-patients, all have been admitted as in-patients to Hospital. Four patients were admitted to the Surgical side of the Hospital and two of these were later transferred to the Medical Wards.

I have been given personal responsibility for the care and management of seventeen of these patients including /
including the six out-patients mentioned earlier.

**COLLECTION OF CASE RECORDS.**

In the Royal Infirmary, Edinburgh, there is no central filing system for patients' case notes and records, and in many of the Wards there is no method of tracing cases by means of an "Index of Diseases." The case records have therefore been traced largely by reference to, and perusal of, Admission and Discharge Ledgers of the Ward concerned. I have been unable to trace the case records of at least six patients diagnosed as suffering from pneumothorax and admitted during the period covered by this study. It is of course possible that some of the patients so diagnosed may have suffered from some underlying pathology in the lung, but as no case notes could be found, it is manifestly impossible to be certain of this or otherwise. These patients are not included in my material. Furthermore, in the presence of a primary lung condition the case notes of the patient are usually indexed under the heading of the primary disease and it was impracticable to examine the record of every patient diagnosed as suffering from pulmonary tuberculosis,
tuberculosis, lung abscess, empyema, etc., to ascertain if a pneumothorax was also present. These figures would have been of some value for comparative purposes, but their value would probably be insufficient to justify the work entailed. Nevertheless, in those instances in which a pneumothorax featured as part of the condition and has been so recorded, for example, as a hydro-pneumothorax, pyopneumothorax, etc., the records have been examined for comparison with the clinical course of those patients suffering from the benign form of pneumothorax.

II. ANALYSIS OF CASES.

GENERAL INCIDENCES.

The number of patients admitted to the Royal Infirmary for the period 1932 to 1947 averages 20,423 per annum. The total number of patients admitted in the eighteen year period covered by this Thesis is approximately 360,000 and this number has been found to include 100 cases who were diagnosed as suffering from benign spontaneous pneumothorax. The incidence in my series is therefore 0.028 per cent. of hospital admissions. This figure includes the six patients seen by myself and not admitted to hospital, but this number is balanced by the six in-patients whose /
whose case records I was unable to trace and who are not included in my material. Other cases admitted to the Surgical Wards of the Infirmary and not later transferred to the Medical Side are also, of course not included in this figure, but these are not likely to be numerous. In most instances, where the patient has been admitted to a Surgical Ward, there is a preceding history of trauma, and these are, according to the definition given in an earlier section, not included.

Perry (1939) collected eighty-five cases seen at the London Hospital over a fourteen year period, during which time about 10,000 patients were admitted annually. The number of in-patients in his series is not stated, but according to Brock (1948) the figure of eighty-five included out-patients. The incidence of his series is therefore 0.06 per cent. of hospital admissions.

Heath (1946) in a survey of 28,000 admissions to an A.A.F. Regional Hospital found only ten cases, an incidence of only 0.036 per cent., which is unusually low for the age and sex group at risk, his cases being taken from a group of young healthy men of military age, in whom it will be shown later in this Thesis,
Thesis the incidence of benign spontaneous pneumothorax is highest, from the point of view of both sex and age. Indeed, Olbrecht's (1930) has designated this type of spontaneous pneumothorax, "Pneumothorax de Conscripts" because of its high incidence amongst recruits for military service.

Schneider and Reissman (1945), examining recruits for military service with the American Forces, found that about one in five hundred of the men examined gave a history - in many cases verified by reference to their family practitioner - of having had a spontaneous pneumothorax previously, an incidence rate of 0.2 per cent. Since their subjects were being conscripted for military service, however, it is not impossible that some at least of these might hope that a history of a previous pneumothorax might be sufficient to excuse them from service with the armed forces.

By way of contrast, King and Benson (1944), also studying military personnel in the American Forces, found only four cases in 5,000 admissions to an American Military Hospital, an incidence rate which is more than double Heath's (1946) figure, for a similar selected section of the population.

Blackford /
Blackford (1939) recording cases occurring in University students at Yale, found eleven cases in a five year period, where the average number of students was 2,500 per annum (0.089 per cent.), and Dickie (1948), examining students at the University of Wisconsin, discovered sixteen cases in 18,000 students between 1943 and 1947 (0.089 per cent.).

No accurate relationship between the incidence rate in these special groups and that in the general population can be drawn from these figures. Many cases occurring in the general population must fail to be diagnosed, especially where the pneumothorax is minimal and physical signs absent. Even if the lesion is recognised, the admission of the patient to hospital, in my series at any rate, is likely to depend on a variety of factors, such as the patient's willingness to come into hospital, particularly if the disability is slight, the availability of hospital beds if the disability is insufficient to require admission as an emergency, and, in this part of the country, the distance between the patient's home and the Infirmary.

Spontaneous pneumothorax has been said to be more common in private or general practice than in hospital work /
**TABLE "A".**

Incidance of Benign Spontaneous Pneumothorax in Recorded Series in Out-Patients and Hospital Admissions.

<table>
<thead>
<tr>
<th>Series</th>
<th>Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perry (1939)</td>
<td>0.06%</td>
</tr>
<tr>
<td>Blackford (1939)</td>
<td>0.089%</td>
</tr>
<tr>
<td>King and Benson (1944)</td>
<td>0.08%</td>
</tr>
<tr>
<td>Schneider and Reissman (1945)</td>
<td>0.2%</td>
</tr>
<tr>
<td>Heath (1946)</td>
<td>0.036%</td>
</tr>
<tr>
<td>Dickie (1948)</td>
<td>0.089%</td>
</tr>
<tr>
<td>Present Series</td>
<td>0.03%</td>
</tr>
</tbody>
</table>

(1) In- and Out-Patients in General population: per 100 admissions.

(2) College Students.

(3) Admissions to a Military Hospital.

(4) Recruits for American Forces. Previous history.

(5) College Students.

(6) In-Patients only from general population: per 100 admissions.
work (Legget, Myers and Levine, 1934; Ornstein and Lercher, 1942), and I believe that this is likely to be so. It is probably more common than is generally recognised and minor degrees of the condition, often with slight disability and few if any physical signs, must frequently be missed (Laennec, 1819; Kjaergaard, 1932; Perry, 1939).

It is difficult to draw any general conclusions from these figures for different series. All that can be said is that Perry's (1939) figure represents the incidence of the condition in hospital In- and Out-Patients per hundred hospital admissions. My own figure represents the incidence of patients admitted to hospital with benign spontaneous pneumothorax per hundred hospital admissions. I have found it impossible to get an accurate number for out-patients for comparison with Perry's figure.

The incidence rates for the different series quoted are shown in tabular form in Table "A".

RATIO OF BENIGN TO PATHOLOGICAL.

In the London Hospital Perry (1939) was able to find a total of one hundred and fourteen cases of pneumothorax. Thirteen of these were due to some gross lesion or trauma, etc., and sixteen occurred as a /
a complication of pulmonary tuberculosis. Of this latter group, nine patients or 56 per cent. died within a month; the remaining eighty-five cases occurred spontaneously in healthy persons. Legget, Myers and Levine (1934) recorded ten of their cases as being due to pulmonary tuberculosis. A further two were secondary to asthma or pulmonary fibrosis and nineteen occurred without demonstrable cause. Glickman and Schlomovitz (1936) reviewing eighty-two cases found that 38 per cent. were due to underlying pulmonary tuberculosis, 12.5 per cent. were apparently due to emphysema and 21 per cent. were of the "idiopathic" variety. They found that the benign form of pneumothorax showed the highest incidence after that due to pulmonary tuberculosis. Myers (1948) describing one hundred consecutive unselected cases of pneumothorax found sixty-four cases in whom underlying lung disease was known before or discovered in hospital. Of these sixty-four patients, thirty-eight had pulmonary tuberculosis or 36 per cent. of the total, a figure which accords exactly with that of Glickman and Schlomovitz (1936).

Most other recorded series deal with subjects in sanatoria or selected groups and cannot be satisfactorily used for comparison. Those patients who /
who are known to suffer from tuberculosis are usually under supervision in a Sanatorium or Tuberculosis Clinic and, as will be shown later in this Thesis, it is usually in the late stages of tuberculosis when the diagnosis has been established that spontaneous pneumothorax is likely to supervene. Such cases are not normally admitted to General Hospitals and the figures comparing the incidence of "benign" pneumothorax compared with that due to under-lying tuberculosis of the lung are likely to be lower for the tuberculous group than the non-tuberculous, in figures recorded from a General Hospital.

I have no comparable figures for the incidence of pneumothorax in cases where there was underlying disease of the lung such as tuberculosis. Many cases are registered in the Ward files in the Royal Infirmary under the heading of the primary disease only. For example, in twenty cases of empyema whose records I have studied, I found evidence recorded of the presence of air in the pleural space as well as pus, in six cases, but no record of this was included with the final diagnosis as recorded.

I am, therefore, unable to give any figures for the incidence of benign spontaneous pneumothorax in the/
the apparently healthy, compared with the incidence of spontaneous pneumothorax complicating disease of the lungs.

MORTALITY.

The mortality of spontaneous pneumothorax in the apparently healthy is not high, and this accounts for the small number of cases which have been recorded as coming to autopsy. Those patients who die, and on whom a post-mortem examination is carried out, either do not fall into the group under consideration on account of underlying disease of the lung, to which the pneumothorax is usually a fatal sequel, or else they have a super-added haemothorax or their pneumothorax is the uncommon simultaneous bilateral type. Since spontaneous pneumothorax occurs, as will be shown later, predominantly in young healthy individuals, when the initial period of shock has passed, the uninvolved lung is easily capable of dealing with the extra volume of blood diverted through it by the cutting down of the circulation through the affected side (Christie, 1934; Monaldi, Ferretti, and Constantine, 1938; Lindskog, 1939). There is, as a rule, some tachypnoea and increased ventilatory movement /
### TABLE "B".

Deaths in this Series after Admission to Hospital.

<table>
<thead>
<tr>
<th>Case</th>
<th>Age</th>
<th>Post-Mortem Summary</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>52</td>
<td>68</td>
<td>No P.M.</td>
<td>Previous history of Dyspepsia and &quot;heart disease.&quot;</td>
</tr>
<tr>
<td>58</td>
<td>42</td>
<td>No P.M.</td>
<td>Asthma set 18 yrs. Duodenal ulcer 20 yrs. Opened as perforated.</td>
</tr>
<tr>
<td>64</td>
<td>30</td>
<td>No P.M.</td>
<td>Cough a few months. Died suddenly.</td>
</tr>
<tr>
<td>72</td>
<td>39</td>
<td>Apical emphysema. Bilateral. 2 bullae ruptured. D.U.</td>
<td>Died less than half an hour after admission.</td>
</tr>
<tr>
<td>90</td>
<td>45</td>
<td>Emphysematous bullae. Enlarged R. heart.</td>
<td>Asthma and bronchi-tis since 1914 War.</td>
</tr>
</tbody>
</table>
movement of the unaffected side of the chest (Christie, 1936), but this seldom persists as long as 24 hours, even when there has been a complete collapse of the lung on the one side, provided the pneumothorax is not of the valvular or tension type.

Seven of the patients in this series have died after admission to hospital and their ages are given in Table "B".

Case 64 - aged 30 years, the youngest of those who died was an extremely unusual case and will be the subject of communication to the literature on spontaneous pneumothorax (Buchanan, 1949, personal communication). After settling down comfortably in the Ward an hour or two after admission, he turned over quietly in bed and died immediately. Permission unfortunately was not obtained for an autopsy.

Case 72 - a man aged 39 years - was seen by myself as a House Physician. He died less than half-an-hour after admission from a bilateral spontaneous pneumothorax. In his case autopsy showed the presence of an active duodenal ulcer (which was possibly at least partly responsible for the vomiting and hiccoughing which he had had for two days prior to admission), and his bilateral pneumothorax /
pneumothorax was shown to be due to the rupture of emphysematous bullae at the apex of each lung, the emphysema being largely confined to this region. Kjaergaard (1932) records a similar type of case whose pneumothorax dated from an attack of hiccupping and vomiting.

Case 90 - was a man aged 45 years who had had asthma for twenty-five years, and "acute bronchitis" for one month. He died two days after admission, and autopsy showed generalised emphysema with many large bullae, one of which had burst and caused the pneumothorax. The association of bronchial asthma and pneumothorax is rare, and Perry (1939) could only find five cases recorded in literature in the years 1877-1937. Further reference to the role of asthma in spontaneous pneumothorax is made later in this Thesis in the section on emphysema, with a review of recent literature. There are probably less than a dozen cases on record (Castex and Mazzei, 1938; Faulkner and Wagner, 1937).

Case 55 - a man aged 54 years - had symptoms of pneumothorax for three weeks, breathlessness on exertion for three years, and a winter cough for ten years. Autopsy showed generalised emphysema, though /
though no cause was demonstrated for the pneumothorax. Death in this case was probably due to a combination of anoxaemia and acute right heart failure due to his emphysema.

Case 93 - a man aged 42 years - was admitted almost in extremis. His record is admitted to the series as a borderline case, since he was found at autopsy to have a terminal broncho-pneumonia, with a history of tracheo-bronchitis of some days' duration prior to admission. Since there were no marked respiratory symptoms other than this, and since the naked eye appearance of the lungs did not show this terminal pneumonia to be enough to prove fatal in a normal individual of his age, without the super-added pneumothorax, I have included this case in my series. Furthermore, I had the opportunity of examining the lungs myself in the Post-Mortem Room, and was able to take sections and have them photographed. These are reproduced in a later section of this Thesis where the full post-mortem reports of those cases are recorded.

Mention should be made at this point of another case (Case 100), which is included in this series. He was initially admitted to the Royal Infirmary in 1943 /
1943, with a spontaneous pneumothorax, but I have been unable to trace his complete records, though I have been able to obtain the Radiologist's reports, and have examined some of the films of his chest that were taken at that time. From such records as I have, there is no doubt that he was suffering from a spontaneous pneumothorax, and that he also had evidence of emphysema of the lungs. In 1950 he was referred to me from the Medical Out-Patient Department because of my known interest in spontaneous pneumothorax. Through the kindness of Dr. James K. Slater, he was admitted to Ward 31 and I was given clinical responsibility for him. He was extremely ill on admission, cyanosed and with marked venous engorgement, including his retinal veins, though there was no papilloedema present. He had a persistent cough with an extremely viscid and tenacious sputum which he had great difficulty in getting up, and this indeed was a striking clinical feature in his case, and resembled the picture of status asthmaticus. After treatment with oxygen, aminophylline, adrenaline and sedations, his condition improved slightly, and, although consideration was given to the possibility of bronchoscopy with a view to removing some of the sticky mucus, he was too unco-operative, his pulse was too weak, and it was also thought /
thought unlikely that the associated spasm of the bronchioles would be improved much by this means. Faulkner and Wagner (1937) have described a similar type of case where they observed through the bronchoscope the walls of the trachea and the bronchi closing down completely with each spasm of coughing, and where removal of the mucus produced only slight temporary benefit. I have observed a similar type of case where rupture of caseous and calcified glands into a bronchus, too small to be reached by bronchoscopy produced a similar severe status asthmaticus, due to these broncholiths. With the measures which were instituted in my patient slight temporary benefit occurred, but he gradually became delirious and noisy, then - due in part to the sedation and in part to exhaustion - lapsed into a semi-conscious state from which he failed to recover. At autopsy he was shown to have marked emphysema affecting the upper lobes of the lungs, numerous adhesions on each side between the visceral and parietal layers, and a degree of broncho-pneumonia, more obvious on microscopic than on naked eye examination.

Fuller details of post-mortem reports and photographic illustrations are reproduced in the section /
section of this Thesis devoted to Autopsy Records.

In each of these cases recorded it will be noted that the patient was in the older age groups, apart from the first mentioned Case 64, and Case 72 (bilateral).

The remaining two cases who died were females, each over 40 years of age.

Case 58 - a housewife aged 42 years - was admitted to a Surgical Ward of the Infirmary and underwent a laparotomy for a suspected perforated duodenal ulcer. She had a long history of duodenal ulcer, but at laparotomy no perforation could be found. She was later found to have a spontaneous pneumothorax and was transferred to the Medical side where she died. No post-mortem examination was carried out.

Case 52 - aged 68 years - was a female receptionist. She was admitted to the Surgical side of the hospital as an acute abdominal emergency. I was asked to see the patient, found she had a tension pneumothorax, and treated this by decompression under a water seal. Her condition improved temporarily but her pulse gradually became weaker in spite of stimulant general/measures, and blood-stained fluid mixed with air was being passed through the drainage tube terminally.
terminally. Unfortunately permission for a post-mortem examination was not granted in her case either, so that the cause of the pneumothorax was not ascertained.

These patients, as I have noted, were mainly in the higher age groups of 40 years and upwards, and the mortality in my material is higher than in other similar series. Perry (1939) records only one death in his subjects and that was due to a concurrent septicaemia unconnected with the pneumothorax. In other cases which have been reported in the literature, death has often been due to bilateral pneumothorax or haemopneumothorax, which will be discussed in the appropriate section later.

In addition to the strain on the right heart due to the pneumothorax, one other reason for acute congestive heart failure often unnoticed by clinicians, is the present of a concurrent pneumomediastinum, a condition which may be associated with the pneumothorax. Pneumomediastinum has been noted in children, where air travels upwards into the subcutaneous tissues of the neck and down into the abdomen retroperitoneally, but is easily missed if this subcutaneous emphysema - "Surgical Emphysema" - is /
is not also present (Cournand, Bryant and Richards, 1935; Rosenberg and Rosenberg, 1938; Gumbiner and Cutler, 1941; Fisher, 1941; Smith and Bowser, 1942; Field, 1943; Cobley, 1946; Yudkin, 1947; Dickie, 1948). Macklin (1937) noticed the increased embarrassment of the circulation through the pressure of air in the mediastinum, when this was produced by local over-inflation of the lung of a cat. He reproduces in his article, photographs of the great vessels in the neck being compressed by air bubbles, and points out that minor degrees of mediastinal emphysema must often be missed, when post-mortem examinations are carried out in the usual manner.

In a patient with a spontaneous pneumothorax not only is there a diminished cardiac output (Richards, Riley and Hiscock, 1932), but when the circulation is further embarrassed by a positive pressure in the mediastinum in which the pressure is normally less than atmospheric (Jehn and Nissen, 1927; Jessup, 1931), acute heart failure may be expected to develop rapidly. Jehn and Nissen (1927) produced this experimentally in dogs. These workers found that as long as the mediastinal pressure in their animals remained about atmospheric there were no symptoms, apart from an increase in the respiratory rate.
TABLE "C".

Immediate Mortality from Spontaneous Pneumothorax in the Present Series.

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Total Cases</th>
<th>Total Deaths</th>
<th>Mortality percentage of Group</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>10-20 yrs.</td>
<td>8</td>
<td>0</td>
<td>0%</td>
<td></td>
</tr>
<tr>
<td>20-30 yrs.</td>
<td>46</td>
<td>1</td>
<td>2.1%</td>
<td></td>
</tr>
<tr>
<td>30-40 yrs.</td>
<td>17</td>
<td>1</td>
<td>5.9%</td>
<td></td>
</tr>
<tr>
<td>40-50 yrs.</td>
<td>19</td>
<td>3</td>
<td>15.8%</td>
<td></td>
</tr>
<tr>
<td>50-60 yrs.</td>
<td>8</td>
<td>1</td>
<td>12.5%</td>
<td></td>
</tr>
<tr>
<td>Over 60 yrs.</td>
<td>2</td>
<td>1</td>
<td>50.0%</td>
<td></td>
</tr>
</tbody>
</table>
rate, and a slight rise in blood pressure. When more air was introduced into the mediastinum, the blood pressure fell, apnoea, dyspnoea and cyanosis appeared, the eyes protruded and air appeared in the subcutaneous tissues of the jugulum. Most animals died at this stage, but some survived, and they were able to show a string of air bubbles at post-mortem examination round the great vessels, compressing these. I have looked for evidence of air in the mediastinum in cases of emphysema which have come to post-mortem examination, and particularly in Case 100 in this series, where the clinical features of cyanosis, exophthalmos, and others noted by Jehn and Nissen (1927) in their animals, were prominent; but so far I have not seen any. It is probable that in most cases the mediastinal pressure is raised by direct pressure on the mediastinum itself from the distended emphysematous lungs when broncho-spasm prevents their relaxing in expiration, and pressure is brought to bear on them by the voluntary expiratory muscles.

The mortality in the different age groups in my material is shown in the table on the opposite page (Table "C").
SEX INCIDENCE.

There are six male and six female Medical Wards in the Royal Infirmary, Edinburgh. The number of cases of spontaneous pneumothorax requiring admission to Hospital can thus be used as a guide to the incidence of this condition according to sex, since equal opportunity of admission exists for both male and female patients. Out of the hundred cases of spontaneous pneumothorax studied in this Thesis ninety-five occurred in men and five in women. This ratio shows a higher incidence of males to females than is usually found in other similar series, the figure usually being something over five to one. All other workers are unanimous, however, in finding benign spontaneous pneumothorax much more common in men than in women.

Perry (1939) collected from literature the publications by West (1884), Galliard (1888), Enneking (1923), Olbrechts (1930), Kjaergaard (1932) and Legget, Myers and Levine (1934), which, with his own series give a total sex incidence of three hundred and one males to fifty-seven females, or 5.3 to 1. Other more recent publications show a similar
similar ratio. Hyde and Hyde (1948) and Myerson (1948) each had thirty males to six females, and Niehaus (1947), twenty males to four females. Brock (1948), however, in his series of chronic and recurrent cases had fifty-two male patients and nineteen female, in this special group.

It is difficult to account for the higher incidence of male to female patients in my material (20 to 1), though in Ornstein and Lercher's (1942) series there were fifty-six male patients and only two female, a ratio of 28 to 1, and Wilson (1937) found eleven cases all males, in students at Yale University. Kirshner's (1938) twenty-four cases were all exclusively male. The recent publication of Rottenberg and Golden (1949) consists of eighty-seven males and ten females.

Recent publications on spontaneous pneumothorax have in many cases been studies of selected groups of persons, such as men of military age, and are therefore not of value for comparative purposes in regard to sex incidence, but there is no doubt that the incidence is very much higher in men than in women. No very satisfactory explanation has been advanced for this, though it has been suggested that muscular
<table>
<thead>
<tr>
<th>Authors</th>
<th>Total Cases</th>
<th>Males</th>
<th>Females</th>
<th>Ratio M/F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Collected by Perry (1939)</td>
<td>358</td>
<td>301</td>
<td>57</td>
<td>5.3/1</td>
</tr>
<tr>
<td>Wilson (1937)</td>
<td>11</td>
<td>11</td>
<td>0</td>
<td>11/0</td>
</tr>
<tr>
<td>Kirshner (1938)</td>
<td>24</td>
<td>24</td>
<td>0</td>
<td>24/0</td>
</tr>
<tr>
<td>Ornstein and Lercher (1942)</td>
<td>58</td>
<td>56</td>
<td>2</td>
<td>28/1</td>
</tr>
<tr>
<td>Niehaus (1947)</td>
<td>24</td>
<td>20</td>
<td>4</td>
<td>5/1</td>
</tr>
<tr>
<td>Hyde and Hyde (1948)</td>
<td>38</td>
<td>30</td>
<td>8</td>
<td>3.8/1</td>
</tr>
<tr>
<td>Myerson (1948)</td>
<td>38</td>
<td>30</td>
<td>8</td>
<td>3.8/1</td>
</tr>
<tr>
<td>Rottenberg and Golden (1949)</td>
<td>97</td>
<td>87</td>
<td>10</td>
<td>8.7/1</td>
</tr>
<tr>
<td>Present Series</td>
<td>100</td>
<td>95</td>
<td>5</td>
<td>19/1</td>
</tr>
<tr>
<td>Total</td>
<td>748</td>
<td>654</td>
<td>94</td>
<td>7/1</td>
</tr>
</tbody>
</table>
muscular effort in robust adult males may play a part in this (Ornstein and Lercher 1942) by the forcing of air into the upper lobes of the lungs during severe exertion when the abdominal and expiratory muscles are contracted and the glottis closed. Further consideration of this aspect of spontaneous pneumothorax and a discussion of the possible mechanisms involved is, to avoid unnecessary repetition, deferred to the section of this Thesis where the Mechanism of Production of Pneumothorax is considered.

AGE INCIDENCE.

For some years now it has been recognised that benign spontaneous pneumothorax is commonest in the age group, twenty to forty years. If those cases collected by Perry from the literature up to 1938, his own cases, and comparable series recorded more recently by Niehaus (1947), Myerson (1948), and Hyde and Hyde (1948), in America are compared with the present series it can be seen that the incidence is indeed highest in the twenty to forty age group.

With the examination in recent years of many men prior to their call-up for service with the Armed Forces /
### TABLE "B".  
**Age Incidence of Spontaneous Pneumothorax in Recorded Series.**

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1-10 yrs.</td>
<td>(14)</td>
<td>(3)</td>
<td>(0)</td>
<td>(1)</td>
<td>(0)</td>
</tr>
<tr>
<td>10-20 yrs.</td>
<td>(51)</td>
<td>17.7%</td>
<td>13.4%</td>
<td>6.3%</td>
<td>20.0%</td>
</tr>
<tr>
<td>20-30 yrs.</td>
<td>(120)</td>
<td>41.7%</td>
<td>40.2%</td>
<td>47.6%</td>
<td>46.6%</td>
</tr>
<tr>
<td>30-40 yrs.</td>
<td>(57)</td>
<td>19.8%</td>
<td>19.6%</td>
<td>20.6%</td>
<td>14.3%</td>
</tr>
<tr>
<td>40-50 yrs.</td>
<td>(39)</td>
<td>13.5%</td>
<td>13.4%</td>
<td>15.9%</td>
<td>14.3%</td>
</tr>
<tr>
<td>50-60 yrs.</td>
<td>(16)</td>
<td>5.6%</td>
<td>8.5%</td>
<td>6.4%</td>
<td>2.8%</td>
</tr>
<tr>
<td>Over 60</td>
<td>(5)</td>
<td>1.7%</td>
<td>4.9%</td>
<td>5.2%</td>
<td>0.0%</td>
</tr>
<tr>
<td>Average Age</td>
<td>30.3 yrs.</td>
<td>33.0 yrs.</td>
<td>32.7 yrs.</td>
<td>26.1 yrs.</td>
<td>32.9 yrs.</td>
</tr>
</tbody>
</table>

**NOTE:** In the calculation of the percentage in each series, subjects under 10 years of age have been excluded.

Hyde and Hyde saw only patients aged 18 years and over.

The present series does not include any patient under 12 years of age. Younger patients are not usually admitted to the Royal Infirmary.
Forces it is not surprising that an increase of interest in this condition has been shown. In America particularly where a radiological examination of the chest was carried out routinely, Schneider and Reissman (1945), found as many as one in every five hundred recruits between the ages of 18 and 38 gave a verified history of a previous spontaneous pneumothorax, and these must be regarded as benign, for in only four cases was there any clinical or radiological evidence of a possible cause for its occurrence.

Table "E" gives the age incidence in the present series compared with those others mentioned. Niehaus (1947) states that the average age of his patients was 28 years and two months, and Rottenberg and Golden (1949) patients' ages averaged 30.2 years. Both these publications, and that of Cohen and Kinsman (1946) state that there were eight hundred and seventy-three hospital admissions among military personnel of the American Forces in 1945, on account of spontaneous pneumothorax. The average age of these men is likely to have been around 28 years, since those recruits examined by Schneider and Reissman were between the ages of 18 and 38.
TABLE "F".
Relative Incidence of Side Affected
In Different Series.

<table>
<thead>
<tr>
<th>Authors.</th>
<th>Right Side</th>
<th>Left Side</th>
<th>One, then the other.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Enneking (1923)</td>
<td>73</td>
<td>66</td>
<td>3</td>
</tr>
<tr>
<td>Olbrechts (1930)</td>
<td>4</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>Kjaergaard (1932)</td>
<td>33</td>
<td>17</td>
<td>1</td>
</tr>
<tr>
<td>Wilson (1937)</td>
<td>5</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>Kirshner (1938)</td>
<td>11</td>
<td>10</td>
<td>1</td>
</tr>
<tr>
<td>Perry (1939)</td>
<td>35</td>
<td>48</td>
<td>2</td>
</tr>
<tr>
<td>Ornstein and Lercher (1942)</td>
<td>25</td>
<td>16</td>
<td>0</td>
</tr>
<tr>
<td>Leach (1945)</td>
<td>76</td>
<td>50</td>
<td>1</td>
</tr>
<tr>
<td>Schneider and Reissman (1945)</td>
<td>55</td>
<td>44</td>
<td>1</td>
</tr>
<tr>
<td>Cohen and Kinsman (1946)</td>
<td>21</td>
<td>18</td>
<td>0</td>
</tr>
<tr>
<td>Niehaus (1947)</td>
<td>15</td>
<td>9</td>
<td>2</td>
</tr>
<tr>
<td>Hyde and Hyde (1948)</td>
<td>31</td>
<td>32</td>
<td>3</td>
</tr>
<tr>
<td>Myerson (1948)</td>
<td>18</td>
<td>18</td>
<td>0</td>
</tr>
<tr>
<td>Rottenberg and Golden (1949)</td>
<td>47</td>
<td>44</td>
<td>6</td>
</tr>
<tr>
<td>Present Series</td>
<td>45</td>
<td>52</td>
<td>3</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>417</strong></td>
<td><strong>394</strong></td>
<td><strong>17</strong></td>
</tr>
</tbody>
</table>
SIDE AFFECTED.

In previous contributions to the literature on benign spontaneous pneumothorax the incidence on the left side seems to be roughly about the same as on the right. Those authors whose subjects have been mostly affected on the right side include Enneking (1923), Kjaergaard (1932), Ornstein and Lercher (1942), Schneider and Reissman (1942), Cohen and Kinsman (1946), Niehaus (1947) and Rottenberg and Golden (1949). The left side has been the most commonly affected in the publications of Olbrechts (1930), Wilson (1937), Perry (1939), Dickie (1948), and in the present series. Both sides were equally, or almost equally, involved in the cases described by Kirshner (1938), Myerson (1948), and Hyde and Hyde (1948).

It would appear from those figures, which are given in tabular form on Table "F" that there is, if anything, a slightly higher incidence on the right side than on the left, but I do not think that this is any more than might happen by chance, though the extra lobe in the right lung may be a factor for consideration in this respect.

Special groups of cases have not been included, such /
such as those with concomitant pneumomediastinum, in which the published series (Lundie, 1891; Lister, 1928; Hamman, 1937; McGuire and Bean, 1939; Griffen, 1942; Dickie, 1948) have shown the pneumothorax to be on the left side in every instance, with the exception of the case of Schwarz et al. (1946), cited by Dickie, and the six cases mentioned in the footnote to her report which she encountered in her student subjects, after this article had been submitted for publication. If these are included the incidence of spontaneous pneumothorax is almost equal on either side. No other selection has been made in the different communications quoted, but series which dealt with less than ten subjects have not been cited.

III. FOLLOW UP OF PATIENTS.

METHOD OF FOLLOW UP.

Seven patients in this series died after their admission to the Infirmary. Out of the remaining 93 cases, 6 had their pneumothorax incident less than six months before the follow-up was completed, and are therefore not included in the follow-up figures. The fact that many of the patients were living in lodgings or temporary accommodation at the time of the onset of their pneumothorax, combined with the intervention of World /
**TABLE "G".**

Length of Follow-Up Period in the Present Series.

<table>
<thead>
<tr>
<th>Length of F.U.</th>
<th>No. of Cases</th>
<th>Percentage of Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Six months to 2 years.</td>
<td>21</td>
<td>27</td>
</tr>
<tr>
<td>2-5 years</td>
<td>20</td>
<td>26</td>
</tr>
<tr>
<td>5-10 years</td>
<td>18</td>
<td>23</td>
</tr>
<tr>
<td>10-15 years</td>
<td>15</td>
<td>19</td>
</tr>
<tr>
<td>Over 15 years</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>78</strong></td>
<td><strong>100</strong></td>
</tr>
</tbody>
</table>
World War II, has not made such a follow-up an easy matter, but I have been successful in tracing and following up 78 of the remaining 87 patients. Under these circumstances, a figure of 90 per cent. followed-up successfully compares favourably with Kjaergaard's (1932) figure of 94 per cent. and Parry's (1939) 88 per cent. of 51 and 85 cases respectively, and is higher than many of the more recent communications on the subject. The length of the follow-up period is shown in Table "G".

In the majority of cases, the follow-up was carried out by means of a letter addressed to the patient, enclosing a short questionnaire for completion, which asked about recurrences, development of tuberculosis, and any history of tuberculosis in near relatives. Other patients were followed-up by correspondence with the General Practitioner who had referred the patient to hospital originally, and others again by a personal call at the patient's home, or an interview and examination at the Royal Infirmary.

It was early found to be impracticable to have each patient return for a full clinical and radiological examination at the Royal Infirmary, however desirable this might be, because in many cases the patient /
patient was far removed from the area, and had suffered no disability since the original attack. Many patients had been actively engaged as combatants during the War, and even with the utmost willingness on their part, it was considered unjustifiable in the absence of any symptoms of disease, to put them to what was at the least an inconvenience, for an examination most likely to be completely negative. Several patients, however, wrote long and informative letters about their subsequent progress. One, writing from Canada, was able to give me the report on a recent radiological examination (Case 77). Another describes his experiences with the Paratroops during the War and gives negative reports on three recent chest X-ray examinations (Case 4). Still another was traced through various Sanatoria and Tuberculosis Clinics and eventually found at his present address, no tuberculosis ever having been proved to have been present, according to the Tuberculosis Officer. On the other hand, attempting to trace one patient through his family practitioner who had looked after him privately prior to the advent of the National Health Scheme, the reply was received from the doctor, that he was "anxious /
"anxious to trace him too, but for a different reason!"

MORTALITY ON FOLLOW-UP.

Four of the seventy-seven patients who have been followed-up were found to have died during this period.

Case 19 - had been killed in action in Burma during the War, nine years after his original pneumothorax.

Case 39 - a man aged 46, had his pneumothorax in 1944. He was found to have died 23 years later, from what appears to have been acute right heart failure, or a recurrence of his pneumothorax, according to the report his family practitioner sent me. On admission originally he gave a history of cough and attacks of breathlessness, and he had radiological evidence of emphysema at that time.

Case 69 - a man aged 30, had a left pneumothorax in 1938. He was admitted to the Western General Hospital, Edinburgh, later in the same year, with a perforated peptic ulcer from which he died. I have no record of any previous history of dyspepsia in this patient at the time of his pneumothorax incident, but I have had the opportunity of examining the post-mortem
FIGURE 1.

CASE 100. Photograph of the X-ray film of a patient who had a left spontaneous pneumothorax three years before. The film was taken two days before he died. Extensive calcification is present in the costal cartilages, and there is thickening of the left pleura, with hazy opacity in the left costo-phrenic angle. The heart shadow is long and narrow, with some prominence of the pulmonary conus.
post-mortem report from the Western General Hospital on the case, and this states he had "a rupture of the stomach at the upper part of the greater curvature, the contents having leaked into the abdomen in this area and given rise to a subphrenic abscess on the left side." He also had an abscess in the right temporal lobe due to a chronic otitis media. The lungs are only briefly reported, general toxic changes being noted, and some adhesions at the right apex which was densely fibrosed.

Case 100 - has already been mentioned. He had a spontaneous pneumothorax in 1943, and was re-admitted in March 1950, and died from acute congestive heart failure and a terminal bronchopneumonia. Details of the post-mortem findings are given later in this Thesis, along with photographs of the lungs and photomicrographs. On the opposite page photographs of the X-ray films of his chest taken three days before he died in 1950 are reproduced. The extensive calcification of the costal cartilages is a noteworthy feature, a condition which is frequently associated with emphysema. (Christie, 1944).
INCIDENCE OF TUBERCULOSIS.

Kjaergaard (1932) was able to follow-up forty-eight of his fifty-one cases from two to fifteen years. Perry (1939) collected from the literature four cases of Enneking's (1923) series followed-up for eighteen, sixteen, eleven and eleven years, and twelve cases of Biesenthal and Snyder (1932) followed-up for nine years. In his own series, as I have noted earlier, he followed-up seventy-five out of his eighty-five patients with benign spontaneous pneumothorax.

One of Kjaergaard's (1932) subjects developed tuberculosis three years after the original attack of spontaneous pneumothorax, but she had no evidence of tuberculosis at the time of her first attack. She had four recurrences of spontaneous pneumothorax on the same side during the follow-up period, but she remained clinically and radiologically free from any evidence of tuberculosis and in perfect health for three years, at which time she began to show signs of tuberculosis in the other lung. She died from a further recurrence of her pneumothorax on the original side, and at autopsy a large thin-walled vesicle was found.
found at the base of this lung which had ruptured. Kjaergaard (1932) argues very convincingly that the two lesions are independent and that the later development of tuberculosis was the result of contact with her consumptive relatives.

None of Perry's patients had developed tuberculosis during the follow-up period. He includes among his cases six in whom radiograms had shown a healed tuberculous focus, but these foci showed no signs of activity at the time or on follow-up. In a review of the literature up to 1939 he was only able to find records of six cases who had developed tuberculosis after a benign spontaneous pneumothorax.

Myerson (1948) following-up twenty-one out of his thirty-six patients with a benign spontaneous pneumothorax for periods varying from one and a half to twelve years found two had died of liver disease and that only one had developed tuberculosis. The history of this patient is very similar to that of Kjaergaard's patient, but no autopsy report is included in Myerson's communication. After four episodes of pneumothorax, between which his lung fields were clear, this patient began to show signs of pulmonary tuberculosis. During his first two attacks of pneumothorax he had a negative tuberculin test twice,
to a dilution of 1:100. He died after his seventh attack of pneumothorax, three and a half years later. Hamman (1916) recorded a similar case, whose skin tuberculin test was negative at the time of onset of his pneumothorax.

Morris (1934) according to Wilson (1937) followed up twenty-six cases of spontaneous pneumothorax for from three to eleven years, and found none who had developed tuberculosis.

Kirshner (1939) followed up eleven cases, of whom six gave negative tuberculin reactions, for more than two years, and found they had remained healthy, and showed no evidence of tuberculosis. Of the original twenty-four cases he reported (1938), one died of advanced tuberculosis and other two were also found to have tuberculosis at the time of their pneumothorax incident.

Blackford (1939) was successful in following up fourteen out of fifteen cases occurring in College students and found them alive and well from eight months to fifteen years later. Four of those patients were followed up for more than five years.

Ornstein /
Ornstein and Lercher (1942) following up fifty-eight cases of spontaneous pneumothorax seen by them as out-patients, found that three of these subsequently developed tuberculosis about two years after their initial attack. In the three cases of Ornstein's that did develop tuberculosis, it was noted that they had pleural adhesions at the time of their attack of pneumothorax, a finding on which these authors lay some emphasis.

Schneider and Reissman (1945) examining recruits for the American Forces, in a hundred unselected cases who gave a history of spontaneous pneumothorax (which in most cases they verified from the subject's family practitioner), from several months to several years before, found a definite lesion in the lung to account for this in only four instances, and in only one case was there active tuberculosis present. In that subject the lesion was in the opposite lung to the one affected by the spontaneous pneumothorax. Cohen and Kinsman (1946) reporting thirty-nine cases in Military Personnel found advanced tuberculosis in two cases and evidence of an old primary lesion but no activity in a further four.

Niehaus /
Niehaus (1947) followed up twenty-two out of twenty-four cases. One had died from what was diagnosed as a coronary thrombosis, and the other twenty-one had remained well over periods varying from one to twelve years, and a similar finding is recorded by Hyde and Hyde (1948), who were able to trace eighteen of their patients from one to eight years after the initial spontaneous pneumothorax, and found that all were in good health and had no evidence of tuberculosis.

In the special group of cases of chronic and recurrent pneumothorax described by Brock (1948), only one patient was found to have developed symptoms of tuberculosis five and a half years later, but at the time Brock made his communication to the Thoracic Society he had been unable to find positive evidence of the disease. Dickie (1948), reporting twenty cases of spontaneous pneumothorax and pneumomediastinum amongst University students, found that only four of these subjects had positive tuberculin reactions, and states that this ratio of one in five with positive reactions was the same as that in the entire student population in the University.
None of the patients in the present series has developed tuberculosis during the follow-up period and only one gives a history of having had the disease (Case 68). This case, a medical practitioner, who was one of the patients under my charge, had had a positive sputum six years before. He was treated during his career as an undergraduate with an artificial pneumothorax, and the lung had been allowed to expand a year before his spontaneous attack. This attack occurred on the side opposite to his original tuberculosis, and came on when he lifted his suitcase down from the rack in a railway carriage. His pneumothorax later became valvular in type and almost proved fatal, but was fortunately successfully treated. X-rays taken at the time of his spontaneous pneumothorax and later, after the lung had expanded, showed no evidence of tuberculosis on the affected side, and no difference from the films of that same side taken six years before. The apex of the other lung showed a healed focus with no sign of activity. During his stay in hospital his temperature was never raised, and his erythrocyte sedimentation rate remained normal at 3 mm. in the hour (Westergren). On follow-up, he was well four years later and in active general practice.
**TABLE "H".**

Incidence of Pulmonary Tuberculosis Following Spontaneous Pneumothorax.

<table>
<thead>
<tr>
<th>Series</th>
<th>Total Cases</th>
<th>Followed up</th>
<th>Developed T.B.</th>
<th>Length of F.U.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kjaergaard (1932)</td>
<td>51</td>
<td>48</td>
<td>1</td>
<td>2-10 yrs.</td>
</tr>
<tr>
<td>Biesenthal and Snyder (1932)</td>
<td>12</td>
<td>12</td>
<td>0</td>
<td>Up to 9 yrs.</td>
</tr>
<tr>
<td>Morris (1934)</td>
<td>26</td>
<td>26</td>
<td>0</td>
<td>3-11 yrs.</td>
</tr>
<tr>
<td>Kirshner (1938)</td>
<td>21</td>
<td>11</td>
<td>0</td>
<td>More than 2 yrs.</td>
</tr>
<tr>
<td>Blackford (1939)</td>
<td>15</td>
<td>14</td>
<td>0</td>
<td>1½-15 yrs.</td>
</tr>
<tr>
<td>Perry (1939)</td>
<td>85</td>
<td>75</td>
<td>0</td>
<td>2-15 yrs.</td>
</tr>
<tr>
<td>Ornstein and Lercher (1942)</td>
<td>58</td>
<td>58</td>
<td>3</td>
<td>Over 2 yrs.</td>
</tr>
<tr>
<td>Schneider and Reissman (1945)</td>
<td>100</td>
<td>100</td>
<td>1</td>
<td>Not recorded</td>
</tr>
<tr>
<td>Niehaus (1947)</td>
<td>24</td>
<td>22</td>
<td>0</td>
<td>1-12 yrs.</td>
</tr>
<tr>
<td>Hyde and Hyde (1948)</td>
<td>63</td>
<td>18</td>
<td>0</td>
<td>1-8 yrs.</td>
</tr>
<tr>
<td>Myerson (1948)</td>
<td>36</td>
<td>21</td>
<td>1</td>
<td>1½-12 yrs.</td>
</tr>
<tr>
<td>Present series (1949)</td>
<td>100</td>
<td>75</td>
<td>0</td>
<td>6 mths.-18 mths.</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>591</strong></td>
<td><strong>480</strong></td>
<td><strong>6</strong></td>
<td><strong>6 (1.3% of total followed up)</strong></td>
</tr>
</tbody>
</table>
practice in the Midlands of England, having had only one slight recurrence of his pneumothorax about six months after his first attack.

Until fairly recently, and probably up to the time that Kjaergaard (1932) emphasised that a spontaneous pneumothorax could occur in healthy persons, most cases in which no other obvious lesion was found were regarded as being tuberculous in origin. Since that time, however, it has become increasingly clear that tuberculosis only rarely develops subsequently in these cases, many of whom, as in the instances quoted earlier, have negative tuberculin tests. The incidence of the development of subsequent tuberculosis in the different series mentioned earlier in this section is set out in tabular form in Table "A".

It is common knowledge that a spontaneous pneumothorax is a not infrequent complication of advanced tuberculosis, and the earlier writers on pneumothorax attributed a much higher percentage of spontaneous pneumothorax, occurring without obvious cause, to pulmonary tuberculosis. Of nine hundred and eighteen cases collected by Biach (1880), 77.8 per cent. were said to be due to tuberculosis. Morse (1900) attributed 70 per cent. of his fifty-one cases to /
to tuberculosis, and Ayer (1910) 69 per cent. of his seventy-two cases. More recently, Legget, Myers and Levine (1934) found 32 per cent. of their thirty-one cases were due to tuberculosis and Glickman and Schomovitz (1936) found 38 per cent. of their eighty-two cases occurred in the presence of frank tuberculosis, and that the incidence of the "idiopathic" type of pneumothorax was next in frequency—21 per cent. in their series. Myerson (1948) in one hundred consecutive patients found tuberculosis in thirty-eight of these, which was either known to exist before or was discovered in hospital.

The usual explanation given by writers twenty years ago was that this apparently benign type of pneumothorax was due to the rupture of a small subpleural tuberculous focus, minute in size, and a spontaneous pneumothorax was regarded then, in the manner we nowadays still regard the "idiopathic" pleural effusion, as a manifestation of occult tuberculosis. Fishberg (1932) gave as his opinion, however, "probably 20 per cent. of cases of really spontaneous pneumothorax are not due to tuberculosis," but this opinion was not universally shared. Perry (1939), in his search of the literature, was unable to
TABLE "I".

Incidence of Spontaneous Pneumothorax occurring as a Complication of Tuberculosis in Unselected Cases.

<table>
<thead>
<tr>
<th>Authors</th>
<th>Total Cases</th>
<th>T.B. Cases</th>
<th>Percentage of Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Legget, Myers and Levine (1934)</td>
<td>31</td>
<td>10</td>
<td>32</td>
</tr>
<tr>
<td>Oechsli and Miles (1934) Review of Lit. on Bilateral SP.</td>
<td>76</td>
<td>37</td>
<td>49</td>
</tr>
<tr>
<td>Glickman and Schlamovitz (1936)</td>
<td>82</td>
<td>31</td>
<td>38</td>
</tr>
<tr>
<td>Perry (1939)</td>
<td>114</td>
<td>16</td>
<td>14</td>
</tr>
<tr>
<td>Myerson (1948)</td>
<td>100</td>
<td>38</td>
<td>38</td>
</tr>
</tbody>
</table>

* Perry's cases were seen in a General Hospital in London. Patients known to have tuberculosis or in close contact with the disease are likely to have been diverted to Tuberculosis Hospitals or Centres.
to find a single case where rupture of a small sub-
pleural tubercle could be shown to be the cause of
this type of pneumothorax, and I have only found one
(Birch, 1936) where a minute unruptured tuberculous
cavity was apparently demonstrated, with a roughening
of the overlying pleura, and this was found in a case
of haemopneumothorax. The bleeding point was found
nowhere in this area, nor was any point of entry of
air to the pleural sac demonstrated.

Mention has already been made of the ratio of
pneumothorax occurring as a complication of known or
simultaneously diagnosed tuberculosis, to other types
of pneumothorax in various series of unselected cases
in the literature (Legget et al. 1934; Glickman and
Schlomovitz, 1936; Perry, 1939; Myerson, 1948), and
the figure of less than 40 per cent. probably
represents a more accurate estimate nowadays of the
incidence of this type than the earlier ones of 70 to
80 per cent. (Table "T").

If rupture of a small tuberculous focus was the
cause of pneumothorax simplex, it is to be expected
that in a certain proportion of these cases there
would be some signs of pleural infection or reaction;
but there is no evidence that this often develops,
and it is indeed rare for more than a small amount of
fluid /
fluid to be present in the pleural space at any stage of the pneumothorax incident. It is to be expected that if these patients were indeed suffering from a minimal tuberculous lesion, that some of them at least would have developed frank tuberculosis during the follow-up period. In fact, none of the present series has developed subsequent tuberculosis, and the general incidence of subsequent tuberculosis, among persons who have had a spontaneous pneumothorax, can be seen from Table "H" (page 48) to be higher than that which might be expected among the general population.

It has been said (Cameron, 1950) that probably 80 per cent. of people in this country have been infected with tuberculosis by the age of twenty. Few of these individuals, however, develop frank pulmonary tuberculosis at a later date. In most cases where there has been pulmonary infection, a small area of scar tissue is left, around which emphysematous bullae may be formed. These may subsequently give rise to a spontaneous pneumothorax, but any other condition such as whooping-cough or broncho-pneumonia may similarly give rise to such bullae, and thus it is the healed tuberculous lesion and not one in a state of activity which is the cause /
cause of the pneumothorax. Rupture of such bullae has been shown to be the cause of the pneumothorax (Kjaergaard, 1932; Wilson, 1937; Cohen and Kinsman, 1946), where the tuberculous focus has been healed and walled off with fibrous tissue, and it is to be expected that a thin walled air vesicle would be much more liable to rupture than a comparatively thick walled tuberculous cavity. The only case in this series (Case 68), in whom a lesion of healed tuberculosis was demonstrated, had his pneumothorax on the unaffected side, and this has been noted in other similar series (Kjaergaard, 1932; Perry, 1939).

The clinical course of patients with benign spontaneous pneumothorax differs markedly from those with active pulmonary tuberculosis. Though there may be a slight rise of temperature in the first few days, this rapidly settles. Cough is slight or absent, and there is little if any sputum and repeated examinations of the sputum for tubercle bacilli are negative. There is no rise in the blood sedimentation rate (as will be shown later), and seldom in the leucocyte count, to suggest the presence of an infective process. I have compared nine case records of spontaneous pneumothorax occurring in the course of active /
active pulmonary tuberculosis with the present benign series. In each of these the tuberculosis was in an advanced stage and, like Perry (1939), I found that about half of these patients died within a month. Furthermore, in none of the cases in this present series on which a post-mortem examination was carried out, was any evidence found of active pulmonary tuberculosis.

One other feature about this relationship of tuberculosis to the benign form of pneumothorax, is the fact that this latter is about six times more common in the male sex than in females, which is, of course, not true of pulmonary tuberculosis. It also occurs in an earlier age group than those in whom spontaneous pneumothorax develops as a complication of phthisis. Myerson's (1948) average age was forty-four years for the phthisical patients and twenty-eight years for those in whom no cause was found for the incidence, which accords closely with the average age of the benign form recorded in Table "E" (page35).

I agree with Perry (1939), when he states categorically "--- active tuberculosis is certainly not a cause of this type of pneumothorax, as neither at the time of the attack, nor in subsequent years, can any evidence be found to suggest tuberculosis," and I believe that the clinical and radiological evidence /
evidence at the time of incident and the follow-up findings of the present series of cases fully substantiate this opinion.

RECURRENTS.

In the present series of cases, seventeen patients give a history of a previous attack of a similar character more than one month before. In a further thirteen there is a history of previous "pleurisy" or similar illness which might have been a previous attack not recognised as a pneumothorax at the time. In the follow-up period, fourteen patients have had definite recurrences of their pneumothorax and a further two have had symptoms suggestive of a recurrence. Out of those fourteen subjects with recurrences on follow-up, four give a history of a definite attack prior to the one for which they were admitted to hospital. Thus, twenty-seven out of a total of one hundred patients in my material have probably had more than one attack. The incidence in my material and number of attacks is shown in Table "J". Those patients who have previously had a spontaneous pneumothorax diagnosed are usually able to recognise a recurrence.

Nikolski /
Nikolski (1912), in a series of ninety cases, quotes nine incidences of recurrence and Wood (1931), states that out of seventy-one patients seen at the Mayo Clinic, 21 per cent. had multiple attacks and 11 per cent. had both a right and left pneumothorax alternately. Kjaergaard (1932), had seven instances of recurrence in his fifty-one cases. Perry (1939) has the surprisingly low figure of only 4.7 per cent. recurrences out of seventy-five cases followed up, and finds himself unable to explain a figure which is much below what had usually been recorded. 10 per cent. (four cases) in Cohen and Kinsman's (1946) material had a history of a previous attack.

According to Brock (1948), many of Perry's cases were collected from the Out-Patient Department of the London Hospital, and it is possible that minor degrees of pneumothorax not requiring hospital admission show less tendency to recurrence. Legget et al. (1934), remark that the condition is more common in private or "office" practice than in hospital work, so that if Perry (1939) included a large number of out-patients his cases might be considered as representative of the type of patient referred by the general practitioner for X-ray confirmation of the diagnosis. Leach (1945), in a short follow-up of up to two years, had seven recurrences out of one hundred and seven /
seven cases. Myerson (1948) records multiple occurrences in three out of twenty-one patients followed up, and states that in other series recurrences rates of 10 to 30 per cent. have been noted. Hyde and Hyde (1948) had a recurrence rate of 19 per cent. on a 1 to 8 year follow-up, and in one hundred unselected cases reported by Schneider and Reissman (1948), nineteen gave a history of multiple attacks.

With reference to Leggett, Myers and Levine's (1934) observation that the condition is more common in private or "office" practice, I believe this is likely to be true. The percentage of recurrences particularly when dealing with in-patients admitted to a large hospital like the Royal Infirmary, Edinburgh, which serves a very wide area is probably also higher on that account. The family practitioner may well treat the patient at home during his first attack, but will want reassurance of a radiological examination of the chest to exclude underlying lung disease, in the event of a recurrence. Moreover, when a patient has been admitted to hospital as in my material, a more accurate, or at least a more detailed history is usually taken with regard to a previous similar occurrence, than in the case of the out-patient who has been referred by his doctor on account of pain in the chest, or some dyspnoea, and in whom the physical signs /
signs of pneumothorax are minimal or absent on clinical examination, and discovered only radiologically. Spontaneous pneumothorax is not always at the forefront of the physician's mind when a patient is seen for the first time complaining of pain in the chest, and it is not always possible to re-examine these patients after a film of the chest has been taken, many of them having come a long distance to the hospital.

The necessity of a long term follow-up is of some importance in estimating the recurrence rate. Kjaergaard (1932), in his own seven recurrent cases and a further seventeen collected from the literature, found that fifteen of these twenty-four had a recurrence within a year, and from the figures he gives the average number of incidents is three.

Locke's (1929) case is not cited by Kjaergaard, where eighteen attacks occurred over a period of seven years, both sides being affected at different times. Perry (1939) found in his survey of the literature that the recurrence rate was highest in the first year, yet he also found several cases in which a relapse occurred after five years. Brock (1948), in his seventy-one chronic and recurrent cases, found forty-two of these were recurrent and that some of these /
# TABLE "J".
Cases with multiple attacks, previous and on follow-up, in this series.

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Side Affected</th>
<th>Previous Incidents</th>
<th>Follow-up Incidents</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>L</td>
<td>2 L.</td>
<td>0</td>
<td>Previous haemo PT.</td>
</tr>
<tr>
<td>3</td>
<td>L</td>
<td>0</td>
<td>2 L.</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>R</td>
<td>1 L.</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>R</td>
<td>? 1</td>
<td>1 L.</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>L</td>
<td>0</td>
<td>? 2</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>R</td>
<td>2 R.</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>L</td>
<td>Many</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>R</td>
<td>1</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>L</td>
<td>2</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>L</td>
<td>1</td>
<td>N.F.U.</td>
<td>Untraced.</td>
</tr>
<tr>
<td>15</td>
<td>R</td>
<td>0</td>
<td>1 R.</td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>R</td>
<td>0</td>
<td>1 R.</td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>L</td>
<td>2</td>
<td>? 1</td>
<td></td>
</tr>
<tr>
<td>22</td>
<td>R</td>
<td>? 2</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>24</td>
<td>L</td>
<td>1 L.</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>25</td>
<td>R</td>
<td>2</td>
<td>1 R.</td>
<td></td>
</tr>
<tr>
<td>35</td>
<td>L</td>
<td>? 1</td>
<td>N.F.U.</td>
<td>2 attacks &quot;pleurisy.&quot;</td>
</tr>
<tr>
<td>38</td>
<td>L</td>
<td>? 2</td>
<td>0</td>
<td>Side forgotten.</td>
</tr>
<tr>
<td>44</td>
<td>L</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>45</td>
<td>R</td>
<td>? 1</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>46</td>
<td>L</td>
<td>? 1</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>50</td>
<td>L</td>
<td>0</td>
<td>1 R.</td>
<td></td>
</tr>
<tr>
<td>51</td>
<td>R</td>
<td>0</td>
<td>1 R.</td>
<td></td>
</tr>
<tr>
<td>52</td>
<td>R</td>
<td>? 1</td>
<td>0</td>
<td>Died.</td>
</tr>
<tr>
<td>53</td>
<td>L</td>
<td>? 1</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>66</td>
<td>R</td>
<td>1</td>
<td>0</td>
<td>2-3 attacks &quot;pleurisy.&quot;</td>
</tr>
<tr>
<td>68</td>
<td>R</td>
<td>0</td>
<td>1 R.</td>
<td>1 attack of &quot;pleurisy.&quot;</td>
</tr>
<tr>
<td>71</td>
<td>L</td>
<td>? 3</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>74</td>
<td>L</td>
<td>1</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>76</td>
<td>R</td>
<td>? 1</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>81</td>
<td>R</td>
<td>? 1</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>80</td>
<td>L</td>
<td>1</td>
<td>1 L.</td>
<td></td>
</tr>
<tr>
<td>85</td>
<td>L</td>
<td>1</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>86</td>
<td>R</td>
<td>0</td>
<td>1 L.</td>
<td></td>
</tr>
<tr>
<td>89</td>
<td>L</td>
<td>? 7</td>
<td>1 L.</td>
<td>7 attacks &quot;pleurisy.&quot;</td>
</tr>
<tr>
<td>98</td>
<td>L. &amp; R.</td>
<td>4 R. &amp; 5 L.</td>
<td>N.F.U.</td>
<td></td>
</tr>
</tbody>
</table>
these eventually became chronic. The average number of incidents in Brock's series was four, and the highest numbers fifteen, eleven and ten. Schneider and Reissman (1948) give the time for relapse in their series as one year in seven cases, two years in three cases, three years in one case, four years in two cases, and one recurrence was found after five years and one after nine. These workers estimate that the chances of recurrence are about 1 in 10 after two years. The most recent comparable publication, that of Rottenberg and Golden (1949), who record the findings in ninety-seven cases collected between 1930 and 1947, shows a recurrence rate very similar to that in my series, of 24.8 per cent. In sixteen cases the recurrence took place within one year of the initial episode, but five subjects had a recurrence after an interval of five years or more.

At least six patients in this present series have a definite history of attacks separated by an interval of five or more years, and one patient (Case 10) gives a history of having had several over a fifteen year period but had none in the two and a half years' follow-up period. This subject was an expert in physical training, but being now aged about thirty-seven years is possibly less active in this respect than formerly. The highest number of incidents /
**TABLE "K".**  
Reurrences of Spontaneous Pneumothorax in Recorded Series.

<table>
<thead>
<tr>
<th>Authors</th>
<th>Total Cases</th>
<th>Recurrences</th>
<th>Percentage of Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nikolski (1912)</td>
<td>90</td>
<td>9</td>
<td>10</td>
</tr>
<tr>
<td>Wood (1931)</td>
<td>71</td>
<td>-</td>
<td>21</td>
</tr>
<tr>
<td>Kjaergaard (1932)</td>
<td>51</td>
<td>6</td>
<td>11.8</td>
</tr>
<tr>
<td></td>
<td>48</td>
<td>8</td>
<td>16.6</td>
</tr>
<tr>
<td></td>
<td>99</td>
<td>14</td>
<td>14.1</td>
</tr>
<tr>
<td>Oechsli &amp; Miles (1934)</td>
<td>76</td>
<td>7</td>
<td>9.2</td>
</tr>
<tr>
<td>Perry (1939)</td>
<td>85</td>
<td>4</td>
<td>4.9</td>
</tr>
<tr>
<td>Leach (1945)</td>
<td>107</td>
<td>7</td>
<td>6.5</td>
</tr>
<tr>
<td>Cohen &amp; Kinsman (1946)</td>
<td>39</td>
<td>4</td>
<td>10.3</td>
</tr>
<tr>
<td>Niehaus (1947)</td>
<td>24</td>
<td>9</td>
<td>27</td>
</tr>
<tr>
<td>Myerson (1948)</td>
<td>21</td>
<td>3</td>
<td>14.3</td>
</tr>
<tr>
<td>Hyde &amp; Hyde (1948)</td>
<td>63</td>
<td>-</td>
<td>19.0</td>
</tr>
<tr>
<td>Schneider and Reissman (1948)</td>
<td>100</td>
<td>19</td>
<td>19.0</td>
</tr>
<tr>
<td>Rottenberg &amp; Golden (1949)</td>
<td>97</td>
<td>24</td>
<td>24.8</td>
</tr>
<tr>
<td>Present) x Previous Series</td>
<td>100</td>
<td>17</td>
<td>17.0</td>
</tr>
<tr>
<td>(xx On F.U. (1949)</td>
<td>78</td>
<td>14</td>
<td>18.0</td>
</tr>
<tr>
<td>)xxx Total</td>
<td>178</td>
<td>27</td>
<td>27</td>
</tr>
</tbody>
</table>

x These patients had a history of a previous attack.
xx These patients had a history of a further attack or attacks on follow-up.
xxx Includes 4 patients only once, who had a previous history of similar incidents (not including pleurisy) and were known to have had recurrence on follow-up.
xxxx Includes patients with probable previous attacks.
incidents in my material is ten (Case 98) and occurred in a patient with radiological evidence of bullous emphysema over a four-year period. He was not traced on follow-up. It would appear, therefore, that recurrence may take place at any time, but is most likely to happen within a year, the risk thereafter diminishing.

Five cases in the present series have either a definite history of a previous attack on the contra-lateral side, or were found to have had a recurrence on the contra-lateral side on follow-up, and Oechsli and Miles (1934), in their survey of seventy-six bilateral cases, found seven with recurrences and stated that recurrence was more common in benign pneumothorax than in other types. Further observations on bilateral and alternating pneumothorax are made later in this Thesis.

The recurrence rates in different publications is given in Table "K" on the opposite page. It should be noted, however, that in some of these the history has been recorded of a previous attack, and in others that the recurrence has been noted on follow-up. Where this has been possible, such as in Kjaergaard's communication, the author's case records have been examined and the previous incidents have been /
been noted. In others this has not been possible, since no case records have been published, and only a figure giving a "percentage of recurrent cases" has been record. In my material, any case with a previous history of "pleurisy" otherwise undetailed in description, has not been included under the heading of "Previous Attacks", and I am therefore of the opinion that the incidence of recurrences is at least as high as a figure of between 15 and 20 per cent. according to the findings in my own series and those taken from the literature on the subject.

**FAMILIAL PNEUMOTHORAX.**

In discussions on the aetiology of spontaneous pneumothorax, occasional reference is made to the occurrence of this in more than one member of a family. Young (1936) records two cases in members of the same family, in which the pneumothorax came on while the patients were straining at stool. Others have also been recorded where members of the same and different generations of the same family have been affected. (Atwood 1926, Willis 1937, Bachman 1940, and six others cited by Macklin and Macklin 1944).

In my earlier studies of case histories in my material, I thought that I had two examples of this familial incidence. On follow-up, however, I found that /
**FIGURE 2.**

**X-RAY OF CHEST AFTER PNEUMONECTOMY**

*Case 66.* Photograph of X-ray film of chest of a patient, over six years after a pneumonectomy for a chronic pneumothorax. There is a complete acquired dextrocardia, with deviation of the trachea, and herniation of the mediastinum.
that they were brother and sister, the sister having been recorded under a different name because of her marriage previous to the pneumothorax incident (Cases 42 and 66).

Case 42 - who was a man aged 25 at the time of his pneumothorax has been followed up for almost eighteen years and has had no recurrences, and he has also a second cousin on his mother's side with a history of a spontaneous pneumothorax. I have tried, but have been unable, to trace this cousin. His sister (Case 66) was a woman aged 28 years at the time of her pneumothorax. The lung failed to expand and she eventually required a pneumonectomy. She had lost over two stones in weight during the time she had her pneumothorax, a feature of chronic pneumothorax to which Brock (1948) has drawn attention. I have been successful in following her up for over seven years, and, although she stays some considerable distance from the Royal Infirmary, she reported back and I had the opportunity of examining her. She is very breathless on moderate exertion, particularly if she has to go up a slight incline, but whether due to lack of exercise or other causes, she is now grossly overweight. Her height is 5 st. 0 in. and her weight /
SPIROMETRIC TRACING AFTER PNEUMONECTOMY

Case 66. Photograph of the spirometric tracing of a patient who had a pneumonectomy for a chronic pneumothorax more than six years before. Vital capacity is now 1200 c.c.
weight is 12 stone 5 lb., the average weight for her height and age being approximately 9 stone 0 lb. Her haemoglobin and erythrocyte count were both below normal, and I therefore took steps to have this corrected, along with her obesity. Her vital capacity is much reduced, and a photograph of her spirometric record is shown on the opposite page. If allowance is made for the fact that it is practically impossible for patients to give an accurate tracing at the first attempt at spirometry owing to their unfamiliarity with the procedure, this record does not show the gross changes in the form of the tracing, which Christie (1936, 1944) has shown can be observed in cases of advanced emphysema. He notes, however (1936) that the significance of this alteration in the shape of the tracing in mild or moderate cases of emphysema is doubtful, and his observations on spirometric tracings in emphysema are discussed in more detail later, in the section dealing with this condition. This patient is able, taking her own time, to carry out all her normal household duties, and I believe that she can be still further improved by a reduction in her weight, and an increase in her haemoglobin.
haemoglobin.

Before leaving this patient's case, I would like to mention an interesting observation that came to my notice at the follow-up interview. It was stated in her case records that she had "lost two stones in weight" while she had her pneumothorax, but no actual note of her weight had been recorded. She herself, however, remembers being weighed at the time she was in the hospital, and gave her weight then as 10 stone 10 lb. If this is accurate, and I have no reason to believe otherwise, she was still about 2 stones overweight for her height and age, which suggests that Brock's (1948) observation that these patients are underweight is not universally applicable in all cases of chronic pneumothorax, since her pneumothorax had probably been present along with her "loss of weight" for over six months. The patient whose records and photographs Brock used to illustrate this point had had her pneumothorax for seven months.

One other patient gives a history of a relative with similar symptoms (Case 89). In this instance the patient gives a history of "pleurisy" seven times in seven years, and has had a recurrence of his pneumothorax within the last eighteen months. It is not unlikely that these attacks of recurrent "pleurisy" were
were in fact repeated pneumothoraces but, as no definite proof of this exists, they are not counted as such in the foregoing section on "Recurrences." His sister likewise gives a history which is very similar. In a letter which she was good enough to write to me she says:— "From a very early age I have been subject to attacks of pleurisy, caused it is thought from a bronchial condition contracted as a child. Actually I have been more or less subject to yearly attacks of pleurisy, though some have been so mild they have not give me much concern."

I was able to trace a report on an X-ray film which was taken at another hospital (she does not reside in this area), and this stated briefly "No focal lesion shown, and no pneumothorax was noted." The possibility that these attacks are small recurrent pneumothoraces cannot altogether be excluded however.

**BILATERAL AND ALTERNATING.**

Only three patients in the cases recorded here had pneumothoraces which occurred simultaneously on the left and right sides of the chest (Cases 32, 72 and 97). In each of these cases the air had apparently entered both pleural sacs at the same time, though Olbrecht's (1930) in his definition of this condition allows for
a second type, where air enters the contralateral pleural cavity while that of the first is in the process of being absorbed.

Mills (1928), reporting a case in the Edinburgh Medical Journal, was able to collect fourteen cases of bilateral simultaneous pneumothorax up to that time. In 1934 Markson and Johnson reported a case in a "young thin college student", and cite another recorded by McMahon (1932), and in the same year Oechsli and Miles (1934), reviewing seventy-six recorded cases of bilateral pneumothorax found that 49 per cent. were due to tuberculosis, 30 per cent. were due to emphysema, and 21 per cent. were of the "idiopathic" variety. Cole and Nalls (1938), collected eighty-two cases, and Moorman (1940) collected a similar number, nine of which showed evidence of silicosis. Macklin and Macklin (1944) refer to twenty-eight "benign" cases in the literature, which they state are only a few of those recorded, and additional cases have been added since that time. (Schneider and Reissman 1945, Komrower 1947, Yudkin 1947). The Macklins point out that most of these patients succumb because of the extreme limitation of respiration, but in the present series two patients have been successfully treated. The third (Case 72) who /
who died has been referred to earlier in this Thesis, and a full autopsy report is given later in the section dealing with post-mortem examinations.

In King and Benson's (1944) subject, autopsy showed a ruptured bleb in the left lung, though no cause was found for the pneumothorax which had occurred on the right. In order to avoid needless repetition, a further discussion of the possible mechanism in such cases is deferred to the section dealing with Interstitial Emphysema.

A further six patients in my material have a history of either a previous attack on the opposite side, or have had an attack on the contralateral side during the follow-up period. One of these subjects (Case 32), aged 26 at the time of admission had had four previous incidents on one or other side. On follow-up 10½ years later he was found to have had only one further attack. Case 81 had had four recurrences in a two-year follow-up, each side being affected, but fortunately not simultaneously. This latter type of case I consider should be referred to the Thoracic Surgeon for treatment, either by induction of an artificial pleuritis or some such other means as may be indicated by the findings on thoracoscopy. I have acquainted the Physician under whose /
whose charge the patient was while in hospital, about 
these recurrences with a view to this being done, 
since the occurrence of a simultaneous bilateral 
pneumothorax at any future date might prove fatal.

References to alternating spontaneous pneumothorax 
in the literature are more numerous than is the case 
in the simultaneous variety. Kjaergaard (1932) in 
his material had one example and refers to another 
eleven. Wood (1931) states that 11 per cent. of his 
seventy-one patients had involvement of both sides at 
different times, and Oechsli and Miles (1934) in their 
survey collected from the literature seventy-six 
recorded instances. Since that time, additional 
cases have been added. (Kirshner 1939; Niehaus 1947; 
Brock (15 cases ) 1948). Moreover, several 
references are made in the literature to the develop-
ment of a spontaneous pneumothorax on the contra-
lateral side during or following the induction of 
artificial pneumothorax (McCallum 1919; Walsh 1924; 
Andosca 1938; Cutler 1938; Zavod 1939).

That a rupture should develop in a few cases on 
the contralateral side is not surprising, and in some 
ways one might expect it to be more common. Christie 
(1936) has shown in experiments on the rabbit and 
confirmed in one case in a man with a bilateral 
artificial /
artificial pneumothorax, where the pneumothorax on one side was minimal, that the variations with repiration in the intrapleural pressure on the side with the minimal pneumothorax (which was only sufficient to allow manometric pressures to be taken), became widely divergent as more air was introduced into the contralateral pleural space. He has also produced convincing evidence that the unaffected lung is more rigid, owing to its increased vascularity, and that with the decreased distensibility of the lung, a force of nearly three times the normal intrapleural pressure may be required to yield a tidal air of only half the normal volume. The extra strain on any part of the lung which is thin or atrophic will therefore be markedly increased during respiration, and tend to produce rupture at that point with subsequent collapse of the lung. Alternatively, an over-stretching of the elastic tissue at such a point may produce a bulla liable to rupture at any time in the future.

With regard to the type of case mentioned by King and Benson (1944), where no tear could be demonstrated in the pleura covering the contralateral lung, it has been suggested that the probable mechanism /
mechanism is a rupture of the first pneumothorax through the posterior mediastinum between the oesophagus and the descending aorta, where the mediastinum is thin (Joannides 1934). If a tension pneumothorax is present, a herniation of the mediastinum may often be seen on X-ray films behind the heart shadow, and as this is a part which is poorly supported by rigid surrounding structures it may be assumed that a tear could occur at such a site, leading to the introduction of air into the contra-lateral pleural sac. Such a communication may seal off rapidly and, under normal routine autopsy procedure, may not readily be demonstrable. In the dog, induction of a pneumothorax on one side leads almost immediately to a pneumothorax on the other, by such a mechanism since the mediastinum is thin. This is not so in the rabbit, which is usually preferred for experimental work of this type on that account, since the mediastinal partition is less flimsy.

There are at least four probable explanations of the occurrence of a bilateral pneumothorax in man. They are:

Firstly /
Firstly, localised over distension of the contra-lateral lung with almost immediate rupture.

Secondly, localised over distension with the production of bulla or bleb which may rupture later, and:

Thirdly, rupture of the first pneumothorax through the mediastinum into the opposite pleural sac.

A Fourth possibility must also be considered in this connection, and that is the extension of interstitial emphysema of one lung into the mediastinum, with the consequent production of a pneumomediastinum, which may then rupture into either pleural space.

Fuller consideration will be given to this possibility in a later section, when the mechanism of pneumothorax is discussed.

OCCUPATIONAL INCIDENCE.

The occupation of all except five of the patients in this series has been studied with a view to comparing the incidence of spontaneous pneumothorax in heavy manual workers, compared with that for those engaged in less strenuous work. The highest incidence I have found to be in shopkeepers and salesman (11 cases). Next come clerks (9 cases) and miners (10 cases).

Some comment requires to be made about this last /
last figure for miners, since the number of these workers admitted to the Royal Infirmary, in relation to the general population in the area, is relatively high. Serving as it does the coal-fields in the Lothians and Fife, the Royal Infirmary provides medical services to those engaged in the coal mining industry and oil workers in these areas, who together form a larger group than any other special class of worker. Since silicosis produces fibrosis of the lung and may lead to the production of spontaneous pneumothorax, it might be expected that this group of workers would show a higher incidence rate than other groups. But this is not apparently the case, at least in those patients who show no obvious radiologic evidence of pneumoconiosis.

Until the year 1931 an Occupational Register was kept in the Registrar's Office of the Royal Infirmary, in which occupations of the patients admitted were recorded, or in the case of married women and children, that of the husband or head of the family. This Register was discontinued in 1931. In that year, however, the total number of patients admitted to the Infirmary is given as 19,184, of whom 2,641 were employed in mines or oil works (Mackinlay, 1949, personal /
**TABLE "L".**

**Occupational Incidence (1).**

<table>
<thead>
<tr>
<th>Occupations of Kjaergaard's subjects.</th>
<th>No. of Cases.</th>
</tr>
</thead>
<tbody>
<tr>
<td>A Heavy physical work (Blacksmith, Farmer, ....) 13</td>
<td></td>
</tr>
<tr>
<td>B Less exhausting physical work (Electrician, ... Shoemaker) 8</td>
<td></td>
</tr>
<tr>
<td>B Housework (Housewife, servant, etc) .......... 10</td>
<td></td>
</tr>
<tr>
<td>Shop clerks, Functionaries, Agents .......... 6</td>
<td></td>
</tr>
<tr>
<td>Students ...................................... 6</td>
<td></td>
</tr>
<tr>
<td>Office Workers ................................ 7</td>
<td></td>
</tr>
<tr>
<td>E Not recorded .................................. 1</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Occupations of Perry's subjects.</th>
</tr>
</thead>
<tbody>
<tr>
<td>A House duties and Labourers .......... 6</td>
</tr>
<tr>
<td>B Tailors .................................... 12</td>
</tr>
<tr>
<td>B Children ................................... 4</td>
</tr>
<tr>
<td>B French Polishers &amp; Drivers of Public .......... 6</td>
</tr>
<tr>
<td>Service vehicles ( 3 each) ............</td>
</tr>
<tr>
<td>B Pressers, Cabinet makers, Retired men, etc. ... 12</td>
</tr>
<tr>
<td>(2 each) .................................</td>
</tr>
<tr>
<td>C Students ................................... 2</td>
</tr>
<tr>
<td>E Clerks ...................................... 10</td>
</tr>
<tr>
<td>E Other Occupations (not detailed) ........ 33</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Occupations of Present Series.</th>
</tr>
</thead>
<tbody>
<tr>
<td>A Miners (10), Engine Fireman (5) Labourers (4) ... 19</td>
</tr>
<tr>
<td>A Linoleum worker, Hotel porter, Land worker, .... 5</td>
</tr>
<tr>
<td>B Shopkeepers &amp; Salesmen (11) Plumber (5) ...... 20</td>
</tr>
<tr>
<td>Driver Motor (4) .....................</td>
</tr>
<tr>
<td>B Electrician, Housewife, Joiner (3 each) ....... 9</td>
</tr>
<tr>
<td>B Window cleaner, Painter, Engine Driver, ...... 16</td>
</tr>
<tr>
<td>Policemen, Rubber worker Fitter engineer, .......</td>
</tr>
<tr>
<td>Foremen, Bottle cleaner (2 of each) ............</td>
</tr>
<tr>
<td>B Other occupations, Medical Practitioner, ...... 12</td>
</tr>
<tr>
<td>Hosiery worker, Stereotyper, Chemical engineer, etc.</td>
</tr>
<tr>
<td>C Students and Schoolboy .............. 5</td>
</tr>
<tr>
<td>D Clerks .................................... 9</td>
</tr>
<tr>
<td>E Uncertain or unknown ................. 5</td>
</tr>
</tbody>
</table>

Notes:

Group "A" are arbitrarily regarded as being engaged in heavy physical work.

Group "B" are considered to be engaged in less strenuous occupations.

Groups "C" and "D" are regarded as being engaged in occupations demanding light physical work.

This Table should be studied in conjunction with Table "M" overleaf.
personal communication), 13.8 per cent. therefore, of the total admission in that year fall into this group, but since there is a high accident rate in workers in these industries, many of the admissions were probably surgical emergencies, as a result of accidents at work. In order to reach a more accurate figure which would show the percentage of miners and oil workers admitted to the Medical Side of the Hospital, and because there has been an increased number of patients of the middle class and professional group admitted within recent years, I have examined the Admission and Discharge Ledger for one male medical charge for the fifteen-year period ending September 1949, and have noted the number of patients admitted who were employed in mines and oil works. The total number of male patients admitted was 6,089, and of these 615 were employed in those industries, that is 10 per cent. This figure of 10 per cent. is exactly the same as that for miners in my material, and I therefore feel justified in stating that there is no apparent increase in the incidence of spontaneous pneumothorax in what is regarded as a particularly heavy industry.

In Table "L" and "M", the occupations of the patients in this series are recorded alongside those of /
### TABLE "M".

**Occupational Incidence (2).**

<table>
<thead>
<tr>
<th></th>
<th>Kjaergaard (1932)</th>
<th>Perry (1939)</th>
<th>Present Series.</th>
</tr>
</thead>
<tbody>
<tr>
<td>&quot;A&quot; Heavy Occupations</td>
<td>13 26%</td>
<td>6 12%</td>
<td>24 25%</td>
</tr>
<tr>
<td>&quot;B&quot; Medium Occupations</td>
<td>24 48%</td>
<td>34 65%</td>
<td>57 60%</td>
</tr>
<tr>
<td>&quot;C&quot; and Light Occupations</td>
<td>13 26%</td>
<td>12 23%</td>
<td>14 15%</td>
</tr>
<tr>
<td>&quot;D&quot; Not Recorded</td>
<td>1 case</td>
<td>33 cases</td>
<td>5 cases</td>
</tr>
</tbody>
</table>

**NOTE:**

For Occupations considered arbitrarily as "Light", "Medium" and "Heavy", see Table "L", on previous page. Percentage is based only on those whose occupation is known.
of Kjaergaard (1932) and Perry (1939). The divisions between what are to be regarded as light, medium and heavy industries must be, to a certain extent, arbitrary, but the occupations have been listed so that it can be seen into which category each has been placed. If these can be regarded as correctly graded, it may then be said that there is no increased incidence of spontaneous pneumothorax in those occupations which demand heavy manual work, or physical exertion.

I have been unable to find any more recent series of published cases than those mentioned to use for comparison. Those that I have studied have either been in specialised groups of individuals, such as military personnel, or the occupation of the subjects has not been recorded. Rottenberg and Golden (1949), however, state that the majority of their patients were engaged in light physical work.

**PHYSICAL ACTIVITY AT ONSET.**

In eighty-four cases of this series the state of activity of the individual at the onset of his pneumothorax has been recorded. Perry (1939), states that, in the opinion of most writers on this subject, muscular effort is an important factor in the production of spontaneous pneumothorax. He quotes Friesdorf /
Friesdorf (1927), who collected 177 cases from the literature in which the physical activity at the time of onset was recorded, as saying that 40 per cent. arose after considerable exertion, 40 per cent. after slight exertion, and 20 per cent. after trivial movements. Perry goes on to say that as such information is secondhand, and the degree of muscular activity difficult to assess, these figures are not of great value. He argues that if this statement were true, then the incidence should be higher in heavy manual workers, which it is not the case, and he compares the occupation of his own and Kjaergaard's patients, which are shown in tabular form alongside my own material on Tables "L" and "M" in the preceding section. In contrast to Friesdorf's (1927) figure, however, Weber (1919), according to Macklin and Macklin (1944) has stated that two hundred cases of spontaneous pneumothorax have been recorded in apparently healthy persons, without any obvious cause beyond sudden respiratory effort.

I am in a position to confirm Perry's impression that extreme muscular effort is not necessarily the immediate cause which precipitates the spontaneous pneumothorax, though I would not go so far as to say that muscular effort plays no part in the production of it, after the muscular effort has ceased or been completed.
TABLE "N".
Activity of Individuals in the Present Series in Relation to onset of Pneumothorax.

<table>
<thead>
<tr>
<th>Activity of Individuals</th>
<th>Number of Cases</th>
<th>Percentage of Known</th>
</tr>
</thead>
<tbody>
<tr>
<td>A At rest</td>
<td>32</td>
<td>38</td>
</tr>
<tr>
<td>B Slight Exertion</td>
<td>42</td>
<td>50</td>
</tr>
<tr>
<td>C Severe Exertion</td>
<td>10</td>
<td>12</td>
</tr>
<tr>
<td>D Unstated or Unknown</td>
<td>16</td>
<td>-</td>
</tr>
</tbody>
</table>

C Individuals engaged in such activities as coughing, vomiting, asthmatic attacks, lifting a heavy weight, running, shovelling, at work (as a labourer), straining at stool.

B Less strenous work, painting, shaving, dressing, walking, working (as a clerk), bending down.

A At rest, 12 in bed, sitting, playing dominoes, eating, standing.
completed. The state of physical activity of the individual in my material at the time of onset of his pneumothorax is shown in Table "N" on the opposite page. In some instances the individual has given a history of a cough or head cold prior to the incident, and though it is not in my cases recorded that the onset was related to a fit of coughing, those subjects who gave such a history of a respiratory infection previous to their pneumothorax are shown, along with those whose pneumothorax came on in the morning, "getting out of bed", "having breakfast", "shaving in the morning" etc., in Table "O". This tendency for a spontaneous pneumothorax to occur in the morning has previously been noticed by Perry (1939), who has suggested that it may be connected with increased respiratory movements following a period when respiration has been carried on minimally such as occurs during sleep.

Within recent years a few contributions to the literature have recorded the physical activity of their subjects at the time of onset, such as Leach (1945) in 126 military personnel of the United States Army, and Schneider and Reissman (1948), in a similar group of unselected cases, also occurring in American Servicemen.
**TABLE "O".**

History of Previous Respiratory Infection and Number with Onset early in the Morning in the Present Series.

<table>
<thead>
<tr>
<th>Previous cough, cold, bronchitis</th>
<th>15 Cases.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset in early morning or just after rising.</td>
<td>25 Cases.</td>
</tr>
</tbody>
</table>
Hyde and Hyde (1948) found as low a figure as 3 per cent. of their sixty-three patients gave a history of sudden onset during or just after muscular effort, and twice that number were aroused from sleep by the onset of symptoms. Twenty per cent. of Myerson's (1948) thirty-six cases gave a history of unusual exertion prior to the acute episode and in the other 80 per cent. the onset was during normal activities or at rest. Cohen and Kinsman (1946), recording thirty-nine cases, show a high incidence also of onset during rest or moderate activity, with a corresponding low figure for those which came on during or immediately after strenuous exertion. Ten out of Ornstein and Lercher's (1942) fifty-eight patients gave a history of exertion - the degree is not stated - and seven of Wilson's (1934) thirty-three, and three of Blackford's (1939) eleven College students developed symptoms while at rest - sleeping, studying, and waiting for a bus, etc. A further three subjects in Blackford's series were at moderate exercise, for example "getting out of bed, dressing or shaving."

Dickie (1948) found a similar relationship to exertion in her group of College students in Wisconsin.

In thirty-two subjects in this Thesis the onset of symptoms first manifested itself while the patient was /
TABLE "P".

Activity of Subjects at time of onset in Recent Communications.

<table>
<thead>
<tr>
<th>Activity at time of onset in Recent Communications</th>
<th>Ornstein &amp; Lercher (1942)</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>A &amp; B At rest and moderate exercise</td>
<td>35</td>
<td></td>
</tr>
<tr>
<td>C Physical Exertion</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>D Unstated or unknown</td>
<td>11</td>
<td></td>
</tr>
</tbody>
</table>

| A & B Walking, eating, shaving, sleeping           | 63                        |
| C Lifting weights, running, etc.                   | 30                        |
| D Unstated or unknown                              | 7                         |

| A At rest (30 in bed, 20 sitting) (11 standing)    | 61                        |
| B Moderate activity (6 leaning over) 20 walking   | 28                        |
| C Extreme exertion (4 running) (8 exercising)     | 14                        |
| D Unknown (includes 4 found on routine X-ray)     | 26                        |

<table>
<thead>
<tr>
<th>Cohen &amp; Kinsman (1946)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A At rest</td>
</tr>
<tr>
<td>B Mild exertion (walking)</td>
</tr>
<tr>
<td>C Severe exertion</td>
</tr>
<tr>
<td>D Unstated</td>
</tr>
</tbody>
</table>
was at rest, and out of this number twelve first noticed symptoms while they were in bed. In a further forty-two individuals the onset occurred during a period of moderate or mild physical activity, such as shaving, painting or walking. In only ten subjects, or 12 per cent. of those in whose case history the physical activity at the time of onset is recorded, does muscular effort or stress appear to have been immediately responsible. Under this last heading of severe muscular effort are included those individuals who are engaged in such activities as "shovelling", "running", "coughing", and "at work" (as a labourer). From Tables "N", "P" and "Q" it can be seen that by far the largest number of patients experience their symptoms either at rest or during mild physical activity. I have included in the number whose activity was "unstated or unrecorded", those cases where the degree of activity was difficult to assess.

Although the actual onset of the pneumothorax is often not precipitated by severe muscular effort - and indeed the subject may sometimes be wakened from sleep by the onset of his symptoms - I have been struck by the fact that if a careful history is taken from these patients who have had a spontaneous pneumothorax some /
TABLE "Q".

Activity of Subjects in Recent Communications and Present Series at Onset of Pneumothorax.

<table>
<thead>
<tr>
<th></th>
<th>Group A</th>
<th>Group B</th>
<th>Group A + B</th>
<th>Group C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ornstein and Lercher (1942)</td>
<td>-</td>
<td>-</td>
<td>78%</td>
<td>22%</td>
</tr>
<tr>
<td>Leach (1945)</td>
<td>48%</td>
<td>38%</td>
<td>86%</td>
<td>14%</td>
</tr>
<tr>
<td>Schneider and Reissman (1945)</td>
<td>-</td>
<td>-</td>
<td>68%</td>
<td>32%</td>
</tr>
<tr>
<td>Cohen and Kinsman (1946)</td>
<td>61%</td>
<td>26%</td>
<td>87%</td>
<td>13%</td>
</tr>
<tr>
<td>Myerson (1948)</td>
<td>-</td>
<td>-</td>
<td>80%</td>
<td>20%</td>
</tr>
<tr>
<td>Present Series</td>
<td>38%</td>
<td>50%</td>
<td>88%</td>
<td>12%</td>
</tr>
</tbody>
</table>

**Group A:** At rest.

**Group B:** Moderate physical activity.

**Group C:** Severe physical exertion.

**NOTE:** Percentages are based on the number of cases in each series in which the physical activity at the time of onset is recorded.
some at least have been conscious of a minor degree of pain in the chest following some muscular exertion, sometimes described as a "stitch" or a "strain". Unless a careful history is taken from the patient, this minor degree of discomfort may be completely overshadowed by the major disturbance from which he dates the onset of the condition. This preceding discomfort in the chest has been noted in the carefully detailed case histories recorded in some of the communications by the earlier physicians whose accuracy of detail in history and examination is certainly not surpassed today.

As an example of this minor discomfort which may precede the onset of the spontaneous pneumothorax, I may quote Case 91 in the present series. This patient, a miner aged 27, was conscious of a slight pain in the right side of his chest which came on while he was in the process of lifting a heavy weight. He was not breathless at that time and finished his shift. At 3 a.m. the following morning he was aroused with the feeling of nausea, dyspnoea and severe pain in the right side of his chest, worse on movement and deep breathing. An X-Ray of his chest fifteen days later showed a right pneumothorax. My impression is, though I am unable to prove it, that an emphysematous bleb or interstitial emphysema of the lung is first formed, and the subsequent pneumothorax occurs following the rupture/
rupture of this bleb, or the tracking of this air in the interstitium of the lung direct to the pleural sac or along the blood vessels to the mediastinum, and thence into the pleural space. Macklin (1937), has shown such a mechanism to occur when the lungs of a cat were locally over-inflated. He has demonstrated quite convincingly that air in such cases tracks along the sheaths of the blood vessels, particularly the arteries, towards the hilum into the mediastinum and from thence burst through the mediastinal pleura into the pleural cavity. One other feature which he found was that air sometimes appeared to leak through the pleura, though there was no break in the continuity of this, and this finding has been recorded as being observed in the living human subject by Brock (1948). Brock refers to those areas which he observed on thoracoscopy as "cuckoo spit" owing to their resemblance to the froth one often sees on plants in early summer. When Brock's patients were instructed to hold the nose and blow up the cheeks, these clusters of air bubbles could be observed to increase, not only in size but also in the number of bubbles and he describes those patients as having "leaky lungs" or "porous pleurae".

While instances of spontaneous pneumothorax occurring during exercise or physical exertion have been recorded, many have also been described where there was /
was no history obtained of exertion at the time of onset, nor even fairly recently preceding the attack. Due in part to the tendency to associate pneumothorax with exercise, or with underlying disease in the lung, usually tuberculosis, many physicians have cautioned their patients against indulgence in any form of active exercise and some patients have been required to live under sanatorium regime. This is regrettable inasmuch as these patients may acquire a pulmonary neurosis, in a manner similar to those individuals who develop a cardiac neurosis on account of an innocent murmur in their hearts. Schneider and Reissman (1945), found that this was a prominent feature in several recruits that they examined. Although in some instances the restrictions which had been placed upon them by their medical attendants were possibly exaggerated by them because of their unwillingness to undertake Military Service, yet in many cases a letter or certificate from a physician was produced at the time of examination. In Schneider and Reissman's own words -

"... Not infrequently a letter from the Selectee's doctor urged a cautious almost vegetable existence, despite the fact that the spontaneous pneumothorax had occurred during no more superhuman physical effort than reading a book, shaving, or sleeping".

From /
From the findings in the present series and those others in the literature to which I have referred, I feel justified in saying that a minority of cases occur during or immediately after strenuous exercise but that many more do not appear to be produced directly by any such exertion.

STATE OF NUTRITION.

In the course of my examination of the case records, one fact caught my attention to which I had not seen any reference made in the literature on spontaneous pneumothorax. I observed that in description of these patients in my material, many were referred to as "thin" or "slightly underweight". I therefore went back over the Ward Charts of my patients and abstracted the height and weight of the individual and compared that with the standard weight for age and height as given by Dunlop, Davidson and McNee (1949).

In one or two communications on spontaneous pneumothorax the observation has been made that the patients were "thin". Hall (1887) was probably the first to make this observation, and describing a spontaneous pneumothorax in a 23 year old clerk, he gives his patient's height as 6 feet and his weight as 9 st. 11 lbs., "which is much below the average for his height". Griffin (1942), reporting three cases of "clicking" pneumothorax noted that they were all /
**TABLE "R".**

Observed and ExpectedWeights of Subject in the Present Series.

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Observed Weight</th>
<th>Expected Weight</th>
<th>Case No.</th>
<th>Observed Weight</th>
<th>Expected Weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>113 lb.</td>
<td>167 lb.</td>
<td>46</td>
<td>99 lb.</td>
<td>150 lb.</td>
</tr>
<tr>
<td>2</td>
<td>119 &quot;</td>
<td>148 &quot;</td>
<td>47</td>
<td>120 &quot;</td>
<td>156 &quot;</td>
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<tr>
<td>3</td>
<td>150 &quot;</td>
<td>160 &quot;</td>
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<td>109 &quot;</td>
<td>128 &quot;</td>
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<tr>
<td>4</td>
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<td>148 &quot;</td>
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<td>154 &quot;</td>
<td>152 &quot;</td>
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<td>157 &quot;</td>
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<td>114 &quot;</td>
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<td>157 &quot;</td>
<td>87</td>
<td>143 &quot;</td>
<td>145 &quot;</td>
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<td>151 &quot;</td>
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<tr>
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<td>130 &quot;</td>
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<td>142 &quot;</td>
<td>158 &quot;</td>
<td>98</td>
<td>124 &quot;</td>
<td>138 &quot;</td>
</tr>
</tbody>
</table>

* The observed weight is higher than the expected in those marked thus.
all tall and moderately underweight, and remarks -

"This observation may not be

significant but the tall thin

body type has been prominent

in our cases".

In Brock's (1948), series of recurrent and chronic cases, he observed as a striking feature in the chronic cases the severe loss of weight, and illustrates this with photographs of a young woman, a sufferer from asthma and chronic bronchitis, who had a pneumothorax of seven months' duration. Her weight dropped from 7 st. 4 lbs. to 4 st. 13 lbs. As far as I have been able to discover, there is no series of cases of "Pneumothorax Simplex" in which the weight of the patient has been shown to be below the average for their height and age at the time of onset, and I believe the findings recorded in Table "R" to be an original observation on this subject.

In computing the weights and heights of the subjects in this series, the following standards have been observed. Ward patients are normally weighed in pyjamas and slippers but the weight of these articles of clothing has been ignored and the patient's recorded weight taken as net. The average weights for comparison are taken from Dunlop, Davidson and McNee (1949). Since one inch is allowed for height in shoes in these Tables, and 10 lbs. for clothing, these /
these amounts have been deducted, so that for comparative purposes the average expected weight for the age and height will be deviated to the low side of normal. For example, a man of 23 years of age, 5 ft. 10 ins. in shoes and indoor clothes should have a weight of 11 st. 1 lb. Without his shoes his height should be 5 ft. 9 ins., and deducting 10 lbs. for his clothes he should have an average weight of 10 st. 5 lbs., i.e., 1 st. less. It is this lower figure which has been used for the comparison with the observed weights of the subjects in my material. Six subjects in this series were found to have weights above the average for their height and age and these cases have been marked with an asterisk.

It is possible that this observation may have some bearing on the production of pneumothorax and further reference will be made to it in a later section when the mechanism whereby pneumothorax is produced is discussed. It is, however, a hitherto unrecorded observation in cases of benign spontaneous pneumothorax.

IV. CLINICAL FEATURES OF SPONTANEOUS PNEUMOTHORAX.

GENERAL.

In nearly every case the onset is acute and the subject complains of a sudden sharp pain in the affected /
affected side of the chest. This pain may occasionally be referred to the neck, shoulder, upper abdomen or back, or be retrosternal in location, and it is usually, but not always, associated with dyspnoea of varying degree. It has been considered by some writers, (Perry 1939; Myerson 1948), that the dyspnoea is more subjective than objective. It is probably true to say that the amount of dyspnoea is associated firstly, with the severity of the pain and its accentuation by deep movements of the chest in its ventilatory capacity; and secondly, by the extent to which the lung is collapsed, or the amount of increase in pressure in the affected pleural cavity.

To state, as do Ehrlich and Schomer (1938), that "sudden sharp pain with a choking sensation on one side of the chest are invariable symptoms", is simply not true. Schneider and Reissman (1948), found six out of their series on routine radiological examination of the chest which were asymptomatic. Brock (1948), cites the case of a doctor whose pneumothorax was found only when he was being examined for life insurance, and who on close questioning was only able to give the information that some months earlier he had noticed a vague discomfort in the affected side, and had been unable, occasionally, to take a deep breath. Wilson (1937), found five cases of spontaneous pneumothorax /
Thorax without symptoms in routine chest examination of students at Yale University in a four year period, and Hyde and Hyde (1947) quote two instances of patients not included in their material, and one of their own who had asymptomatic pneumothoraces. Many other cases are also on record where a pneumothorax has been found to be present without any history of pain in the chest. This type has been called "Pneumothorax Muet" by some writers, particularly in the French literature (Olbrechts, 1930).

Two cases in this present series (Cases 48 and 85) were referred to Hospital on account of dyspnoea on exertion or a "heavy feeling" in the chest. At no time did the first patient (Case 48), who was a joiner, complain of pain in his chest and only complained of a "heavy feeling" of two days' duration. The second patient (Case 85) only complained of a feeling of constriction across his chest which was associated with his dyspnoea and came on when he exerted himself.

Pain, therefore, while a very common presenting symptom is not a universal one, or if it is present at all it may be insufficient to be recalled by the patient at a later date.

In those patients where the pain is referred to the shoulder, some difficulty may be experienced in differentiating such pain from a local lesion in the shoulder /
Simultaneous bilateral spontaneous pneumothorax in a young man of 23 years of age, who had a history of several attacks of "rheumatism" in the left shoulder region.
shoulder itself, the gall bladder or the heart. One subject in my material (Case 94), was admitted to hospital with a bilateral pneumothorax and had a history of several preceding attacks of "rheumatism" in the left shoulder. His age was 23 years, and his occupation that of Police Constable. On close questioning him about this "rheumatism", I found that these attacks had often come on at rest and were not associated with any particular strain, and that the pain was not related to the joint but over the area between the scapular upper border and the clavicle. Though no gross dyspnoea was noticed by this patient, in connection with this pain in the shoulder region, it is not impossible that these attacks were in fact recurrent attacks of spontaneous pneumothorax, since no clinical or radiological evidence could be found in the region of the shoulder to account for them, and no similar pain had been felt in any other joint region. In the photograph of the X-Ray film of the chest of this patient there is, however, some pleural thickening in the left lung which, while not proof, suggests that some previous irritation of the pleura has occurred.

SIMULATION OF ABDOMINAL DISEASE.

The /
PARTIAL RE-EXPANSION OF BILATERAL PNEUMOTHORAX.

Case 94. Film taken eleven days after that illustrated on the previous page (Fig. 4). Re-expansion is taking place in each lung spontaneously. No aspiration or other procedure was carried out.
The patient whose presenting symptom is of severe pain in the upper abdominal region may easily be mistaken for an acute abdominal emergency. The differential diagnosis may be rendered more difficult if the individual has a history of preceding pain or discomfort in this region and has been treated for dyspepsia or as a peptic ulcer. Steigman and Singer (1936), could not find any reference to spontaneous pneumothorax in the standard text books in America and the Continent, as a differential diagnosis to be considered in cases of upper abdominal pain, and one only (Adams 1923), in the English textbooks. They cite Beardsley (1911), Hurxthal (1928), Siebner (1932), and Oechsli and Skillen (1933), as the only published cases in the English literature and add four cases of their own. Muller and Mogavero (1933), have also recorded a case where the abdomen was opened for a diagnosis of perforated gastric ulcer, and abdominal symptoms as a feature of spontaneous pneumothorax have also been reported by Settle (1936) and Beatty (1938). Earlier cases, however, have been recorded by Rolleston (1900) and Fischer (1922), though in each instance a haemo-pneumothorax was present and Hurxthal's (1928), patient also had blood as well as air in the pleural space. Perry (1938) reported two cases /
cases of haemo-pneumothorax, each case being originally diagnosed as a perforation, in one of whom laparotomy was carried out. It would appear that those patients who have an associated haemothorax are more likely to refer the pain to the upper abdomen, probably owing to the irritation of the peripheral part of the diaphragm by the blood. Hartzell (1942), collected forty cases of spontaneous haemopneumothorax from the literature, and added three of his own, and in many of these recorded, abdominal pain was a feature, and in some the abdomen had been opened surgically; others have been recorded since that time.

In no less than seven cases in the present series the abdominal symptoms were a prominent feature, and led to a laparotomy in three instances, (cases 6, 9, 43, 52, 58, 72 and 82).

Case 72 - was a young man admitted in extremis with a history of hiccough and vomiting of three days' duration prior to admission, and came under my charge as the House Physician on duty. He only lived for a very short time after admission to Hospital, and died of bilateral pneumothorax. The post-mortem examination, in addition to revealing rupture of bullae in each lung which had produced his pneumothoraces, showed also the presence of a duodenal ulcer. Fuller details of the autopsy report are given in the later section/
section of this Thesis, where the pathological reports are recorded.

The second case in which I had a personal interest (Case 52) was referred to the Surgical side of the hospital by an experienced and astute practitioner, who was aware of the possibility of spontaneous pneumothorax mimicking an acute abdominal condition. Since he knew that I was on duty that evening, he requested that she might be seen by the Physician-on-Duty if there was any doubt in the surgeons' minds as to the diagnosis. I was therefore called to see the patient, a woman aged sixty-eight, who had been suddenly seized with pain in the back while playing dominoes. She gave a history suggestive of peptic ulcer, with recent recurrence of attacks of "indigestion" for which she had been taking alkaline powders. She also gave a history of a "heart attack" about five years ago. With the onset of her present attack of pain she had vomited, and was short of breath. On examination she was cold, clammy, dyspnoeic and extremities were cyanosed. The abdomen was guarded and almost board-like. It did not move with respiration. There was no apparent loss or increase of liver dullness. The chest movements were poor, but apparently equal, the trachea central, percussion /
percussion note equal on either side, and the breath sounds were somewhat faint at the right base, with some coarse crepitations or friction sounds. The pulse was regular and of good volume, and the blood pressure 150/80. The apex beat was in the 6th space in the anterior axillary line, and a thrill could be felt and a rough systolic murmur heard at the mitral area. On these and other findings, I considered that she had had a spontaneous pneumothorax, and she was therefore treated conservatively with oxygen and general supportive measures, and sedation. The following morning I saw her again, when signs of a right pneumothorax were then obvious, and on inserting a needle into the pleural sac on the affected side, air came out under pressure. Her condition improved, but it shortly became obvious that air was again collecting under pressure in the sac, aided by a short cough which had developed, and a needle was again inserted into the chest from which a length of rubber tubing was led to a bottle containing water to form a valve, in order that air might escape when the pressure in the pleural space rose above one cm. of water. About 5 c.c.m.s. of air escaped through this mechanism with each expiration, more being blown out if the patient coughed. Again her condition improved /
improved slightly, though her pulse began gradually to weaken. She became unconscious latterly and died about five hours later, and in the later stages a blood stained fluid was being expelled through the tube. Unfortunately permission for a post-mortem examination was not granted, so that the cause of the pneumothorax was not ascertained.

Other three patients underwent operative treatment for abdominal symptoms, and in two of these a positive history of duodenal ulceration existed, with radiological confirmation. Case 43 had a history of ulcer of 11 years' duration, and was a man aged 29. On fluoroscopic examination, prior to operation, he was seen to have a small right pneumothorax, as well as evidence of duodenal ulceration. He underwent a partial gastrectomy for his ulcer. After his operation his condition was precarious since his pneumothorax had increased in size and he was transferred to the Medical side of the hospital for treatment of this. X-Ray at this time showed a further collapse of the right lung. With three weeks' treatment, the lung was seen to be almost fully expanded, and he was well and had had no recurrence of his pneumothorax three years later.

Case 55 was a housewife aged 42. She was admitted /
admitted as an emergency with a diagnosis of perforated duodenal ulcer, and she gave a history of twenty years' symptoms of dyspepsia. She had had a slight cold and cough prior to admission, and during the night she had been seized with a severe pain below the left hypochondrium. This was diagnosed as a probable perforation of a peptic ulcer. At operation she was found to have scarring of the duodenum but no perforation was discovered and after laparotomy, she was much more dyspnoeic and the signs of a pneumothorax became obvious on the left side, with deviation of the trachea and apex beat. In spite of treatment, she died fifteen days later. Permission was not granted for post-mortem examination.

The third patient in this group who had a laparotomy (Case 82), was a young solicitor who had no previous history of dyspepsia. He was sitting by the fire when he was suddenly seized with a severe pain in the epigastrium and admitted to the Royal Infirmary three hours later. Though he had no history of previous dyspepsia, he also had no previous trouble with his chest, apart from whooping cough at the age of four or five. The signs were sufficient to make a laparotomy justifiable, but at operation no intra-abdominal lesion was found. The anaesthetist, however /
however, noticed that the right side of the chest was moving less freely than the left, and on the liver being retracted, the right leaf of the diaphragm was seen to be bulging into the abdominal cavity, and moving paradoxically with respiration (Falconer 1949, personal communication). The abdominal incision was repaired and treatment directed to the pneumothorax and he made an uninterrupted recovery. I have seen and examined this patient just over a year after this incident, and he had no clinical or radiological evidence of disease in his lungs, and is completely free from symptoms.

Case 1 - a man aged 54, was admitted in 1947, with a left spontaneous pneumothorax. He gave a history of having had a spontaneous haemopneumothorax fifteen years ago for which he had an abdominal operation. He says that at that time he was unconscious for four days, but he eventually made a successful recovery. Following his last attack, he was seen by Mr. Logan at the Thoracic Unit, Eastern General Hospital, Edinburgh, and I am indebted to his Clinical Tutor for a report on the Thorascopic and other examinations which were carried out by Mr. Logan. Briefly, these examinations showed no evidence of tuberculosis, nor any emphysematous bullae, and the tear in the pleura was/
was not visualised. This latter finding is not uncommon. Brock (1948) in six out of his 71 chronic and recurrent cases did not visualise the site of the lesion, and as the patient, in this instance, had had his pneumothorax for three weeks, it is most likely that the opening was already sealed off and that the lung would have re-expanded on its own without treatment, as the majority of them do. Nevertheless, I consider it is sometimes a wise measure in a man of this age to undertake thorascopy since a lesion may be seen suitable for treatment at that time, which left alone, would endanger such a patient's life should he have a recurrence, or one of the complications of pneumothorax in some situation where the highly skilled technique for treating thoracic emergencies is not readily available.

In those cases where there is no evidence of blood in the pleural cavity and yet who complain of abdominal pain, the Macklins (1944), have offered convincing evidence that this may be due to air behind the peritoneum. The air in such cases tracks down from the mediastinum along the descending aorta and inferior vena cava, and unless it bursts through into the peritoneum, it is inaccurate to describe this condition as pneumoperitoneum. Macklin (1937), demonstrated /
demonstrated by local overinflation of a rabbit's lung, how the air dissected its way down behind the peritoneum. So much air accumulated that the kidneys were elevated from their beds of fat and lay floating on huge vesicles of air (Macklin and Macklin 1944). It is not unlikely that such a mechanism is operative in the human subject since pneumomediastinum is not infrequently an accompaniment of pneumothorax. Hamman (1945), goes as far as to say that he believes pneumomediastinum or mediastinal emphysema to be present in one third of cases of pneumothorax, and if the air is in sufficient quantity to produce symptoms in the retroperitoneal space, pain is likely to be referred to the region supplied by the appropriate somatic nerves. On the other hand, where there has been a direct leakage of air through the diaphragm, a pneumoperitoneum will be produced which will most likely not give rise to any symptoms of pain, unless by the loss of cohesion between the liver and diaphragm, the latter will drag on its mesentery (Newell 1944).

Helwig and Schmidt (1947), collected thirteen fatal cases of spontaneous haemopneumothorax from the literature to which they added one of their own and Crawford and Shafer (1946), describing one of their /
their own cases collected a total of 43 cases of haemopneumothorax from the literature to that date. Further cases have been added by Mckyn (1947, 2 cases), Orsi (1947, 2 cases) and Harrington and Frelick (1947). I have not had the opportunity of studying these last two publications, but in many of the cases cited, abdominal pain was a prominent feature of the presenting symptoms. In passing too, it is interesting that the source of the bleeding was unable to be demonstrated in not a few instances.

Before leaving this aspect of spontaneous pneumothorax it may be of some significance to note that a further seven cases in this series had direct or presumptive evidence of peptic ulceration in the shape of positive barium meals, haematemesis, or gastroenterostomies (Cases 5, 13, 30, 31, 58, 84, 99).

SIMULATION OF HEART DISEASE.

Occasionally the presenting symptoms may be such as to suggest the diagnosis of angina pectoris or coronary thrombosis (Miller, 1945), and this is not uncommonly the case if the pneumothorax is small with few if any physical signs, and on the left side. The shocked appearance of the patient, low blood pressure, and somewhat faint heart sounds may prove a trap for the /
FIGURE 6.
ADHESION AND PNEUMOTHORAX.

Case 96. Left pneumothorax with apical adhesion. The translucent areas below the pulmonary trunk, and above the right auricle are probably due to air in the mediastinum, or pericardial sac.

FIGURE 7.
RE-EXPANSION WITH ABSORPTION

Case 96. The same patient as above. This film was taken on follow-up 2½ years after the pneumothorax incident. There is some emphysema, with slight prominence of the pulmonary conus, but the film is other-wise essentially normal.
the unwary, and some cases are undoubtedly missed or wrongly diagnosed. Radiological examination is usually helpful, however, and the absence of changes in the electrocardiogram, apart from occasional axis deviation, usually succeeds in differentiating the two conditions. The average age incidence of the two conditions is also helpful in arriving at the diagnosis, benign spontaneous pneumothorax being most common in the age group around thirty years, as has been shown earlier in this Thesis, whereas coronary vessel disease is more common in the "over forties". Photographs of the X-Ray films of Case 96 are reproduced on the opposite page. This patient had a severe gripping pain behind the sternum associated with his left pneumothorax, and the film shows what is probably air in the mediastinum or pericardial sac. I have shown this film to two Senior Consultant Radiologists to the Royal Infirmary, Drs. W. S. Shearer and J. McGibbon, along with others taken at the same time, and while they are not prepared to be dogmatic about the diagnosis without further views in different positions, they consider that the shadows in the region below the pulmonary trunk, and above the right auricle are most likely due to air in these situations. On the same page a follow-up film taken almost 2.5 years later /
later is shown, during which time the patient had enjoyed perfect health. With the clinical history, supported by these X-Ray films, I am of the opinion that this patient probably had a pneumomediastinum at the same time as his pneumothorax. I have reproduced a similar type of pain by mistake on one occasion in the course of aspirating a pericardial effusion.

It is not unlikely that the "heart" symptoms associated with pneumomediastinum are due to the air in the mediastinum producing a "Cardiac Tamponade" (Beck, 1944). Pneumomediastinum is a condition which is not easy to demonstrate but is sometimes recognised in children, in whom it is often associated with subcutaneous emphysema appearing about the clavicles. Two cases of pneumomediastinum, neither of which was, however, associated with pneumothorax, have been encountered in my study of case records in this research. I was given clinical responsibility for one of these but as neither of them had a pneumothorax, they have not been included in the material under review. The first case occurred in a young girl with whooping-cough, and was associated with "surgical" subcutaneous emphysema in the neck. Her complaint of sudden gripping pain behind the sternum, travelling up into the /
the neck, might have led in an older person to consideration of coronary disease as a possible cause for this, and the preliminary diagnosis was pericarditis in her case. The subsequent appearance of crepitus and bulging above the clavicles established the diagnosis of mediastinal emphysema (Case 4757/26).

The second case was one for which I was given clinical responsibility, and occurred in a man aged 51 (Case 9781/32). In this case, pain had come on while at work as a labourer, was retrosternal, and was unassociated with those physical signs of pneumomediastinum which will be discussed in the next section, the only sign being a fullness above the left clavicle which became prominent when the patient coughed, and which gradually disappeared as the air was absorbed. Miller (1945) describes eight cases of spontaneous pneumomediastinum, six of which were associated with a left-sided pneumothorax; all had symptoms suggestive of primary heart disease.

CLICKING PNEUMOTHORAX.

An interesting clinical finding in some cases of pneumothorax is the occurrence of "clicking" or "crunching" sounds synchronous with the heart beat. Hamman (1937), is usually given the credit for being the /
the first to describe this sign, and indeed in the American writings on the subject, it is often referred to eponymously as "Hamman's Sign" and as being diagnostic of mediastinal emphysema. In his communication to the Association of American Physicians (1937), however, Hamman himself states that such signs had been already described by Muller and by Hoffman. Such a finding was also recorded by Lister (1928), and Kjaergaard (1932), cites Ljungdahl (1918), as describing two cases. The earliest description of these "clicking" or "crunching" sounds which I have come across is in the communication of Lundie (1891), a general practitioner in Edinburgh, who described a case of pneumo-pericardium and pneumothorax to the Edinburgh Medico-Chirurgical Society, in which the diminished cardiac dullness and the "clicking" and "crunching" sounds are excellently described. This was in the days when the benefits of radiology were not available as an ancillary diagnostic measure.

Some difference of opinion exists as to whether the air in these cases is in the pericardial sac or in the mediastinum, and an attempt has been made to differentiate between air in the two situations on clinical grounds, (Scadding and Wood, 1939). Ellman and Hussain (1948), advance the theory of a weakness /
weakness in part of the pericardium, quoting various authorities, eighty recorded cases and three of their own, and consider that this is the cause of the pneumopericardium which sometimes occurs when an artificial pneumothorax has been induced. Edwards and Simpson (1939), also describe three cases where this "clicking" sound was noted after induction of an artificial pneumothorax. In all three of the latters' patients, a bilateral pneumothorax was present, and the "click" or "knock" was audible at times to the patient, though it was not always present. In two instances the sound could be abolished by changes in position or by firm pressure over the pericardium with the hand. Scadding and Wood (1939), state that the sound produced in pneumopericardium is a distinct "click" or "knock" and they differentiate it from the "crunching" sound which is present in mediastinal emphysema, though the two must fairly frequently co-exist, one would think. These workers were able to produce this same "clicking" sound experimentally by the induction of a small therapeutic pneumothorax in two patients who were suffering from minimal tuberculous lesions in the lungs. They noted that the sound was only produced when the amount of air in the pleural space was small, and that it rapidly /
rapidly disappeared when the amount of air was increased, and being on the lookout for this sign, they recognised it in four patients in a two-year period.

Macklin and Macklin (1944), describe how they were able to produce mediastinal emphysema in their experimental animals by local overinflation of the lung, but have noted that in only one animal did they find air in the pericardial sac, though air in the pleural spaces and mediastinum was present. It is, of course, not possible to argue from results in experimental animals, that the same train of events will necessarily happen in human subjects, but I believe that a very high level of diagnostic skill must be required to differentiate between a small amount of air lying within the pericardium or outside it in the mediastinum. Even radiology is not able to help very much in such cases, though Thompson (1947), describing three cases of his own and referring to a further five, was able to demonstrate an air bubble in two by this means. The exact location, however, of any such bubbles must necessarily be a matter of personal opinion, or at any rate demand a high level of diagnostic skill and experience from the Radiologist. Harp and Peeke (1949), state that in all ten cases of proved pneumopericardium which have been reported between /
between 1931 and 1949, most of these have been associated, like their own case, with disease elsewhere in the body.

An interesting short contribution to the writings on "Clicking Pneumothorax", is that of one who is himself a sufferer from recurrent spontaneous pneumothoraces, and who gives an excellent description of the phenomenon from a patient's point of view, as well as a doctor's (Dickinson 1949).

The faint "clicking" or "crunching" sounds which may be present, may occasionally be mistaken for pericardial friction, and mention has been made in the preceding section of such a case where the diagnosis of pericarditis was made prior to the appearance of air above the clavicles. In another case included in this series (Case 40), the patient has been treated in the Royal Infirmary as a case of pericarditis one year before. On re-admission for the second time with exactly the same symptoms a small pneumothorax on the left side was shown to be present on the X-Ray films. His symptoms were severe pain behind the sternum and breathlessness of three days' duration. According to him, he had been wakened up with a severe pain behind the sternum three days before, but got up and went to his work. The pain, however, became more severe /
severe and travelled through to the back. It was persistent and unrelated to effort, and his doctor sent him in to Hospital. On examination at the time of admission, he was found to have a friction rub with a diastolic "click" at the apex. On follow-up he was found to have had one recurrence of symptoms in his left chest. The "clicking" sound in his case was only heard with the aid of the stethoscope.

Another four examples of this phenomenon are present in my material (Cases 50, 57, 84 and 95), and are sufficiently interesting to refer to briefly.

Case 57 - was a young medical student at the time whom I have seen frequently since he graduated some seven years ago. In his case, he tells me, the "click" was synchronous with systole, and this has been so in most of the recorded instances. The sound on this occasion was so loud that it could easily be heard at the other side of the room. His pneumothorax came on at 2 a.m., wakening him up from sleep, and the clicking was clearly audible at the time. He was more than a little alarmed about his condition since he was unable to fit his symptoms with any of the usual conditions about which he had read in the ordinary Textbooks of Medicine! The phenomenon, however, which varied in its loudness with his position, disappeared within /
within about twenty-four hours, and the pneumothorax which he had, was shown radiologically to be a small one on the left side. Nine years later after active service with the Royal Air Force, during which he was engaged in energetic sporting, as well as other activities, he is fit and well and has had no recurrences.

Case 50 - was a young man of 21 who complained of pain in his left shoulder of one day's duration. On rising up after lighting the fire in the morning of admission, he had felt a sudden pain in the region of the left nipple. This pain travelled to the left shoulder and down the back of the left arm to the elbow. He was very breathless and had to sit down and the breathlessness soon passed off. He noticed a "clapping" sound at this time which accompanied his heart beats. An X-Ray of his chest at this time showed a small pneumothorax on the left side, the lung being in about 1-2 cms. from the chest wall. The presence of air was not noted in the mediastinum, and he was discharged from the Infirmary in under three weeks. After a short convalescence, he resumed work (as a railway fireman), but had to go off again owing to his right lung collapsing, he informs me. He was /
was able to start again shortly, however, and is now in Northern Rhodesia whence he replied to my follow-up enquiry in June 1949, telling me that he has had his chest X-Rayed frequently, without evidence of any disease being found, and has had no recurrence of his pneumothorax.

Case 84 - was a civil engineer aged 41. While on his way to his office in the morning, he started sneezing and was seized with a sudden severe pain in the left side of his chest posteriorly. He was unable to drive his car and was brought into the Infirmary in an ambulance. No definite signs of pneumothorax were made out on physical examination, but a "clicking" sound synchronous with the cardiac systole was noted. No other abnormal findings were made out, and an electrocardiogram and X-Ray of the chest were normal four days later.

Case 95 - a plumber aged 27, had been seized with a severe pain in the left side of his chest seven days before admission. At that time or shortly after, he had been conscious of a "scratching" noise in his chest concurrent with his heart beat, which he felt more than heard. When he was examined on admission, he was found to have a "clicking" noise synchronous with his cardiac systole, but this could be heard with /
with the stethoscope only, and was not audible otherwise. It was heard when he leaned forward and to the left, or if he lay on his left side, and it disappeared within about twenty-four hours. As I was given clinical charge of this patient, I was able to study the phenomenon closely, and it was noticeable that he could tell me by altering his position when I would hear the sound through my stethoscope (though neither of us was able to hear it ordinarily), if I placed my stethoscope over the position of the apex beat.

Unfortunately I have not, however, been able to demonstrate the presence of air in the mediastinum in any of these last four cases by radiological means nor in the pericardium. It is likely that if any was present that the amount was small, and the interpretation of radiological shadows as air bubbles in the mediastinum in such cases might be considered as being biassed by "wishful thinking" and are in many cases a matter of personal opinion in the eye of the beholder.

All of my subjects had a left pneumothorax, and this has been the case in most recorded series. Griffin's (1942), three cases were all left-sided, as were those four of mine of which mention has been made. Edwards' and Simpson's (1939), three cases had bi-lateral /
bi-lateral pneumothoraces and Dickie (1948), records that in the seven cases in her series where mediastinal emphysema was associated with pneumothorax, the pneumothorax was always on the left side. She draws attention to the fact that she was only able to find one recorded case where a right pneumothorax was associated with mediastinal emphysema, (Schwartz et Al. 1946). One, however, where the "clicking" was related to walking, is recorded by Black (1948), and in this instance the pneumothorax was on the right side. Dickie (1948), in a footnote to her contribution, states that since the time her article was submitted for publication, she has seen a further seven cases of mediastinal emphysema, each of which was associated with a right pneumothorax.

The diagnosis of mediastinal emphysema where the amount of air is small, and where this diagnosis is made on hearing transient faint "crunching" or "scraping" sounds over the pericardium, and largely on this alone, suggests an extremely high degree of clinical skill, combined with awareness of the possibility of its existence, or a modicum of prejudice. Hamman (1945), gives as his opinion that one third of cases of spontaneous pneumothorax have an associated mediastinal emphysema. With further advances in the means of demonstrating this convincingly, such may be shown /
shown to be the case, but I am not aware that any such proof as yet exists. Nevertheless I am sure that the diagnosis of co-existent mediastinal emphysema must be missed in many cases of spontaneous pneumothorax, and it was only Hamman’s awareness of the condition that led him to interpret correctly the physical signs of it, and led him to search for it in every case of pneumothorax which he encountered. Since the signs are frequently transient, they are probably frequently missed, when the clinical picture is dominated by the major features of the pneumothorax itself.

Since the time I read of the clinical signs of pneumomediastinum, I have been on the lookout for a case in which I could make a confident diagnosis, but up to the present time I have not encountered one in which I could do more than suspect the possibility of its existence.

TEMPERATURE, PULSE etc.

Kjaergaard (1932), and Perry (1939), have each pointed out that pneumothorax occurring in the apparently healthy is not associated with any marked increase in the temperature or pulse rate, and if there is any such rise, that it should lead to a search /
search on the part of the clinician for some underlying cause for this. In many of the cases in this series the temperature on admission has been found to be normal or almost subnormal in some instances.

Though there is not the same communication with the outside atmosphere which leads to the known rapid loss of heat in open pneumothorax, such as that following penetrating wounds of the chest wall, it is possible that this low temperature is associated with the shocked condition of many of these patients following the incident. It is a noticeable feature in most of the patients that they are pale rather than cyanosed in appearance, and the cardiac output after pneumothorax has been shown to be diminished in cases where this procedure has been carried out therapeutically, (Courmand, Bryant and Richards 1935). The blood pressure is also on the low side of normal, but unless this and the pulse rate are taken immediately after admission, the shocked state is likely to have passed off, and normal findings are the rule. Since the pulse rate and the temperature are usually recorded by a junior member of the nursing staff, the accuracy of the recorded figures may sometimes be open to question. This criticism is particularly applicable to records of the respiratory rate owing to the fact that it is in most cases assessed at the same time as the pulse is/
CASE "S".

Pulse, Temperature and Respiratory Rates of Patients admitted within 24 hours of onset.

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is being counted. In this way minor increases of the rate may be missed. Bearing these possible sources of error in mind, it is nevertheless of value to consider the figures for those cases that have been admitted within twenty-four hours of the onset of their pneumothorax.

The ward charts of thirty-seven patients in this series who had been admitted within 24 hours of the onset of their symptoms have been examined and the temperatures, pulse rates, respiratory rates and blood pressures are shown in Table "S". If those patients whose pneumothorax was of only an hour or two's duration are taken out of the group, the figures do not differ markedly from those of the group as a whole.

The figures given for the blood pressure in each case must also not be regarded as accurate in relation to the period immediately following the incident. In many cases the pressure was not taken even immediately following admission, and I have no means of checking the time in relation to onset, or how many hours afterwards the blood pressure was taken.

I have found no relationship between the age of the patient and these pulse, temperature, respiratory rate /
rate and blood pressure figures, nor have I been able to deduce from my figures any observations of value in relation to the amount of lung collapsed or the suddenness with which a total collapse of the lung is brought about, though one would imagine that a condition of shock would be found to exist where the collapse was both sudden and complete.

The only case who had an elevation of temperature over 100°F. was a medical student (Case 65), for whom I was given clinical responsibility. He had a slight head cold for a few days prior to the incident, but as he was in the midst of sitting his "Finals", he had neglected this. On the morning of his last Oral Examination, he had been seized with a sudden pain in his chest following a bout of coughing. He managed to carry on until after the examination was over, when he was seen by me, and admitted to Dr. Hewat's Ward with a spontaneous pneumothorax. I was glad to be able to assure him as he lay in bed a few days later, that he had not only no underlying disease in the lung, but that he had been successful in satisfying the Examiners!

**SEDIMENTATION RATES.**

In the earlier series of publications on spontaneous pneumothorax this useful adjunct to the clinical
clinical assessment of activity of disease processes was not available, and the recent ones which mention its use are small in number.

Olbrechts (1930), noted that the sedimentation rate in four of his patients was not altered, and Perry (1939) cites Oechsli and Miles (1934), Rossel (1935), and Willis (1937), as also having patients with normal sedimentation rates, as well as three of his own. Hyde and Hyde (1948), found the rate normal in 22 patients and slightly raised in 8, and Dickie (1948), in 20 cases of spontaneous pneumothorax or pneumomediastinum found the sedimentation rate was not raised nor the leucocyte count, except in three who had a concurrent tracheo-bronchitis.

The method in use at the Royal Infirmary is a modification of Westergren's method. 0.4 ml. of 3.85 per cent sodium citrate is drawn into a special 2 ml. syringe which is fitted with stops on the piston handle. Blood is then withdrawn from a vein up to the stop at the 2 ml. mark, and this is set up in a vertical column in a graduated tube 200 mms. high. The fall in the erythrocytes is read off at the end of one hour. For ordinary ward work no correction is made for anaemia, nor for room temperature etc. Figures of between 5 and 8 mm. per hour are regarded as /
TABLE "T".

Sedimentation Rates in the Present Series.

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**NOTES:**

A. Bronchitis. Pneumococci ++ in sputum.
B. Admitted 6 weeks after onset. Dyspnoea ++ and ankle oedema.
C. Haemopneumothorax.
D. Bronchitis 18 months.
E. Past operative.
as being within normal limits.

In my material, the B.S.R. (E.S.R.) has been carried out on 49 patients. In 38 of these the sedimentation rate has been 8 mm. or less in the first hour. In only 3 cases has the rate been above 20 mm. in the hour, (Cases 7, 31 and 39). In two of these it is recorded that the patient was suffering from chronic bronchitis and emphysema and in the third that he had a small amount of fluid in the pleural space and he had had his pneumothorax for six weeks. During this time he carried on his work, and he was sent in to hospital when his dyspnoea had become very marked and his ankles had begun to swell. It is likely that he had a respiratory infection present as well.

In many of the patients that I have seen myself, there is often a history of a slight head cold for a day or two preceding the onset of the pneumothorax; in others again there is no such history. In the case histories that I have examined, a preceding respiratory infection is sometimes recorded as being present (15 cases) but since this is often not noted in taking a history, I do not feel justified in drawing any conclusions as to the aetiological importance of preceding head colds etc., in cases of /
of spontaneous pneumothorax.

Since the foregoing paragraphs in this section were originally drafted, however, I have had the opportunity of hearing Scadding (1950), give a lecture on bronchial obstruction. He illustrated cases which had, following a head cold, small areas of "consolidation - collapse" or "aspiration pneumonia" in the lungs, associated with areas of emphysema. This was of considerable interest to me in view of what I had already drafted out in my discussion on the "Mechanism of Pneumothorax" which follows in a later section of this Thesis.

I believe, however, that these figures that I have given Table "T", from this series of cases indicate that the sedimentation rate is not normally raised in benign spontaneous pneumothorax. If the rate is raised, some underlying cause for this should be sought, in the lung or elsewhere in the body.

PHYSICAL SIGNS.

The physical signs of pneumothorax depend to a large extent on the amount of air in the pleural cavity, and pressure exerted by it. They may vary from none at all, if the pneumothorax is small, to the "Classical" or "Textbook" picture.
In a case where the lung is more than 50 per cent collapsed, there is usually a bulging of the affected side of the chest, the interspaces between the ribs being expanded, and the chest wall is kept immobile in the position of full inspiration on that side. The trachea may be seen to be deviated to the unaffected side, and the sterno-mastoid muscle on the unaffected side may be more prominent than on the side of the lesion. The apex beat, if visible, may be seen to be deviated away from the normal position towards the unaffected side and may appear to the right of the sternum or in the axillary line. Cyanosis is seldom a feature, and there is no congestion of the neck veins except in a minority of cases. Cough is not prominent and is often entirely absent. In those cases where cough is noticeable there is often an associated coryza or tracheo-bronchitis, or it is of the dry, irritant type associated with an increased respiratory rate, similar to that occurring after exercise.

Palpation will confirm or establish many of the features already mentioned, and the diminished vocal fremitus on the affected side.

On percussion, the note over the pneumothorax is hyperresonant, and on the right, there may be a loss /
loss of liver dullness if this is the side affected and percussion over the area below the right costal margin may show the lower border of the liver to be pushed downwards.

Breath sounds over the affected side of the chest are either vesicular or distant bronchial, and it is probably the case in complete collapse that the breath sounds from the unaffected side are the ones heard, or the distant note from the trachea.

One further interesting phenomenon which is sometimes to be made out on auscultation, is the presence of curious "tinkling" accompaniments, not unlike the musical notes produced by the Japanese lampshades, made up of suspended strips of glass, which were popular in many Victorian households. These "tinkling" rales have a curious "far away" sound about them, or echoing quality, as if they were being heard in a large cavern or cathedral. In such cases the "Bruit d'Airain", "Coin Sound", "Bell Sound", or "Anvil Sound", may be found. This sound is usually said to be diagnostic of pneumothorax, though it may be also elicited in the case of a large cavity near the surface of the lung (Osler 1895).

THE ANVIL SOUND.

In order to produce this sound, one coin is placed /
placed on the chest wall and tapped with another, while the examiner listens with the stethoscope at another part of the chest wall on the same side. A musical note is produced, which is similar to the note made by a hammer striking an anvil. I have never found this sound to be present where the breath sounds or accompaniments did not also have this same musical quality, though in passing I can state that I have also heard it in cases where an artificial pneumoperitoneum has been introduced in the treatment of pulmonary tuberculosis.

Laennec (1819), has described these "tinkling" accompaniments though he does not describe the "Anvil Sound", and Hilton Fagge (1886), groups together "The metallic phenomena", but warns against ascribing undue importance to them in diagnosis. Osler (1895), reported their presence in a patient with a large, rigid-walled tuberculous cavity.

Though two coins are usually recommended for use in eliciting the sign - it has even been suggested that silver coins are preferable - two pieces of wood or similar hard objects may be used just as effectively.

This sign has been recorded as being present in nineteen cases in this series. In many more it has not /
not been mentioned as being present or absent, and in a few its absence has been noted. For its production certain special conditions are necessary, and it is often only a transitory feature. Perry (1939), noted the presence of this sign in seventeen out of his eighty-five patients.

The actual amount of air in the pleural space does not seem to have any direct effect on determining whether this sign will be present or not. I have found this sign present early after the onset of a pneumothorax, and disappearing within about forty-eight hours, during which time there was no appreciable difference in the size of the pneumothorax, nor was there for some time afterwards. The presence of the sign is not entirely dependent on the pressure within the pleural cavity, though my own impression is that it is more frequently met with in those cases where there is reason to suspect that the pressure is raised above that of the atmosphere. I have never found this sign to be present where the intrapleural pressure was less than atmospheric throughout the respiratory cycle, for example in cases where the collapsed lung is re-expanding, though Coope (1945) states it may be found even then.

A third factor may enter into the production of this /
this sign, and it may be that the rigidity of the walls of the cavity is important in the absorption of some of the overtones, a certain amount of rigidity of the structures being produced in those cases where the pressure is high. Rigidity may also be produced as a result of glands, fibrosis calcification or thickening of the pleura etc.

When pneumothorax occurs, there is an immediate partial collapse of the lung on the affected side, due to its elastic recoil, until the pressure in the pleural space is atmospheric, or until the tear in the pleura is sealed off. If the degree of collapse is small, and the tear seals itself off and is not forced open again by the increased pressure of muscular effort or coughing, it is unlikely that this sign will be present. If, on the other hand, the intrapleural pressure rises above that of the atmosphere, then such pressure will itself produce a rigidity of the collapsing lung and its overlying pleura. Part of this increased intrapleural pressure of gases in cases where the lung has collapsed to its full extent of elastic recoil at once and the tear is sealed off is possibly due to the rapid diffusion of carbon dioxide into the space from /
from the pleural capillaries. Carbon dioxide diffuses about twenty times as fast as oxygen, and so within an hour or two the volume of gas will be increased. Later, as oxygen is absorbed, equilibrium is reached. Pinner (1945), states that, in the presence of a pleural effusion where the pneumothorax is closed, the carbon dioxide is usually more than 9 per cent and the oxygen less than 0.25 per cent when equilibrium is reached. I believe that this may be a factor in the production of this sign when it is noted in the early stages, but rapidly disappears, as has been the case with some of the cases of pneumothorax I have studied clinically.

These observations however, are largely hypothetical on my part, and it is my intention to investigate the physical requirements for the production of this phenomenon more fully at a later date.

V. CLINICAL TYPES OF PNEUMOTHORAX.

CLOSED PNEUMOTHORAX.

The closed type of spontaneous pneumothorax is the commonest one encountered. In such cases the rent /
rent in the pleura becomes sealed off as the lung collapses, and no further collapse takes place. The air is then gradually absorbed from the pleural space and the lung re-expands. The time taken for the air to be absorbed depends on the amount of air present, and it may take from a few days to a few weeks. For an average degree of collapse, the time taken is usually from three to eight weeks, but it may be either longer or shorter than this. Recovery may be retarded by a further breakdown at the point of previous rupture, if the rate of expansion is faster than the healing process in the torn pleura, or if this part is subjected to sudden pressure changes, such as may be produced by bouts of coughing, or muscular exertion with the glottis closed.

The treatment of this type of spontaneous pneumothorax is by sedation in the acute stages, with morphine grs.\(\frac{1}{4}\) repeated if necessary, or even better, if there is a cough present, by heroin grs.\(\frac{1}{12}\), along with bed rest, and reassurance. Prinzmetal and Kountz (1935), suggest that the patient should be instructed to lie on the affected side, since the intrapleural pressure is more negative on the side that/
that is uppermost, but in many cases I have found that the patients are unwilling to do this, and are uncomfortable in this position until such time as the lung is already re-expanding, when there seems little point in insisting on it. From one to three weeks is usually long enough for the period of bed rest, but this may be shorter or longer, depending on the clinical condition of the patient. It is probably wise to limit within reason, the physical activity of the patient for about a year after the incident, though this is not imperative, and many of the patients in my material have resumed full activity after a short period of convalescence. It is certainly unwise to place such restrictions on the patient as to turn him into a chronic invalid or give him a respiratory neurosis, such as Schneider and Reissman (1945), found in not a few of the recruits they examined for Military Service. Respiratory exercises are sometimes of use, though it is doubtful if they do much to speed up the expansion of the lung, but they induce confidence in the patient, and to a certain extent ensure that the lung is capable of standing up to a full range of respiratory movements prior to the patient's discharge from hospital. They also aid in getting rid /
rid of mucus which may have accumulated in the bronchi, blocking these, and prolonging the period of expansion by the production of an absorption atelectasis in the lung.

OPEN PNEUMOTHORAX.

In this type of spontaneous pneumothorax, the intrapleural pressure is the same as that of the atmosphere, and a broncho-pleural fistula exists because of the failure of the tear in the pleura to seal off when the lung has collapsed. In many cases the communication is through a small bronchiole so that there is little passage of air between the pleural space and the outside atmosphere with quiet respiration. If, however, communication is made through a fairly wide bronchus, there may be quite a sizeable amount of air moving in and out of the pleural space with respiration, and there may be quite a large excursion of the mediastinum with each inspiratory and expiratory movement, and a paradoxical type of ventilation of the affected side of the chest may occur. In this paradoxical respiration, air is forced into the affected side of the chest on expiration and drawn out again on inspiration. The mediastinal swing or "flutter" produced /
produced by this abnormal movement of air in and out of the pleural sac, may be such as to endanger life, but as a rule, the opening is reduced in size as the lung collapses towards the hilum and the mediastinum is itself made more rigid by the support of the collapsed lung.

The general management of the patient in this variety of pneumothorax is similar to that of the closed type. Some authors have found that the introduction of more air into the pleural sac on the affected side to produce a more positive pressure, results in greatly lessening the distress of the patient, by preventing this mediastinal flutter and collapsing the lung down more quickly. This is probably a wise line of treatment if the distress is marked and the flutter pronounced. Usually, however, the patient settles quite quickly and the pneumothorax becomes the closed type, or the chronic variety. This does not happen in every case, however, and occasionally it happens that the communicating bronchiole closes down as the lung collapses to form a type of purse-string or slit valve, (Zavod, 1939). This valve remains closed as long as respiration is quiet, but if the intratracheal pressure should be suddenly raised, as happens with the
the expiratory phase of coughing, air is forced through the valvular opening into the pleural space, from whence it cannot escape backwards into the bronchial airway, and thus produces a tension pneumothorax, a variety which forms the subject of the next section of this Thesis.

If the fistula should not seal off and convert this variety of pneumothorax to the simple closed type, the pneumothorax may become chronic. This may be due to the presence of pleural adhesions keeping the opening patent, or to other causes. Three months is probably the maximum time to allow for the unaided re-expansion of the collapsed lung, Brock (1948), suggests less than this, though not a few re-expand spontaneously even after a longer period haselapsed since the incident. If no signs of recovery are seen by that time, the future treatment falls into the hands of the Thoracic Surgeon, and outwith the scope of this Thesis. Attempts at aspiration of the air may be made during this period, but these are usually unsuccessful, and further delay may lead to difficulty in re-expanding a lung that has been long collapsed, and is not justified nowadays, when a minor surgical procedure may restore full function within a day or two.

It /
It sometimes happens that a bronchus has become blocked with secretions, and what was originally a collapse of the lung due to air in the pleural space becomes the "absorption collapse" due to this blocked bronchus. I know of one case where an apparently chronic pneumothorax was cured by inverting the patient and percussing heavily over the chest. In this instance a plug of secretion was dislodged and coughed up, and the lung re-expanded successfully thereafter by itself without further treatment.

**TENSION OR VALVULAR PNEUMOTHORAX.**

This type of pneumothorax is of more serious moment than either of the foregoing two types. The slit-valve mechanism has been described in the previous section, and other valvular mechanisms have also been described by various authors.

Zavod (1939), describing a case in which a spontaneous pneumothorax appeared on the contralateral side following the induction of an artificial pneumothorax, has discussed these valvular mechanisms, and states that there are three main types described in the literature on this subject. The three types are:

(1) /
(1) The Flap Valve.
(2) The Slit Valve.
(3) The Purse String.

He was unable to find any accurate record in the literature of either the first or second types, but demonstrated a valvular mechanism in his autopsy material of the "Purse String" variety, which was open in one direction to the flow of air, but closed in the other to the passage of water. This description is open to some criticism, not only on purely physical grounds, but owing to the fact that it is almost, if not quite, impossible to reproduce after death, the conditions which exist during life. The description of a contracted bronchiole opening on to the pleural surface in a post-mortem specimen, is not necessarily a picture of what in fact was present when the lung was under hormonal and nervous control during life. Enlargement of the cross section of the bronchioles, however, occurs during inspiration (Macklin 1929), and contraction takes place during expiration, so that it would be possible for such a valvular opening to exist.

Kjaergaard (1935), is sometimes wrongly credited with having described a valvular mechanism to account for this type of pneumothorax, but a study of his original /
original paper makes it quite clear that by his description of the "valve vesicle" he only attempts to explain the rupture of a bulla by the raised pressure inside it which has been brought about by this valvular mechanism he describes. A pneumothorax is produced spontaneously thereby, but not necessarily of the tension variety.

Halliday Sutherland (1934), has described a "fluid valve" in a patient suffering from a tuberculous pyopneumothorax. In this report it is stated that the fluid in the pleural cavity rose on expiration, thus closing the valve, and on inspiration, the fluid fell, thus opening the valve and allowing air to enter the cavity. The assumption that more air entered the space after the pressure therein had already become atmospheric, infers that the pressure in the bronchial airways must be above atmospheric in the inspiratory phase of respiration for this to happen, whereas on inspiration, of course, the pressure in the respiratory passages can never be more than that of the outside air.

The statement that air flows from the lungs to the pleural space during inspiration to produce this valvular type of pneumothorax is made in many writings on this subject, and in not a few textbooks, (Norris /
(Norris and Landis, 1938: Chandler, 1939: Ellison and Carabelli, 1940: Price, 1946: Beaumont, 1948), and is quite definitely wrong. It is worth while at this point to demonstrate that this belief cannot be correct. The reasons are, as the Macklins (1944), have shown, as follows:

1. Air will not flow from a point of lower to one of higher pressure.
2. To produce spontaneous pneumothorax, air must flow from the lung to the pleural space.
3. Therefore, to produce pneumothorax, the pressure in the lung must be higher than in the pleural space.
4. Air in the lung can never be under greater than atmospheric pressure during inspiration, since the lung is open to the atmosphere.
5. Once the pressure in the pleural space has reached atmospheric, no more air can escape into it from the lung during inspiration.

Until such time, therefore, as the pressure in the pleural space is equal to that of the outside air /
air may escape from the lung into this space during inspiration. When this equilibrium has been reached, no more air will flow from lung to pleural space during this phase of respiration.

The argument may be carried further, and it is true to say,

(6) Air in the lungs may be under a pressure greater than atmospheric, during forceful expiration as in coughing, or in straining with the glottis closed.

(7) Therefore air under increased pressure in the lungs may flow into the pleural space during violent or forced expiration.

This fact is recognised in some of the more recent textbooks on Diseases of the Chest, but is worthy of wider recognition in general medicine.

In order that the pressure in the pleural sac should not return to atmospheric, which would be the case if a bronchopleural fistula was present, it is obvious that some sort of valvular mechanism must exist in the cases which have an intrapleural pressure which is above that of the atmosphere throughout the respiratory
respiratory cycle. I believe that it is possible for any of these types of valve I have mentioned to be produced.

There is, I suggest, another variety - a type of "sleeve valve" - which might be produced following interstitial or mediastinal emphysema, in which air could escape into the pleural space when the pressure in the lungs was raised, by a perivascular route through the interstitial tissues. Macklin (1937), has shown air tracking along the perivascular tissues of the lungs in his experimental animals, back towards the hilum and sometimes forming a pneumothorax by rupture of the thin mediastinal pleura. He failed, however, to make air, introduced into the pleural sac, travel in the opposite direction, that is from pleural space to mediastinum. Hamman (1945), also stated that this was also true in his experience. As far as I am aware, such a "sleeve valve" has not been generally recognised or mentioned in other publications on spontaneous pneumothorax.

That a valvular mechanism does exist, I think there can be little doubt, and I believe that any of the types which have been described may be the one responsible in any particular case. Its exact nature /
nature is of relatively minor importance.

The treatment of cases of tension or valvular pneumothorax should be directed to the relief of the positive pressure which exists in the pleural space, along with those measures advocated for the foregoing types already described. It may be sufficient in some cases to aspirate air from the sac with a two-way syringe on one or two occasions, or even, in an emergency, to insert a needle through an intercostal space and allow the air to escape. Alternatively, if a pneumothorax induction apparatus is available, this may be used in the reverse direction, and air withdrawn from the chest. One or several aspirations may be required, and even with these measures, the pleural sac may refill rapidly, though care should be taken not to remove too much air at the one time, since the valvular opening may be forced open by the induction of a negative pressure. Hennell and Steinberg (1939), are opposed to the aspiration of air in cases of tension pneumothorax, but they are in a minority, and it would seem to be unjustifiable not to attempt to reduce the pressure to that of the atmosphere, in cases where the greatly increased pressure is embarrassing both respiratory and cardio-vascular systems. Even if just /
FIGURE 8.

DRAINAGE OF A TENSION PNEUMOTHORAX.

Tension Pneumothorax. Illustration of the water seal connected up to the needle which is inserted through the intercostal space into the pleural sac. If the pressure of air in the pleural cavity rises above that of the atmosphere, it can escape out through the tube. Since the tube dips only 1 cm. into the water, it will escape from the pleural cavity if the pressure in the latter rises more than 1 cm. of water above atmospheric. Alternative sites for insertion of the needle are discussed in the text.
just enough air is removed to relieve the immediate distress of the patient, leaving a slightly positive pressure, the valve may seal itself off, and the patient is out of immediate danger. Komrower (1947), describes such a case with both sides affected where this measure was life saving.

In those instances where the pneumothorax requires frequent aspiration to prevent the pressure becoming unduly high, a needle may be left in the chest wall in the pleural sac to which a length of rubber tubing may be attached. The other end of this tube is led into a vessel containing water, which thus forms a water seal. This method has been mentioned by many authors as the method of choice for this type of pneumothorax, and the description of it has been equally brief. I have not come across a detailed description of this method, but there are one or two small points in its use which deserve attention.

The first point to note is that a fairly wide bore needle or cannula should be used for insertion into the pleural space. A fairly wide bore "Polythene" tube, which is comfortable and relatively non-irritant to the tissues may be introduced through a cannula instead, the cannula being withdrawn after
The tube is in situ. No advantage is to be gained by using a narrow gauge needle, since this may easily become blocked, and is not in any way more comfortable for the patient.

The position or site of introduction of the needle is usually a matter of choice for the physician. Some workers favour the second or third interspace on the front of the chest, the advantages of this position being that there is little chance of damaging any vital structures in this situation, and that there is less chance of the tube becoming blocked should any effusion develop. The advantages of a site in or about the sixth space in the axillary region, are that the development of a hydro- or haemopneumothorax may be noticed earlier, and appropriate steps taken to deal with this, and that it is less alarming for the patient if the site of introduction is not immediately visible to him.

Descriptions of this procedure usually end by saying that the end of the rubber tube is led into a Winchester flask or bucket filled with water into which it dips. If, however, the tube is led to the foot of the water container, the pressure in the pleural sac will have to displace the height of the column /
The glass tube dips one centimetre below the surface of the water. The rubber tubing is connected to the needle which has been inserted through the intercostal space on the affected side. The bottle stands on the floor beside the patient's bed.
column of water in the tube before any air can escape, and this height may easily be more than 200 mm. If, on the other hand, the tube is only allowed to dip in a matter of 1 cm., air will be free to escape from the cavity of the chest whenever the pressure rises above 10 mm. of water, so that no markedly raised pressure is possible. I have been in the habit of using a bottle with a relatively wide base, partly filled with water which is fitted with a rubber bung pierced with two holes. Through one of these holes a glass connection dips under the surface of the water to a depth of about 1 cm. The bottle stands on the floor beside the patient's bed, and when the pressure rises above 1 cm. of water, air bubbles out through the water from the end of the tube. The pressure in the thorax never becomes sufficiently negative to draw any water up more than a few cms. into the tube. In most medical wards a simple piece of apparatus such as this can easily be assembled to deal with the occasional emergency that requires it.

I have used this apparatus with considerable relief to the patient on more than one occasion. In Case 68, particularly, I believe that it was a life saving measure, and in Case 52, an elderly lady of 68/
68, it resulted in considerable relief of the more distressing symptoms. In this latter case, I was able to measure the amount of air expelled with each expiration, and I found that, during quiet respiration, this was approximately 5 ccs. When the patient coughed, however, the amount was about three or four times this amount, and I estimate that approximately the same amount was discharged by Case 68, though I did not actually measure it on this occasion. Nowack and Churchill (1931), report an instance in a man of 31 with a tension pneumothorax where an average of 3 ccs. was expelled per breath, but this rose to 10 ccs. on coughing.

Fuld (1944) has described how he has used the air inlet valve from an army transfusion set, connected by about eight inches of tubing to a blood transfusion needle for decompression in these cases. The needle is inserted through a rubber diaphragm, and then through the selected intercostal space. Air can then come out through this valve, but none is drawn in on inspiration. This apparatus is considerably less cumbersome than that which employs the waterseal, and I have constructed one, but so far I have not had the opportunity of using it, tension pneumothorax being a somewhat uncommon medical emergency. The disadvantages /
disadvantages of this particular device would seem to be the possibility of the valve becoming stuck or incompetent, though this is of relatively minor importance since it can easily be rectified, and the fact that it is not always easily available in the ordinary medical ward, for dealing with emergencies, though the valve from a Higginson's syringe can be used in a similar manner. I have tried other types of valves, such as that on the B.L.B. Oxygen Mask, but I have not been able to find a more satisfactory one, which combines easy availability in emergency with efficiency, and is comfortable for the patient.

When the tension pneumothorax is on the right side, the liver may be pushed down into the abdomen, and is often palpable below the rib margin. This was so in two of my cases (Cases 52 and 82). In the latter instance, the patient underwent a laparotomy, and the liver was observed to be depressed downwards. On retracting this organ, the right leaf of the diaphragm could be seen bulging downwards and moving paradoxically on respiration. (Falconer 1949 personal communication).

On the left side the diaphragm lacks the firm support of the liver, and paradoxical movement is more liable to occur. Laennec was aware of this bulging /
bulging of the diaphragm downwards, and Forbes (1827),
his translator says (p. 492):-

".... When the disease exists on the
left side of the chest, the muscle
is found considerably prominent
downwards; and when it is on the
right side, the liver is thrust
below the margin of the ribs".

The paradoxical movement of the diaphragm
(Kienboeck Phenomenon), in these cases has the effect
of making it a muscle of expiration instead of
inspiration. On inspiration the diaphragm on the
affected side, which is bulging into the abdominal
cavity, contracts, and this has the effect of in-
creasing the pressure in the pleural space, (von
Muralt 1922, Stivelman 1935, Christie 1936, Kaltreider
and Fray 1939, Ruggiero 1946).

If the pressure in the pleural cavity is only
sufficient to remove the normal concavity of the
diaphragm, the latter can no longer act as a muscle
of inspiration on that side, and the inspiratory
filling of the other lung in association with the
increased vertical tension on the mediastinum will
increase the pressure in the affected side of the
chest. Confirmation that this does indeed take
place /
place has been recorded by Maestrini (1929) and Joannides (1934). Studying cases of artificial pneumothorax that developed an effusion, Joannides observed in his patients a movement of one to four centimetres in the mediastinum towards the affected side on inspiration, and a rise in the fluid level in the pleural sac. Lilienthal and Amberson (1929), using a less negative pressure in cases of closed pneumothorax, found that inspiration or straining with a closed glottis, resulted in an immobile mediastinum, but that expiration caused a displacement of the mediastinum towards the healthy side. Variations in the intrapleural pressure alter markedly the effects produced as Christie's (1936) experiments have demonstrated. Using the rabbit as his experimental animal, which is suitable for this type of experiment because of the relatively stout mediastinum and lack of communication between the two pleural sacs, he showed that when a pneumothorax was produced on the right side and was gradually increased, the intrapleural pressure at first became steadily more near to atmospheric, and the fluctuations with respiration became greater, in the early stages. As the pressure became still greater inside the cavity the /
the fluctuation of pressure between inspiration and expiration became very much less. On repeating the experiment on the left side of the chest, the initial changes were the same as had been observed on the right, until a point was reached where the fluctuations with respiration almost disappeared, and on introducing more air, true paradoxical fluctuations appeared. The failure of paradoxical respiration to appear when the pneumothorax was on the right side, was due, he states, to the support to the hemidiaphragm on that side afforded by the liver, and it therefore could not bulge into the abdominal space in the way the unsupported left leaf could.

It is therefore true to say, that true paradoxical respiration will take place, at least on the left side, if the intrapleural pressure is sufficiently raised to obliterate the natural concavity of the diaphragm. Kaltreider and Fray (1939), have observed it on both sides in one case.

I have not had the opportunity of actually taking the intrapleural pressures in a case of tension pneumothorax, during the different phases of respiration, but I have observed a fixity of the lower margin of the liver, when this was palpable below the costal margin /
margin, and a higher pitch of the "Bell Sound" on maximum inspiration, which suggested that with the diaphragm in a fixed position, the increase in pressure was brought about by the straightening out in a vertical direction of the mediastinum.

**BILATERAL PNEUMOTHORAX.**

Bilateral pneumothorax, if both sides are affected simultaneously, may be a serious medical emergency, and is often fatal.

When the condition is recognised clinically, air should be gently, and if necessary repeatedly aspirated from one side of the chest, until it is possible to obtain apparatus for applying gentle constant suction. In not every case, however, is this necessary, and Case 97 in my material, is an example of this latter type. A young police constable was asleep in bed when his pneumothorax came on. He was admitted to the Royal Infirmary about twelve hours later, and was not markedly distressed. The signs of a pneumothorax were obvious on the right side of his chest, but the smaller one which was present on the left side, was only noticed after radiological examination. Photographs of the films taken /
taken at that time are shown in Figures 4 & 5 opposite pages 86 and 87 and it can be seen that the right lung is almost completely collapsed. In spite of this, I was able to carry out a spirometric test on him. He was quite comfortably lying at rest when I first saw him and did this, about two days after his admission. The air in the right pleural sac appeared to be under pressure, and the "Bell Sound" was present, but this disappeared after about four days. No treatment was given in this case, nor was it required. Other cases, however, may require decompression under a water seal, as described in the previous section, if the air is under considerable pressure in one of the pleural spaces, and at least one case has been described (Elte, 1938), where bilateral continuous suction drainage resulted in saving life.

The three cases in the present series with simultaneous bilateral pneumothorax (Cases 32, 72 and 97), have been mentioned in an earlier section. References in the literature to this type are also fairly common, and in some of these post-mortem examinations have shown a ruptured bulla in one lung to have been the cause, though no lesion was found to account for the contralateral pneumothorax. (Hayashi 1915, Le Wald 1931, Wilcox and Foster-Carter 1937, Priest /
Priest 1937, Hasney and Baum 1937, Hamman 1939).

The question now arises in these cases as to how the air has arrived in the second pleural cavity, when there is no apparent break in the continuity of the visceral pleura.

There are two schools of thought about the mechanism in these instances. The first believes that direct communication is established between the two pleural spaces via the mediastinum through a "weak spot" in this, and such is certainly the case in dogs, where the mediastinum is thin, the induction of a pneumothorax on one side leading almost immediately to the involvement of the other by this route. The second school believes that mediastinal emphysema is first produced, (which itself in most cases is preceded by interstitial emphysema of the lungs), and that the air in the mediastinum then burst through the mediastinal pleura, forming the pneumothoraces. Hamman (1945) states:-

"Air from the mediastinum frequently enters the pleural cavity, but that air from the pleural cavity does not enter the mediastinum".

Macklin (1937), has shown this to be true in some experimental animals at any rate.

My /
My own impression is that either explanation may be true in any particular case, and there is probably sufficient individual variation in human beings for some to behave as the experimental dogs and others as the experimental rabbits. In either case, one of the governing factors is almost certain to be the means whereby the initial pneumothorax arose, and the different theories about this are considered in a later section, when the mechanism is discussed.

Consideration has to be given, after the early critical stages have been dealt with, to the question of prophylactic treatment for these patients, lest a recurrence should take place in the future and prove fatal. It is probably wise to forestall that possibility by preventive measures, and if thoracoscopy does not reveal any lesion which requires major surgical treatment, it may be advisable to induce an artificial pleuritis, with a view to forming adhesions between the two layers of pleura. It is usually sufficient to do one side only.

A variety of different irritants have been used for this purpose, and all of them result in a fair amount of pain and systemic upset for the patient. Glucose solutions in varying concentrations have been used (Spengler, 1901; 1906; 1919; Kulcke, 1920; Brunner /
Brunner, 1921; Fogelberg and Wallgren, 1924; Harvey, 1938; Hennell and Steinberg, 1939). In a similar manner venous blood has been used (Watson and Robertson, 1928; Ruben, 1948), and varying strengths of Silver Nitrate from 0.5 per cent to 20 percent (Spengler, 1901; Kenner, 1932; Morlock, 1933; Brock, 1942; 1948). Plain or iodised oils are preferred by some workers (Hetherington and Spencer, 1947), and Brock (1948), states that he is planning to use a solution of Copper Sulphate.

Poudrage (the insufflation of iodised or sterile talc) has been employed by some workers (Bethune 1935, Steele 1947, Brock 1948), and was the method of choice in one case in my series. In this instance (Case 4), the patient gave a history of a previous pneumothorax on the other side. He was aged eighteen at the time of his second pneumothorax, and the thoracoscopy and poudrage were carried out by Mr. Walter Mercer. Nine years later, on follow-up, he informs me that he has had no further trouble with his chest, and in a letter he wrote me from Aberdeen where he was then in his fourth year as a medical student, he says:

"Two months after my discharge from Ward 22, I joined the army and served six years /
"years, three years in Tanks and three years in Paratroops. This may help to show that, not only have I had no trouble with my chest, but that I have enjoyed the rudest of health! .... I have been X-rayed three times. All the X-Rays have been negative".

**CHRONIC PNEUMOTHORAX.**

I have only one example of this in my material, (Case 66). In this case pneumonectomy was carried out and as I have stated earlier, I am of the opinion that the treatment of this type of pneumothorax falls rightly into the province of the Thoracic Surgeon. Other cases that I have come across in my studies of records in the Royal Infirmary, which have appeared to be chronic, have usually had fluid present in the pleural space or/ and underlying disease in the lung.

It sometimes happens that after the lung has collapsed because of a spontaneous pneumothorax, the bronchi become blocked with secretion and an absorption collapse or atelectasis is superimposed. I know of one such case, not in my series, where inversion /
"VANISHING LUNGS".

Photograph of the X-ray film of the chest of a patient with gross bullous emphysema. The lung markings are prominent at the right apical region, but are almost indistinguishable towards the bases. The outline of a bulla can just be distinguished at the left base. This condition is sometimes mistaken for a pneumothorax.
inversion of the patient and heavy percussion of the chest was sufficient to dislodge such a plug of secretion, the lung thereafter re-expanding without further interference. In cases such as this, the plug of mucus which originally blocked a large bronchus may be drawn down into the bronchial tree as the lung becomes atelectatic, so that bronchoscopic examination at a later date will not show any blockage of the main bronchi, the mucus plug having been broken up on its passage downwards into the smaller bronchioles. Occasionally in cases of bullous emphysema a large cyst or bulla may be mistaken for a chronic pneumothorax. A photograph of the X-Ray film of the chest of one such case is shown opposite, the appearances in the original film at the left base, with loss of lung marking, closely resembling a pneumothorax. Instances in which such a cyst occupied the whole of one side of the chest in a female aged 19, causing only minimal disability, is described by Cheyney and Garland (1938), and these cases with advanced bullous emphysema and large cysts often resembling pneumothorax have been described as having "vanishing lungs" by Burke (1937) and Ruben 1948).
VI. The Role of Emphysema in Spontaneous Pneumothorax.

Types of Emphysema.

One finding which is recorded in almost all autopsy reports on cases of spontaneous pneumothorax which have died, is that of emphysematous bullae, in the lungs, one of which may have been shown to have a tear in it which is presumed to be the cause of the pneumothorax. It is therefore fitting that some consideration should be given to the role of emphysema in spontaneous pneumothorax in the apparently healthy.

For this purpose, it is convenient to divide emphysema of the lungs into two main types, which Laennec (1819) claims to have been the first to distinguish. The two types are:— (1) Pulmonary or alveolar emphysema. (2) Interlobular or interstitial emphysema.

In a footnote to his translation of Laennec's description of the first type, Forbes (1827), however, gives the credit for an earlier description of it to Sir John Floyer (1698), who described it in a broken winded mare, but suggested the same causes were also operative in man and produced the same condition.
ALVEOLAR EMPHYSEMA.

Alveolar emphysema occurs chiefly in the older age groups, and it is usually preceded by a history of chronic bronchitis or asthma, and the consensus of opinion nowadays is that the primary cause is bronchial obstruction. This type of emphysema has been produced experimentally in animals by partial bronchial occlusion, and if the obstruction is kept up long enough, the changes in the lungs are permanent (Kountz, Alexander and Dowell, 1929). A few cases, however, have been reported in individuals who have suffered from neither (Kountz and Alexander, 1934; Christie, 1944), and it occurs in those acclimatised to high altitudes (Campbell, 1928; 1929; Hurtado, 1932; Christie, 1944).

If this type of emphysema is the underlying cause of benign spontaneous pneumothorax, it should not be uncommon to find records of the occurrence of pneumothorax in association with either asthma or bronchitis, or both. In fact, however, Perry (1939) could not find an autopsy record of a single case occurring in the London Hospital between the years 1924 and 1937, and could only find records of twelve cases with one or both in the literature up to that time. Faulkner and Wagner (1937) recording a fatal case of spontaneous /
spontaneous pneumothorax in an asthmatic remark on the infrequency of occurrence of these two conditions together. Castex and Mazzei (1938), recording two cases of asthma and spontaneous pneumothorax, point out also how rarely the two conditions are associated, and say in their final summary:

"La pneumothorax spontané est un rareté chez les asthmatiques. Les cas connus n'atteignent pas la douzaine."

Since that time a few additional isolated cases have been published (Harvey, 1938; Elliot, 1938; Field, 1943; Trowbridge, 1944), mostly in young persons, as were those of Castex and Mazzei (1938), but their number is small when it is remembered, as I have mentioned before, that Niehaus (1947) states that there were 873 cases of benign spontaneous pneumothorax recorded as occurring in the United States Forces in America in 1943.

In comparison with the large number of patients admitted to the wards of a general hospital such as the Royal Infirmary suffering from asthma or an exacerbation of their chronic bronchitis, the number of patients is extremely small who are admitted on account /
FIGURE 11.

LOSS OF ELASTICITY IN UPPER LOBE OF LUNG.

Case 100. Photograph of the right lung of this patient post-mortem. The lack of elasticity can be seen in the pleura covering the upper lobes, and emphysematous bullae can be made out towards the apex and along the anterior border. There are also a few smaller bullae at the antero-inferior angle of the lower lobe. The left lung was the one affected by the pneumothorax. At post-mortem it was found to be densely bound to the chest wall by adhesions. (See X-ray Fig. 1).
account of a spontaneous pneumothorax, and in only two cases in the present series was either of these conditions found to be present in association with the pneumothorax, or preceding it.

If generalised emphysema is to be considered as the underlying cause for benign spontaneous pneumothorax then it should be found in that age group in which this type of emphysema is most common. But it has been shown, both from the present series and from others recorded in the literature, that the age incidence of this type of pneumothorax is in the twenty to forty years group, the average being just below thirty years, whereas alveolar emphysema is usually not recognised clinically until the later period of life, after the age of forty.

A possible explanation of the relative rarity of spontaneous pneumothorax in persons who suffer from this type of emphysema, may be found if consideration is given to the fact that emphysema has been shown to result in a loss of elasticity in the lung tissue, with an associated loss of elasticity in the visceral pleura, and costal and vertebral cartilages. A photograph of the right lung of Case 100 is shown opposite. This patient had a left pneumothorax three /
EMPHYSEMATIC UPPER LOBE.

Case 100. Section from the upper lobe of the left lung shown in Fig. 11. There is marked emphysema, and the normal architecture is largely destroyed.

Case 100. Section of the lower lobe of the same lung illustrated above. There are open spaces in the lung, suggesting emphysema, but these are not so marked as in the upper lobe. There is also some collapse, congestion and a degree of pneumonia.
three years before at the age of forty-five. He was admitted again in 1950 with acute congestive heart failure, but with no pneumothorax, and died. At post-mortem examination the left lung was found fixed by adhesions to the chest wall. The right lung was inelastic, and the inelasticity of the visceral pleura can be seen in the photograph. Emphysematous bullae were found at the apex and along the anterior border, some of which can also be seen in the photograph. An X-ray of his chest taken two days before death is reproduced in Figure 1, and this shows the calcification in the costal cartilages, associated with the emphysema.

Christie (1934) has shown that his loss of pulmonary elasticity is associated with a characteristic spirometric tracing pattern. If the emphysematous patient inspires deeply, the succeeding expiration does not return to the previous respiratory level as it does in the normal individual. A new and higher level is established, or more characteristically, successive expirations are recorded extending progressively lower until the original level is reached. The inelasticity of the emphysematous lungs is further reflected in the tracing by the contrast /
contrast between a maximum expiration following quiet breathing, and the same following a maximum inspiration. With normally elastic lungs these are more or less equal; in the emphysematous subject the former is usually greater than the latter. From this it can be seen that the reserve air after a period of quiet breathing is greater than the amount following a maximum inspiration, and indeed it may sometimes happen that the reserve air is represented by a negative quantity.

A third feature of the spirometric record in emphysema is the irregularity of the respiratory level in quiet breathing. The explanation of this usually given is that it is attributable to the influence of voluntary muscles of expiration, normal expiration being a passive phase in healthy individuals, but an active one in the emphysematous subject (Scott, 1920; Christie, 1934).

These findings of Christie's (1934) have been confirmed by Cournand Richards and Darling (1939), Paine (1940) and Christie (1944). Cournand and his co-workers stress the retardation and prolongation of expiration as the characteristic feature of the spirometric changes in emphysema. This is the equivalent /
**FIGURE 14.**

**NORMAL SPIROMETRIC TRACING.**

**Case 21.** Spirometric record of a patient aged 20 at the time of his pneumothorax, taken two years after the incident. Vital capacity is 3,500 c.c. and there are no changes suggestive of diminished pulmonary elasticity. The base line of quiet breathing is straight and lacks the undulant character of that found in emphysema.

"C" Complemental Air.

"V.C." Vital Capacity.

"R" Reserve Air.
equivalent of the prolonged expiratory murmur, which is heard clinically in emphysema, and has been interpreted as the mediation by voluntary muscle action of the normal expiration. These facts are illustrated in Figures 14 and 15 on the opposite and following pages. Figure 14 shows the normal spirometric tracing (Case 21), and Figure 15 was taken from a patient, not in my material, who was suffering from emphysema.

In the same way as it was found impossible to have each of the patients in this series to report back to have a complete physical examination and X-ray of the chest, it was also found not possible to carry out spirometry on them. This was done in a few who were seen, but the number was small, and no conclusions could be drawn from this small number, particularly since they were seen on only one follow-up occasion. It is necessary to accustom them to breathing through the spirometer, as a rule on one or two occasions, if the results are to be regarded as accurate. In one case the patient had had a pneumonectomy for her pneumothorax and her record is reproduced in Figure 3. No gross evidence of emphysema is apparent in it, though early changes might be interpreted as being present. The record, it must be remembered, is the result of only one test on a patient who had no previous /
SPIROMETRIC TRACING IN EMPHYSEMA.

Case 404/31/3. Spirometric tracing of a patient with fairly advanced emphysema. Note the uneven line of the respiratory level during quiet respiration, the gradual return to the resting level following a maximum inspiration, the diminished ability to empty the chest after a maximum inspiration, and the prolongation of the expiratory phase of respiration.

"C" Complemental Air (maximum inspiration).

"V.C." Vital Capacity (maximum inspiration and expiration).

"R" Reserve Air (maximum expiration).
previous experience of using the apparatus. A photograph of the X-ray film of her chest is reproduced in Figure 2 opposite page 61. This X-ray was carried out at the same time as the spirometry, over six years after her pneumonectomy.

Associated with emphysema there are found, particularly at the apices and along the free anterior and inferior borders of the lungs, emphysematous bullae or blebs. These two names are used synonymously by some writers, though Miller (1934) correctly defines the bulla as a dilated portion of alveolar tissue, in contrast to the bleb, which may appear under the pleura without a covering of alveolar substance, and is the result usually of interstitial emphysema. Strictly speaking, therefore, those under consideration now are "bullae", and "blebs" will be considered under the section headed "Interstitial Emphysema."

Christie (1944) and other workers have pointed out that these bullae are to be found chiefly in those parts of the lung that are subject to the greatest strains and stresses. It is also well recognised that emphysema is more marked in the upper lung lobes than in the lower. That this is so can be seen from the photographs on the opposite and subsequent pages, which are taken from the lungs of one of the cases in /
Photograph of the lungs of a patient who died from a spontaneous pneumothorax. Emphysematous bullae are visible, notably at the apices and along the anterior borders, and to a lesser extent, at the inferior margins. The bullae, many of which were collapsed on opening the chest wall, have been re-inflated by the introduction of air through the bronchi. The pneumothorax was on the left side, and one of the apical bullae was found to have a tear in it.
in this series (Case 93). The microscopic view shows the emphysematous bullae concentrated at the apices of both lungs and along the anterior margins. These parts are less well supported by rigid structures, so that there is more opportunity for over-stretching and for the formation of such bullae. I believe that this is not the sole explanation for the localisation of bullae at these sites, for I have had the opportunity of observing, in association with Dr. K. Rhaney, that if the lungs of a still-born infant are placed in a sealed jar, with the trachea in communication with the atmosphere, and air is then withdrawn from the jar to create a partial vacuum, expansion takes place at the apices and free upper borders first, and that it is possible to form large bullous swellings in these regions if the pressure is made more sub-atmospheric in the jar. It is possible, among other explanations for this, that the weight of the lungs themselves may be a factor (Wirz, 1923; Rohrer, 1925; Christie and McIntosh, 1934), but further investigation is required and discussion of this subject and the formation of such bullae is outwith the scope of this Thesis.

The illustrations (Figs. 12 and 13) are sections of the upper and lower lung lobes magnified X5, and they also show the emphysema to be much more marked in /
Photograph of the lungs of a patient who died from a spontaneous pneumothorax. Emphysematous bullae are visible, notably at the apices and along the anterior borders, and to a lesser extent, at the inferior margins. The bullae, many of which were collapsed on opening the chest wall, have been re-inflated by the introduction of air through the bronchi. The pneumothorax was on the left side, and one of the apical bullae was found to have a tear in it.
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APICAL BULLAE.

Case 23. Section of the apical region of the lung, showing emphysematous bullae. The pleura is not thickened. (X 5).

FIGURE 17.

BASAL CONGESTION.

Case 23. Section of lower lobe of the same lung. Emphysema is much less marked. There is some terminal congestion, and no thickening of the pleura. (X 5.)
in the upper lobes than in the lower, as well as the presence of bullae.

While it is not unlikely that rupture of an emphysematous bullae may be the cause of the pneumothorax in many cases, it is certainly not found in every one. The lungs in this type of emphysema are, as a rule, distended, have diminished or absent elastic recoil, and the negative pressure within the thoracic cavity is less negative, that is, nearer atmospheric than normal (Kountz, Pearson and Koenig, 1932; Christie and McIntosh, 1934). In the absence of respiratory obstruction, therefore, that is, where a free communication exists between the bulla and the atmosphere through the bronchial airways, an emphysematous subject can put less strain on his lungs than a person whose lungs are normal can put on his, at least during the inspiratory phase of breathing. During forced expiration, such as in coughing or straining with the glottis closed, air may be forced into these emphysematous bullae, which I have shown exist in those sites where the lung lacks rigid support. In such a case, if the bulla can distend owing to this lack of support, it may then rupture, thus producing a pneumothorax. The only way it can distend further, however, is if part of the adjoining lung becomes collapsed
collapsed or atelectatic. The amount of collapse must be fairly large, however, in such a case, since the pores of Kohn will permit of a free exchange of air between adjoining alveoli if a complete segment of lung is not involved. This I think is one possible explanation of the pneumothorax which may be found where an inhaled foreign body, or a bronchial neoplasm has obstructed a large bronchus, but cannot be the case in those patients where no such evidence of collapse exists and in particular in young individuals who are apparently healthy. In brief, it may be said that rupture of a bulla is unlikely to occur in subjects who are emphysematous, because:

(a) The thoracic cavity is already completely filled with the lungs.

(b) It is in the position of full inspiration, and cannot expand further.

(c) Any increased intra-pulmonary pressure such as in coughing or straining with the glottis closed, is immediately transmitted to the intrapleural space, and pressures inside and outside the bulla are therefore equalised.

(d) The diminished elastic recoil of the emphysematous lungs not only results in a diminution of the amount of negative pressure, that is, the pressure /
pressure in the intrapleural space is always more near to that of the atmosphere than in the normal subject, but the emphysematous lung cannot recoil if a pneumothorax should occur, to the same extent as a healthy lung, because of this inelasticity.

One further point should be mentioned here, and that is that it has been found that the emphysematous individual is unable to hyperventilate on breathing carbon dioxide (Scott, 1920; Davies, Brow and Binger, 1925; Christie, 1934), or on exercising (Hurtado, Fray and McCann, 1933; Hurtado and Boller, 1933). This inability to hyperventilate must result in a diminution of the stress on the emphysematous lungs, and reduce the likelihood of a spontaneous rupture occurring.

To sum up these observations on the role of generalised emphysema in spontaneous pneumothorax, it can be said with conviction that, while spontaneous pneumothorax is known to occur in persons with evidence of this condition in their lungs, it is a comparatively rare complication when the emphysema is marked in extent. Neither in the records of cases in the literature, and in post-mortem examinations recorded in such cases, nor in the present series, has this type of emphysema been shown to be commonly present.
INTERSTITIAL EMPHYSEMA.

One other type of emphysema requires to be considered, which differs from that described in the previous section. The foregoing type is recognised as "intra-alveolar" emphysema, and in it the alveoli are stretched, thin-walled and atrophic, and may be in part destroyed, but the layers of interstitium are not separated. In the type now to be considered, the histological pattern is different, air being found in the interstitial tissues of the lung.

Sir John Floyer (1698) has been mentioned earlier as having described emphysema in a broken winded mare, and thought that investigations should be made into the similar condition in man. In his own words -

".... It ought to be considered how far the holding of breath in hysteric fits, or the violent coughing in lung catarrh may strain the bladders, and their muscular fibres, and thereby produce the same rupture, or dilatation, or hernia, as happens in the broken winded. ....... 'tis certain some injury is done to the ventiducts; the bladders are either broken, and admit air into the membranous interstices, or else /
else they are overdistended like a hernia in the peritoneum; and this will produce an inflation of the whole substance of the lungs, and that a continual compression of the air and blood vessels, which will produce a constant asthma."

Laennec (1819), however, claims the distinction of being the first to describe the interstitial type of emphysema, separating it from the alveolar type. Forbes's (1827) translation of his description of it reads as follows:

"The pulmonary or vesicular emphysema, as we have just seen, is a disease essentially chronic; that which I am now going to describe on the contrary is, in most cases, a real traumatic lesion almost suddenly produced. This is the emphysema admitted by surgeons; universally admitted, indeed, yet very little known according to its true anatomical characters. This is so much the case, that I do not know where an exact description of it, drawn from nature, is to be found."

The accuracy of Laennec's description of the condition that follows these introductory words makes interesting /
interesting reading, when we consider that even today interstitial emphysema is seldom diagnosed by physicians, unless it has extended into the subcutaneous tissues, very often in the supra-clavicular region, by which time it is recognised as "the type admitted by surgeons" according to Laennec, or "surgical emphysema" even today.

Macklin and Macklin (1944) have been able to demonstrate very convincingly in a series of experiments carried out over a twenty year period, a mechanism of production of this type of emphysema. They postulate two different types of alveoli as existing in the lungs. These are - (a) partitional alveoli; (b) marginal alveoli.

The partitional types are those alveoli which have their bases lying between alveoli, and the marginal types have their bases resting against other structures, bronchioles, blood vessels, connective tissue or pleura. Since pores exist (Van Allen, Lindskog and Richter, 1931; Macklin, 1934; 1935; 1936), (pores of Kohn) between the partitional types, air can move freely between them; but for air to escape from the marginal type, it must take its way through underlying connective tissue, chiefly along the course of blood vessels, and in their sheaths. Macklin (1937) has /
has shown experimentally by local over-inflation of a cat's lungs, that the air is thus forced into the interstitial tissues of the lung, and has demonstrated by serial micro-sections that the air then travels backward towards the hilum of the lung, along the line of the blood vessels, chiefly in the sheaths of the arteries, which are often collapsed by the pressure of air surrounding them. Further pressure of air introduced through the bronchus, resulted in the extension of the interstitial emphysema of the lungs into the mediastinum, and from thence up into the tissues of the neck, and down retroperitoneally into the abdomen (Macklin, 1939). The changes which took place in the area of lung tissue involved were observed also by the use of uroselectan and X-rays, the part being first shown in its normal state; then, after the introduction of the draught of air, it is shown to be first dilated, and then contracted. Evidence of damage to the tissues was observed histologically by the finding of a fibrinous exudate, often accompanied by blood cells, which had gathered in the alveoli. Towards the periphery, in addition to the sheaths of the vessels being filled with air bubbles,
bubbles, they were also found to be filled with fluid. A pressure gradient is thus created between the alveoli and distended sheaths. There was also evidence of a leakage of air through the visceral pleura, but no direct tear in this (cf. Brock, 1948), but a definite tear in the pleura overlying the mediastinum was sometimes demonstrated. Moreover, they were able to cause air to pass from the mediastinum into the pleural cavities, but were unable to make it pass in the reverse direction, into the mediastinum, when air was introduced experimentally into the pleural space under pressure. Reference has been made to this latter finding in a previous section of this Thesis (cf. Hamman, 1945).

How the air actually gets into the interstitial tissues of the lungs is explained by these workers by the postulation of two factors "A" and "B".

Briefly, factor "A" depends on the local over-inflation of the marginal alveoli, without a corresponding widening of the lumen of the blood vessels which they border. A pressure gradient is thus created between these two. This gradient does not exist between alveoli whose bases rest on bronchioles, and the underlying bronchiole, the pressure in these two being of course equal, since they /
they are connected either directly or indirectly through the communicating pores between the alveoli. (Van Allen, Lindskog and Richter, 1931; Macklin, 1934; 1935; 1936).

Factor "B" depends on a reduction of the calibre of the blood vessels in the lung without a corresponding diminution in the size of the alveoli. A pressure gradient is again created between the two, and air leaks into the interstitial tissues. Under certain circumstances, both of these factors may be operative, thus heightening the opportunity for air to escape into the tissues.

On the other hand, leakage of air may fail to take place in spite of over-inflation of the lungs if there is a corresponding increase in the size of the blood vessels. An example of this may be seen in the over-inflation which occurs in the other lung after massive collapse of one, or that which occurs in the remaining lung following pneumonectomy. In each of these examples, the blood supply is diverted from the non-functioning area to the uninvolved lung (Fine and Drinker, 1931; Christie and McIntosh, 1936; Monaldi, Ferretti and Constantine, 1938; Lindskog, 1939).

Some of the clinical conditions involving factor /
factor "A", in which a local over-inflation produces a pressure gradient, are atelectasis, obstructive laryngitis, foreign body in the bronchus, fibroid tuberculosis, and silicosis. In the presence of any condition causing sudden atelectasis, alveolar rupture may occur either as a result of inadequate support of the collapsed alveoli, or from compensatory over-distension of the adjoining alveoli.

Factor "B" is involved when there is a decrease in the blood flow through any portion of the lung. This may be brought about by lessened return of venous blood to the right heart, or by an obstruction to the pulmonary arteries, such as in the pulmonary embolism, and as these conditions are often associated with raised intra-alveolar pressure the pressure gradient is thus further increased.

Straining with the glottis closed is one of the methods whereby the intra-alveolar pressure is increased, and the blood circulating through the lungs reduced. I have observed this in a group of student volunteers by noting the reduction in the pulse volume after a few seconds, when they were asked to breathe out against the resistance of a column of mercury. Fuller details of this experimental work, which was undertaken to determine the comparative /
comparative intra-pulmonary pressures between males and females attempting to breathe out against resistance, appears in a later section of this Thesis. It is worth mentioning at this point that in the case of one of the students in this series, the increase in venous back pressure was sufficient to cause a spontaneous rupture of some of the smaller blood vessels at the root of the neck. This young man came to me the day following the test, somewhat alarmed about a "rash on his neck"; the "rash" was a number of petechial haemorrhages, mostly situated in the supraclavicular regions on each side of the neck, which were obviously a result of the increased venous pressure produced by the test. Unfortunately the rash faded rapidly before I was able to get a photograph of it. In Case 100 in this series similar haemorrhages developed terminally over the right palpebrum and neck.

Trauma to the chest wall is often associated with the subsequent developments of either interstitial emphysema, mediastinal emphysema, pneumothorax or a combination of the three. One case, which I have not included in my series, developed a spontaneous pneumothorax following a blow on the back of his chest received /
received at his work. The pneumothorax came on some hours after this blow, but there was no sign of any damage to the ribs, and not any obvious bruising to the chest wall. Similar cases have been reported in the literature (Tchertkoff, 1936; McGrath, 1944; Ruben, 1947). In these cases it is likely that the bruising of the lung tissue will weaken the alveolar bases, and allow air to escape from these into the perivascular tissues, rather than that the relatively tough pleura should be directly torn and the pneumothorax be the first incident in the series of events.

An interesting series of experiments has been carried out by Griffin (1941; 1942), on dogs. Instead of introducing air under pressure into the trachea, which it might be said in the experiments of Macklin, might by itself have caused the rupture, he produced a generalised over-inflation uncomplicated by an increased intra-tracheal pressure, by putting the dogs in a decompression chamber, with a tracheal tube extending to the outside air. When the pressure was lowered in the chamber, the dogs developed pulmonary interstitial emphysema, pneumothorax and subcutaneous emphysema. Although the pressure in the alveoli was never more than atmospheric, a relative increase was produced by the lowering of the pressure outside /
outside the chest wall. Polak and Adams (1932) found that the alveoli would withstand a great increase in pressure, provided the lungs could not over-expand, and they bandaged the chests of their dogs tightly to prevent the air which was blown in from ballooning out the lungs and chest wall, and found that under these circumstances the alveoli did not rupture even with relatively high pressures. A similar state of affairs probably exists when blast injuries to the chest are produced by sudden pressure changes.

Any condition which will produce atelectasis of a group of alveoli may result in the stretching and dilatation of the alveoli surrounding that area. If such an atelectasis is produced rapidly, it is more likely to result in the production of pulmonary interstitial emphysema, than if the atelectasis is more slow to develop. When the atelectasis develops slowly, as in the case of a bronchial carcinoma, compensatory changes take place by the elevation of the diaphragm on the affected side, falling in and fixation of the chest wall on that side, deviation of the mediastinal structures, and sometimes the development of an effusion. On the other hand, if the atelectasis is rapidly produced, such as happens after the aspiration of a foreign body into the bronchus,
**FIGURE 19.**

**INTERSTITIAL EMPHYSEMA.**

Section of the lungs of an infant showing air in the interstitium of the lung and under the pleura. (X 20).

(Section kindly lent to me by Dr. Agnes Macgregor).

**FIGURE 20.**

**INTERSTITIAL EMPHYSEMA.**

Another section from the lungs of an infant. Air is again seen in the interstices of the lung and under the pleura. The alveolar tissue is distended, and not compressed to the same extent as in Fig. 19 above. (X 20).

(Section kindly lent to me by Dr. Agnes Macgregor).
bronchus, or the development of a bronchopneumonia in infants, the bronchioles in such cases being blocked with a sticky secretion, or again in the case of the newborn where meconium, liquor amnii or other secretions may have been aspirated, the conditions are such as to favour the development of interstitial emphysema, subcutaneous emphysema, and pneumothorax. The amount of lung tissue collapsed need not be great for this to occur, and as the Macklins have pointed out, the alveoli affected by the overstretching are those whose bases lie along blood vessels, and may be situated anywhere in the lungs, and not only at the periphery. Silver (1939) describes a case in an infant and reviews the literature up to that date. Others have been recorded since then (Gumbiner and Cutler, 1941; Fisher, 1941; Smith and Bowser, 1942; Copley, 1946; Yudkin, 1947).

Through the kindness of Dr. Agnes Macgregor I was allowed to photograph and reproduce sections of the lung of an infant suffering from interstitial emphysema. These are shown in Figures 19 and 20. Air can be seen in the interstitial tissues of the lungs and also free under the pleura. I have not been /
been successful as yet in demonstrating air in the interstitial tissues of an adult in any of those cases which I have studied at post-mortem, but the Macklins (1944) have pointed out that special methods of fixing and staining are necessary to do so successfully. There is, however, a certain superficial similarity between the section from my Case 93 (Fig. 12) and the section lent to me by Dr. Macgregor. It is at present only a hypothesis on my part, though I am conducting further investigations into the subject, that the bullae in my section may be an end result of the interstitial emphysema illustrated in those sections of the lungs of an infant. Interstitial emphysema of the lungs may easily be missed if a post-mortem is carried out in the usual manner.

At an early stage of my follow-up investigations of the patients in my series, I attempted to find out how many had had whooping-cough in their youth. Many of them were uncertain, and many more had vague histories of respiratory illnesses which they were too young to remember in any detail, so I abandoned this line of investigation as being of insufficient accuracy to be of value. Scadding (1950), however, has shown areas of "collapse - consolidation" - "aspiration pneumonia", which have followed mild respiratory /
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respiratory infections.

If the Macklins' (1944) theory as to the method of production of interstitial emphysema is correct—and the proofs they advance are very convincing—what happens to the air once it has got into the interstitial tissue of the lung? One of these workers (1940) has shown in the excised lungs of a newly-killed calf that when air was introduced into the cannulated trachea under pressure it started to leak from the vascular sheaths at the hilum. When the lungs were distended to the maximum, air was found to be escaping from the hilar region as fast as it was being injected through the trachea. In his earlier studies (1934, 1939) air was consistently found along the vascular sheaths and not along the sheaths of the bronchioles, and air in sub-pleural blebs was considered to have come either from the rupture of sub-pleural alveoli or by dissection of venous sheaths as they run towards the pleura. In most of the experimental animals the air dissected its way along these channels to the mediastinum, where it was shown to be compressing the great vessels in the neck. This condition has been termed by Macklin (1940) "air-block", and reference is made to a similar /
similar finding described by Torrey and Grosh (1919) in patients dying from influenza and pneumonia. Once the air has reached the mediastinum it may travel up into the neck, down retroperitoneally into the abdomen, or into either pleural sac, and it is in this way that a pneumothorax may be produced. Berkley and Coffen (1919) also state that the vascular sheaths act as highways for air from the ruptured alveoli to the hilum, and demonstrated "air streaks" by X-rays. Sub-pleural blebs were also present, and these workers state that air escaped from the ruptured alveoli under the pleura, and owing to the resistance of the latter, could not get into the pleural cavity. Hence it traversed the vascular sheaths to the hilum, ruptured the mediastinum, causing pneumothorax, and escaped into the tissues of the jugulum causing subcutaneous emphysema. Hamman (1945), discussing mediastinal emphysema cites the work of Jehn and Nissen (1927), who showed in dogs that as long as the pressure of air in the mediastinum remained around zero, there were no symptoms. As the pressure of air in the mediastinum increased, the blood pressure fell, and apnoea, dyspnoea and cyanosis developed. The eyes protruded and air appeared in the subcutaneous tissues of the jugulum and the pleural space. At post-mortem /
post-mortem they were able to demonstrate strings of bubbles along the line of the great vessels in their experimental animals.

The question now arises as to whether this interstitial type of emphysema can ever be responsible for the production of spontaneous pneumothorax in the apparently healthy human being. This is discussed in the following section when the various explanations for the occurrence which have been advanced by different authors are considered.

VII. THE MECHANISM OF BENIGN SPONTANEOUS PNEUMOTHORAX.

POSSIBLE MECHANISMS.

Several theories of the mechanism involved in the production of simple spontaneous pneumothorax have been advanced by various writers on the subject, and reference has been made to some of these explanations in earlier parts of this Thesis. The most important of these are:

(1) Rupture of the pleura by a small sub-pleural tuberculous focus.

(2) Rupture of a small adhesion between the two layers of pleura, producing a tear in the visceral pleura.

(3) /
(3) Direct rupture of the visceral pleura, following sudden effort or due to a weakness in the pleura, congenital or acquired.

(4) Rupture of an emphysematous bulla or bleb the result of (a) generalised alveolar emphysema; (b) localised emphysema or scar tissue formation; (c) congenital cyst.

(5) The consequence of interstitial emphysema of the lungs, however produced.

RUPTURED TUBERCULOUS FOCUS.

Enough has been said in a previous section of this Thesis to make it clear that there is no longer any justification for postulating a hypothetical subpleural tuberculous focus rupturing into the cavity of the chest to explain the occurrence of a spontaneous pneumothorax in the apparently healthy. That spontaneous pneumothorax does occur as a complication of advanced pulmonary tuberculosis is, of course, an accepted fact, but so far as I am aware, no small subpleural focus has ever been shown to be the cause of benign spontaneous pneumothorax, and the absence of the clinical features associated with a tuberculous infection, negative skin tests, and failure to develop frank /
frank signs of tuberculosis on follow up, when there has been no alteration in the patient's mode of life, must make this an extremely rare cause of this type of pneumothorax, if indeed it ever is at all. The absence of any sign of tuberculous infection in those cases which come to autopsy, both in this series and in others which have been cited from the literature on the subject, and those cases where even a healed tuberculous lesion is found to be present, suggest that the pneumothorax in even such latter circumstances may be post hoc sed non propter hoc. In any case, it cannot be the explanation in a series such as this, in which no patient has apparently developed the disease subsequently.

Scar tissue has been noted at the apices of the lungs in cases which showed no evidence of pulmonary tuberculosis, and it is possible that some of the patients recorded as having a healed focus at one or other apical region may in fact have had a fibrosis due to other causes. If the Macklins' (1944) theory is accepted, the presence of scar tissue in this area will pave the way for local over-inflation of the lung, and a diminution of the local blood supply, which, according to these workers, are each separately or together pre-requisites for the production of interstitial /
interstitial emphysema.

I believe that the evidence I have produced from my own series of cases and from others recorded in the literature on this subject, cited in an earlier section of this Thesis, shows closely that minimal pulmonary tuberculosis is seldom if ever the cause of benign spontaneous pneumothorax.

RUPTURE OF ADHESIONS.

Rupture of adhesions between the two layers of the pleura have been thought to be the cause of spontaneous pneumothorax by some workers. Adhesions may form between the two layers of the pleura as a result of any irritation to the pleura, and were a conspicuous feature of Case 100 in this series. They can also be seen in the radiograph of Case 96 shown in Figure 6 and Figure 23.

Perry (1939) states that it has been suggested that fragile sub-pleural vesicles may be formed at the point of insertion to the visceral pleura of such adhesions, and that during violent respiratory effort, they pull on the lung and tear these vesicles. He points out, however, that if a pleural adhesion of sufficient size to cause a tear in the lung is ruptured, it will almost certainly bleed and give rise to a haemopneumothorax.
haemopneumothorax, and in most cases of benign spontaneous pneumothorax there is no more evidence of fluid in the pleural cavity than a slight blunting of the costophrenic angle. It is probably true to say also that in the majority of cases where there are adhesions, there is an associated thickening of the pleura, which will make it less liable to rupture than the normal membrane.

If there is a "leaky lung" or "porous pleura" as observed by Brock (1948) in the human subject, and earlier by Macklin (1937) in his experimental animals, it could happen that a small pneumothorax is first brought about by this means, leading to an increased strain on any adhesions present, during deep, or in the course of sudden inspiratory movements. Since in the majority of patients with a benign spontaneous pneumothorax there is no evidence of adhesions, neither on radiological examination nor on thoracoscopy nor in many of those cases that come to autopsy, rupture of adhesions cannot be the commonest cause. Moreover, it cannot explain those cases which came to post-mortem examination where there is no evidence of either adhesions or a tear in the continuity of the visceral pleura.

I conclude /
I conclude therefore that if this is the mechanism in some cases, it is not the commonest, since it fails to explain many cases where adhesions can be shown to be absent, and in those cases where a torn adhesion is demonstrated this may well be a secondary event in any case, due to such a mechanism as I have described.

**DIRECT RUPTURE OF THE PLEURA.**

West (1884) and later Emerson and Beeler (1923) have shown that the pleura is capable of withstanding considerable pressure. Macklin (1940) produced blebs under the surface of a calf's lungs by inflating them and found that, though the air was escaping rapidly from the hilum, the pleura remained unruptured though blebs might be formed.

In order to get over the difficulty of explaining how a healthy pleura comes to rupture under such circumstances, some writers have postulated a hypothetical "weak spot" in the pleura, of congenital origin, and suggest that these areas rupture and cause pneumothorax. (Ehrlich and Schomer, 1938; Kirshner, 1938; Lorge, 1940). Ehrlich and Schomer (1938), consider that some bronchioles terminate directly under the pleura, and that if this occurs in a part where /
where the pleura is congenitally weak, or "worn thin" that rupture will take place at this site. I have not come across any confirmation from other workers that bronchioles do actually terminate under the pleura, and as the Macklins (1944) point out, the areas of pleura worn thin by friction are hypothetical since friction, if anything, would tend to thicken the pleura. The suggestion that the pressure in these bronchioles terminating under the pleura is different from that in the alveoli is, of course incorrect.

In favour of a congenital or familial weakness of the pleura, cases of pneumothorax occurring in more than one member of a family have been recorded. These have been referred to earlier in this Thesis, and they are rare, and one would expect that there would be a marked tendency for the pneumothorax to be recurrent if a congenital cause was present. While a certain number of cases which have been recorded do have recurrences - the percentage in my material is as high as 27 and it varies between 5 and 30 in other series - a much larger number of recurrent cases might be expect if this congenitally thin pleura was the sole explanation for all the incidents. In association /
association with congenital cystic disease of the lung, pneumothoraces are frequently recurrent, but often associated with infection.

In view of Brock's (1948) finding of these porous areas of "cuckoo spit" in the pleura, it must be accepted that these are capable of causing some of the cases of spontaneous pneumothorax, or at least a proportion of those that become chronic. If it were the only or the commonest cause, the number of pneumothoraces which become chronic would be expected to be much higher than it is. I have noted in an earlier section, however, that in the majority of incidents the lung re-expands rapidly, which would be unlikely to happen if such areas were a persistent as well as a common feature of benign spontaneous pneumothorax. Brock hints that such areas may be due to a congenital (or possibly acquired) defect of quality that renders the pleura liable to rupture or leak easily: an alternative explanation is suggested in the next paragraph.

In discussing interstitial emphysema in the previous section, I have mentioned that it is not unlikely that this may be associated with those areas described by Brock (1948), and I believe that the local interstitial emphysema of the lungs, such as has been demonstrated by Macklin (1937), in animals
to be associated with the oozing of air through the pleura, combined with Brock's (1948) actual visualisation of such areas in the living human subjects, are facts and are therefore one acceptable explanation of the mechanism of production of a pneumothorax in the human subject. It is therefore unnecessary to invent any hypothetical areas of congenital or acquired pleural weaknesses to explain the occurrence of a pneumothorax in these cases if interstitial emphysema is present in the underlying lung.

RUPTURE OF AN EMPHYSEMATOUS BULLA.

It is interesting to note, that if a small pneumothorax is artificially produced in a patient suffering from emphysema, that it is not uncommon for the amount of air in the pleural space to become spontaneously much more than that which has been introduced in the production of the initial artificial pneumothorax. In other words, a spontaneous pneumothorax develops in addition to that which has been produced artificially. Instances of this happening have been frequently recorded, since Ganter (1926) first suggested that artificial pneumothorax might be used in the treatment of generalised /
generalised emphysema (Wiele, 1928; Christie, 1934; Paine, 1940). Paine (1940), studying pulmonary elasticity in patients with a history and clinical features of chronic bronchitis, asthma and emphysema, introduced a small amount of air into the pleural space of his subjects, and found that five out of eleven in whom this was carried out developed a spontaneous pneumothorax in addition. It has been noted that a spontaneous pneumothorax on the same or contra-lateral side and interstitial emphysema occasionally follows the induction of an artificial pneumothorax in patients suffering from pulmonary tuberculosis (Parfitt, 1937; Cutler, 1938; Andosca, 1938; Zavod, 1939). The development of a spontaneous pneumothorax following the induction of an artificial one in the treatment of tuberculosis, is not however very common if one is to judge from the number of cases which have been recorded, but is certainly nowhere near this figure in Paine's (1940) emphysematous subjects of almost 50 per cent. Andosca (1938) gives an incidence of two in over 34,700 punctures of the pleura, and states that there are only five recorded in the literature up to that time.

One explanation of the high incidence of this complication /
complication in Paine's cases, may be that the lack of firm support from the chest wall to the underlying emphysematous lung, in association with the increase in the range of the intra-pleural pressure that occurs when a pneumothorax is induced in a subject suffering from emphysema (Christie, 1934) may lead to local over-distension of the lung. The part which is most likely to be affected by these two factors is one which also lacks the support of the alveolar network of the lung, that is, a sub-pleural bulla. If a small pneumothorax arises in an emphysematous subject, no matter how it is originally produced, it will allow those two factors to become operative, and if a ruptured bulla is found at autopsy this, instead of being the cause of the pneumothorax, may be only a secondary contributor to the air in the pleural space. The fact which has been noted at post-mortem examinations on more than one occasion, that emphysematous bullae were present under the pleura, though none were found to be torn, proves that the pneumothorax in these cases at any rate was not caused by the rupture of such a bulla (Le Wald, 1931; Kjaergaard, 1933; Wilcox and Foster-Carter, 1937; Priest, 1937; Hasney and Baum, 1937; Lorge, 1940; King and Benson, 1944; Brock, 1948; and Case A'
Case A in the present series, recorded as an addendum in the section on Post-Mortem Reports, and Case 55).

Briefly then, if a small pneumothorax is produced or arises in any manner, sub-pleural emphysematous bullae lacking the support of the chest wall will be more liable to become over-distended and ruptured, and this rupture is therefore a result or consequence of the initial pneumothorax and not necessarily the primary cause.

LOCALISED EMPHYSEMA OR SCAR TISSUE.

From the time when it became recognised that the occurrence of a spontaneous pneumothorax could no longer be attributed to occult tuberculosis, dating in the main from Kjaergaard's (1932) monograph on the subject, it has been suggested by many writers that the incident had been caused by the rupture of an area of localised emphysema or of scar tissue formation. This assumption has largely been founded on the statements made by Kjaergaard (1932; 1933), who cites the earlier pathological studies of Fischer (1922), and his pupil Hayashi (1915), in support of his own deductions. Since references to Kjaergaard's work are in some instances, at least, quoted second-hand, /
secondhand, and not the result of a study of the original publication, it is of value to consider what in fact Kjaergaard did demonstrate.

Kjaergaard's (1932) explanation of a spontaneous pneumothorax in a healthy person is that it occurs as the result of the rupture of an emphysematous or scar tissue or congenital vesicle which incorporates a valve in its structure. This valve will permit of the entry of air into the vesicle, but will not allow it to pass out in the reverse direction, and as a result of increased intra-pulmonary pressure such as is produced by coughing or exertion, the vesicle is gradually distended.

I have not had the opportunity of studying the original reports of Hayashi (1915) and Fischer (1922), but they are described in some detail by Kjaergaard (1932). Kjaergaard himself describes valve vesicles in three patients (1932), who had no pneumothorax but died from other causes, and (1933) in a further two patients who, dying from other causes, were found at post-mortem to have pneumothoraces.

On examining Kjaergaard's recorded cases and his discussion of them, I find that he states as his opinion that pneumothorax simplex is due to the rupture of a valve vesicle on the surface of the lung, and /
and that (1933) - "The vesicle may be either a scar tissue vesicle, or an emphysematous valve vesicle (lung cyst)."

Criticism may reasonably be made regarding these claims for such a mechanism in benign spontaneous pneumothorax if we consider the following points about the cases he records.

(1) In two of his (1932) cases he was unable to demonstrate at autopsy that there was any connection between the vesicle and any underlying bronchiole.

(2) In one of his (1933) cases no connection could be shown to exist between the tear in the valve vesicle and the bronchial airways.

(3) In Case 2 (1933), although he demonstrated that air could be made to pass through the bronchial tree and come out at the site of rupture in the "valve" vesicle, he did not demonstrate the inability of the air to pass in the reverse direction. This would have helped to establish the valvular nature of the opening.

(4) All his five cases were suffering from a terminal broncho-pneumonia, and this can scarcely be ignored in arguing in favour of a similar mechanism as the cause in healthy persons.

(5) /
Only one of his subjects was in the younger age groups, a boy of twenty-four years of age. In addition to his pneumothorax, he also had diabetes mellitus, chronic bronchitis, broncho-pneumonia, bronchiectasis, lung abscesses and empyema.

The ages of the other patients were 61, 65, 79, and 82 years. They are therefore considerably older than the average patient who suffers from a spontaneous pneumothorax, even if their terminal broncho-pneumonia is disregarded.

Three patients, aged 61, 65 and 79 years, are considered to have "congenital" cysts with valvular openings. In view of their age, however, it seems unlikely that these patients in this last category, past the most active period of their lives, should have "congenital" cysts, in which a valvular opening was demonstrated, without having symptoms of pneumothorax at an earlier age.

However carefully the post-mortem section of lung is prepared, it may happen that the appearance of a flap of tissue/seems to form the tongue of a valve, is the result of an artefact. The sections of my Case 93 (Figure 17), show in some parts, flaps of tissue, which in serial section or fixed in a slightly different way, might have appeared to form the tongue of a valve.
For these reasons I am unable to accept that a valvular mechanism of this nature has been convincingly shown to exist in these emphysematous bullae. Though I cannot exclude the possibility that this may be the cause of a spontaneous pneumothorax occasionally, I do not believe that it can be assumed to be the cause of benign spontaneous pneumothorax in healthy persons, and particularly in the younger age group in whom the incidence is highest, as I have shown.

The failure of some bullae to collapse after death even when pressure was applied to them, is remarked on by Laennec (1819); and noted again by Kjaergaard (1932). Kjaergaard's interpretation of this is that it is due to a valvular mechanism, and that these are "valve" vesicles. Another explanation may be that those vesicles from which he was unable to express the air or in which to demonstrate a connection with an underlying bronchiole, may have been in fact, in the interstitial tissues of the lung. I therefore suggest that a fourth type of valvular communication can exist between the air in the lungs and these vesicles, which might be described as a "sleeve valve", in which the communication between the vesicle and the alveolar air lies along the sheaths/
sheaths of the blood vessels. In the section of lung tissue from my Case 93 (Figure 17), blood vessels can be seen adjoining the bullae and traversing it, and I believe that if air can dissect along the sheaths of these vessels, such as I have shown does happen in interstitial emphysema, valve vesicles of a different type from those described by Kjaergaard (1932, 1933), will be formed. Kjaergaard's pathological descriptions are excellent and complete. A study of the photographs of his histological sections and his description of these, shows the presence of blood vessels at the base of the vesicle or in the wall of it, in four out of the five cases which he describes. In the fifth case he does not mention the blood vessels at the base of the vesicle (Case "C", 1932), but these can be seen in the photograph of the section which he reproduces, surrounded by air spaces. Kjaergaard's own description of the microscopic findings in one of his cases (Case "B", 1932), is as follows:

"Examination of a number of sections from the largest vesicle, shows, .... that the vesicle is resting on a base of massive scar tissue in which there are no remnants of the original alveolar /
alveolar structures. The scar tissue contains some branching blood vessels, and a little below the vesicle is a small bronchus, but no direct communication is found between this and the vesicle. In some sections one finds in the periphery of the base, immediately below the vesicle, a small area containing some flattened, hollow spaces, but, unfortunately, this communication cannot be demonstrated in any of the sections examined.

"The other vesicle is a little broader and less protruding. The base of the vesicle is formed in part by markedly emphysematous lung tissue, and partly by greater or smaller streaks of connective tissue. On the microphotograph ... one will notice an area where these streaks of connective tissue are rather thin, while the base of the vesicle is composed of a system of membranes enclosing flattened hollow spaces of varying size which are presumably communicating /
"communicating with the lumen of the vesicle in such a way as to produce the function of a valve mechanism. The structures of this tissue are so entangled, however, that it is impossible to have any clear idea as to how this valve mechanism is built".

Kjaergaard has attempted in each of his cases to establish a valvular connection composed of strands of tissue between the vesicle and an underlying bronchiole. He admits himself that he has not succeeded in doing this in every case. He has, however, shown the presence of blood vessels lying in dense or reticular fibrous tissue, in close proximity to, or traversing the vesicle, and in some instances surrounded by clear spaces, which may have contained air. It is my suggestion that the air in these vesicles has not come direct from an underlying bronchiole, but indirectly through the interstitial tissues of the lung. I believe therefore, that his observations are an accurate picture of the histological appearances to be seen in these cases, but the conclusion that he arrives at from these observations, namely that a flap of tissue forms a valvular connection between the vesicle and an underlying bronchiole, is possibly incorrect, and I consider that the explanation I have offered /
offered is more likely to be true in the majority of cases.

Comparison of Figures 19 and 20 which show acute interstitial emphysema in the newborn, and Figure 17 which shows emphysematous bullae in Case 93 in my series, suggest to me that the former illustration may represent the acute stage of a condition which has become chronic in the latter. Should these vesicles or bullae remain in free communication with a bronchiole, they are unlikely to rupture, so long as they have the support of the chest wall. Should they not be in free communication with a bronchiole, one of three things is likely to happen:–

(a) The air will be absorbed from them and they will become areas of fibrous tissue.

(b) They may remain in communication with the surrounding alveoli, through the collateral air circulation (alveolar pores of Kohn), and remain expanded.

(c) They may remain expanded through the "sleeve valve" mechanism with the underlying bronchiole, alveolar air being forced in when the pressure rises as in forced expiration (e.g. cough).

It /
It would appear to be reasonable to assume therefore, that if more air is introduced into a vesicle which is not in free communication with the surrounding alveoli, or bronchial airways, it will first increase in size and then rupture either into the bronchial airways, or, less commonly because of the protection of the pleura which is liable to withstand pressures of up to 200 mm. of mercury (West 1884) into the pleural space. The other route of escape is along the vessel sheaths to the hilum and mediastinum.

How then can more air be introduced into a vesicle not in free communication with the bronchial tree? My explanation of this is that it is the result of this "sleeve valve" mechanism or the occurrence of further interstitial emphysema, the air being introduced into the vesicles along the sheaths of blood vessels in their bases. This I believe explains Kjaergaard's inability to demonstrate a connection between some of his vesicles and any underlying bronchiole, but is compatible with his histological observations of the presence of air spaces in which the blood vessels lie, and with the fibrosis, and the emphysematous or compressed alveoli, which he noted around the vesicle.
Other writers have drawn attention to the fact that these cystic swellings are often not in direct connection with the bronchial airways. Wilcox and Foster-Carter (1937), record a case in which a sub-pleural bulla was diagnosed during life. At post-mortem examination no connection could be shown to exist between it and any bronchus. Gordon (1936), records five cases of benign spontaneous pneumothorax. In two of these Lipiodal examination was carried out, but the dye could not be made to enter some of the bullae. Gordon suggests that the cysts are either congenital or formed in connection with scar tissue. It is just as likely in my opinion that they are the result of interstitial emphysema. Oswald and Parkinson (1949), describing 16 patients with "honeycomb lungs" eight of whom had spontaneous pneumothoraces were also unable to get Lipiodal to enter some of the cystic spaces, in the four patients in whom this examination was carried out.

CONGENITAL CYSTS.

The difficulty of differentiating between cysts which are congenital in origin and those which are acquired has been stressed by many authors, and it is outwith the scope of this Thesis to discuss the various theories and arguments advanced by the many /
many different writers on the subject. (Koontz, 1925; Schenk, 1937; Sellors, 1938; Kaltreider and Fray, 1939; Maier, 1941; Stanford and Nalle, 1942; Willis and Almeyda, 1943; Korol, 1947). It is sufficient in the present study to consider these as cystic areas in the lungs, either in communication with the bronchial tree or separate from it, and the mechanism whereby a spontaneous pneumothorax is brought about must be similar to that discussed in the two preceding sections.

There is no reason to suppose that congenital cystic disease of the lung can be the cause of a spontaneous pneumothorax in healthy persons except on rare occasions. No clinical or radiological evidence of this condition was found in any of the patients in the present series, and the only other case that I came across and studied had recurrent pneumothoraces, and the cysts were grossly infected. In the occasional case of spontaneous pneumothorax that comes to autopsy, and that has been reported in the literature, few instances have been recorded where there was reason to suspect the presence of a congenital cystic condition of the lungs. Some of these recorded have been in children or infants, and may have /
have been the result of atelectasis elsewhere in the lung. It is very difficult to be certain of the congenital nature of these cysts; Kaltreider and Fray (1939), and Ruben (1948), among other writers, have drawn attention to the difficulty of differentiating the congenital from the acquired variety.

One further point against the theory that pneumothorax in the apparently healthy may be due to a congenital cyst, is the fact which I have demonstrated, that the average age incidence of this type of pneumothorax is around thirty years. If a congenital defect was commonly present in these patients' lungs, it might be expected to manifest itself at an earlier age, and not at a time when most people have modified their more strenuous forms of physical activity.

I cannot exclude the possibility that a congenital cyst may be the cause of a benign spontaneous pneumothorax occasionally, but it has certainly not been convincingly shown to be the explanation for the incident in more than a few cases.

**INTERSTITIAL EMPHYSEMA.**

Much has already been said in the foregoing sections /
sections of this Thesis about the cause and effect of interstitial emphysema in relation to spontaneous pneumothorax, and to the other conditions in the lung which have been considered at one time or another, as possible underlying or predisposing causes for the incident. In this present section, I do not intend to repeat what has been said before, but rather to summarise these causes and effects, and consider how interstitial emphysema, which I consider to be the prime factor, might arise as it were *de novo* in the healthy person, and relate it to occurrence of a spontaneous pneumothorax in my cases.

I have already suggested that interstitial emphysema may be superimposed on any of the lung conditions that have already been studied in the last few sections of this Thesis. To recapitulate very briefly, the suggested predisposing factors that have been mentioned, and that have in the past been advanced as the explanation of the incident, we may take first the theory of occult or latent tuberculosis.

Healed tuberculosis will produce an area of scar tissue in the lung, and around this it is not uncommon to find evidence of localised emphysema. There is, therefore, an area of local overdistension of the lung alveolar tissue which is distorted and in which there may not exist, because of the surrounding fibrosis, free /
free communication with adjoining alveoli through the alveolar pores. As a result of a sudden increase of intrapulmonary pressure, such as follows a bout of coughing or straining with the glottis closed, the alveolar bases will be liable to rupture, and allow air to leak into the interstitial tissues. Macklin (1939), has been able to demonstrate tears in the alveolar bases, and shown the air in the interstitial tissues. Marcotte, Adams, Phillips and Livingstone (1940), in dogs and cats, produced experimentally interstitial emphysema with as low an intrabronchial pressure in the former as 24 mm. of mercury, and in the latter of 16 to 20 mm. of mercury. I have shown in an experiment detailed in the next section, that the human subject can raise a considerably higher intrabronchial pressure in forcible expiration against resistance.

In a small exudative tuberculous lesion, the same conditions may hold good as in a similar area of fibrosis, if the interveolar pores are blocked with sticky exudate. Small areas of atelectasis may be caused by the tuberculous process, thus initiating the chain of events which will lead to the production of interstitial emphysema. It might therefore be thought that this should lead to the frequent production of a spontaneous pneumothorax in these cases. It is not improbable that it does do so occasionally, and/
and I believe that this is maybe the explanation in those subjects who develop frank pulmonary tuberculosis at a later date. Reference has already been made to such cases in the section of this Thesis dealing with tuberculosis. That it is not more commonly found in cases with an early or occult tuberculosis lesion is possibly due to at least two causes:—

Firstly: It has been shown (Macklin, 1937, 1939; Marcotte et al., 1940), that the air in the interstitial tissues and vessel sheaths, travels towards the hilar regions, and Macklin (1937), has shown that the peripheral parts of the interstitium are filled with a fluid exudate, so that fluid rather than air might be expected to be found under the pleura in that region.

Secondly: There is a pleural reaction to the underlying infective process, the result of which is the thickening of the pleura in the area overlying the lesion, and the formation of adhesions between the two layers, visceral and parietal. In the three of Ornstein and Lercher's (1942), fifty-eight cases that subsequently developed pulmonary tuberculosis, the presence of adhesions between the two layers of the pleura was noted at the time of the spontaneous pneumothorax, but signs of tuberculosis did not appear /
appear till about two years later. The absence of signs of infection of the pleural space might suggest that the air may have reached the pleural space in these and similar cases indirectly through some other route such as the mediastinal pleura, rather than by a direct rupture at the site of infection.

The follow-up record of the cases in this series, and of other series to which reference has been made earlier, the frequently noted absence of signs of tuberculosis in those cases that come to autopsy, the fact that the incidence of tuberculosis in subjects who have had a spontaneous pneumothorax is not higher than in the general population, suggest to me that a mechanism may be operative in individuals with a minimal tuberculous lesion in the lung, similar to that in those who at no time show signs of tuberculosis. I believe that the explanation I have given is in accordance with the observed facts in these cases, and that interstitial emphysema may be the common denominator in the majority of cases of spontaneous pneumothorax, even if minimal tuberculosis is also present.

Rupture of a small adhesion between the two layers of the pleura, as I have already mentioned, has been suggested as the cause of the pneumothorax.
Pleural irritation must have preceded the formation of this adhesion and the very presence of it suggests an underlying lesion in the lung. The absence of adhesions is frequently noted in cases of benign spontaneous pneumothorax and it cannot therefore be the case that the tearing of these is the commonest cause of the incident. The pros and cons of the torn adhesion theory have already been discussed, and it is possible, as Macklin and Macklin (1944), point out, that the "stitch" in the side, which sometimes comes on after exercise, relieved by compression of the chest over the area, and upward compression of the lung by bending forward and to the side on which the pain is felt, may be indicative of the development of interstitial emphysema, which may be followed later by a spread to the mediastinum and pleural space. If a small pneumothorax is thus produced, there will be traction on any adhesions, most marked on inspiration, since the lung will have collapsed away from the chest wall, and these may then rupture and tear the visceral pleura. Air will then enter direct from the bronchial airways through this tear in the visceral pleura into the pleural sac, with the production of the symptoms and signs of pneumothorax and at post-mortem the tear is considered as the primary cause. As the lung collapses, the rent in the visceral pleura will gradually /
gradually become sealed off as it does in most instances, and the tear in the alveolar bases which initiated the train of events, will also have a chance to become healed at the same time. Failure to expand on the part of the lung will result either from the tear in the visceral pleura not closing owing to the persistence of an adhesion which prevents the lung from collapsing completely (Brock, 1948), or to the continuation of the interstitial emphysema for the same reason. In either case the treatment is the same, that is, to divide the adhesion.

It is unnecessary for me to repeat what I have already said about the relationship of emphysematous bullae and cysts to the development of spontaneous pneumothorax, except to summarise what I believe to be the reasons why spontaneous pneumothorax is relatively uncommon in subjects suffering from generalised alveolar emphysema. Briefly these are:-

(1) The **loss of pulmonary elasticity in generalised emphysema**, results in a lessened tendency for the lungs to recoil from the chest wall.

(2) The thorax is in most cases, usually in the position of maximum, or near maximum inspiration. There is diminished respiratory movement, and the /
the lungs fill the thoracic cavity completely.

(3) There is usually a free communication between the adjoining dilated alveoli, without areas of collapse to cause any local hyperinflation.

(4) The stresses and strains which an emphysematous subject can bring to bear on his lungs, are less than those which the normal healthy individual can bring to bear on his.

(5) Any increase in the intra-pulmonary pressure such as occurs with coughing or straining is immediately transmitted to the pleural space, and the pressure inside and outside the visceral pleura are therefore equalised.

(6) There is an increased pressure of blood in the pulmonary circulation and therefore there is less opportunity for a pressure gradient to develop between the alveoli and the underlying blood vessels (Macklin's, 1937 Factor "B"). Interstitial emphysema is therefore less likely to occur in such cases.

Having /
Having considered the various theories that have formerly been advanced to account for the occurrence of a benign spontaneous pneumothorax, it remains to consider if there is any explanation which will fit the observed facts in the cases in this series, and in those others from the literature that have been studied. Is there, in other words, any explanation of the occurrence which can supplant or modify the theory of the rupture of a subpleural bulla or bleb, which has been postulated for the past twenty years or more?

The rupture of a bulla or bleb cannot be the explanation in those cases which come to autopsy, and in whom it is impossible to demonstrate any bulla which has ruptured, or indeed, any leak or tear in the visceral pleura overlying the lungs. Reference has already been made to such cases recorded in the literature, in earlier sections of this Thesis. If we accept the work of Macklin and his colleagues however, the explanation in such cases is that interstitial emphysema has been first produced, and has travelled along the vessel sheaths to the hilum, and from thence has ruptured into the pleural space. It may, and does, also travel outwards to form a "bleb" under the pleura at least experimentally. The frequent /
frequent finding in children with broncho-pneumonia, whooping-cough or other obstruction to the bronchial airways, of subcutaneous emphysema, above the clavicles, extending in some cases over the chest and down to the hands, associated with interstitial emphysema of the lungs, pneumomediastinum and pneumothorax, suggests that a similar mechanism may be operative in the adult, even if less extensive in its clinical manifestations. The fact that subcutaneous emphysema is not so common in adults in association with pneumothorax, is probably due to the fact that the mediastinal structures are more firmly bound together than they are in the child and the air therefore takes the path of least resistance and goes directly through the mediastinal pleura into the pleural sac. It may possibly dissect its way from the mediastinum along the intercostal vessels, and rupture through the parietal pleura at a part that is either weak, or subject to greater stresses than the rest, lifting the parietal pleura along the course of the vessels. A rupture in this site would be extremely difficult to demonstrate at post-mortem examination carried out in the usual manner. I have looked for bubbles of air along the line of the intercostal vessels, and while I have seen them, in Case /
Case 93 for example, I have not been able to satisfy myself that they were not an artefact produced in the course of the autopsy.

I am of the opinion that a "sleeve valve" type of mechanism may be the cause of some tension pneumothoraces and may well be missed at a post-mortem examination if the attention is concentrated solely on the pleura covering the lungs, and the inside of the thorax not carefully examined at the same time.

It is interesting to note in passing that Kjaergaard (1933), in his careful examination of Case 2 in his series reports:

"The place where the lung has ruptured cannot be made out with certainty, in spite of a very careful examination. None of the large valve vesicles are ruptured. There is a little hole on the mediastinal surface of the superior lobe, where it looks as if a pea-sized vesicle may have been torn off after rupturing, but it may be merely a break in the continuity of the pleura as the lung is taken out".

It is possible in this instance and in others that have been reported, that examination of the mediastinal or parietal pleura might have revealed a /
a break in its continuity, allowing for the entry of air from the mediastinum to the pleural sac.

The persistence of the pain in the side of the chest affected, which is often noticeable in some cases of spontaneous pneumothorax, but which is probably less common in cases where a therapeutic artificial pneumothorax has been induced, suggests that there is some persistent irritation of the parietal pleura still present. Since it is the parietal pleura which is sensitive to pain, it cannot be irritated by the layer of visceral pleura overlying the lung when the latter is no longer in contact with it. I have sought for an explanation of this pain in textbooks and in various writings on the subject of spontaneous pneumothorax, but I have found no mention of a possible cause for it. It is my suggestion that the cause might well be air from the mediastinum lying in the extra-pleural space along the line of the intercostal vessels and nerves and irritating the pleura from without. Air is certainly more slowly absorbed from the extra-pleural space, than from the space itself.

If interstitial emphysema of the lungs is accepted as a possible cause of spontaneous pneumothorax, and I believe that I have shown that it is so,
so, the question now arises as to how the interstitial emphysema originates in the first place.

I believe that there are two possible explanations of this occurrence.

In an earlier section of this Thesis, I have noted the state of activity of my subjects, and noted that the pneumothorax incident has occurred while the subject has been at rest or even asleep in bed. From these findings, I have concluded that effort does not immediately precipitate a spontaneous pneumothorax. I have however, noted in one case the presence of a slight pain (Case 91) following the lifting of a heavy weight. This passed off and was succeeded by the onset of a spontaneous pneumothorax at 3 a.m. the following morning, while the subject was asleep in bed. I have attempted to elicit a similar history from other patients, but in most cases there has been no remembrance of any preceding pain in the chest. This in itself is not necessarily an argument against my hypothesis, since the pain may have been trivial or even non-existent, and such a symptom may not have been sought by others in taking a patient's history.

It has been pointed out (Macklin and Macklin, 1944), that it is not only at the periphery of the lung that the interstitial emphysema occurs, but anywhere /
anywhere where marginal alveoli, resting on blood vessels or other structures are found. If, therefore, the site of rupture should be in that part of the lung which lies near to the hilum, the escaped air may be considered to have relatively easy access to the mediastinum. A similar mechanism exists when the trachea is punctured by a needle or small tracheotomy tube, for air will then be driven into the mediastinal tissues with forcible expirations such as coughing, etc. On the other hand, if the site of original alveolar base rupture is peripheral, or rather subpleural, the air may either remain in a localised area, and eventually be absorbed, or it may travel under the pleura to the hilum; or the pleura becomes locally over-distended. Alternatively, it may rupture or ooze directly into the pleural space. In any event, if a pneumothorax is not directly or immediately produced, the necessary predisposing factors are present for its production later on.

In any event, if even a small amount of air is introduced by any of the routes I have described, strain will be put on any adhesions by even a slight collapse of the lung away from the chest wall. Similarly, and I believe this is an important observation, the resulting absence of the restraining and /
FIGURE 21.
EMPHYSEMATOUS BULLAE.
Case 32. Left pneumothorax showing bullae towards apex of lung, and slight mediastinal hernia. There is also a small right apical pneumothorax.

FIGURE 22.
DISAPPEARANCE OF BULLAE AFTER LUNG HAS EXPANDED.
Case 32. The lung has re-expanded, and the bullae are no longer distinguishable. Apart from the emphysema which is present, this might pass for a normal film. The patient suffered no disability at the time this film was taken on follow-up, almost 11 years after Figure 21 above.
and supporting effect of the chest wall on bullae and cysts, will allow these to become over-distended. The normal lung tissue recoils on account of its elasticity, leaving the inelastic bullae distended (Figure 21), and liable to rupture.

Van Allen, Lindskog and Richter (1931), found in their experimental animals that if a small column of Lipiodal was introduced into a small bronchiole, it remained there during respiration, riding to and from permitting no air to pass. Its expulsion was only brought about by a forceful expiratory effort (cough). No more than a droplet of fluid is required to obstruct a capillary bronchiole in this way, and these workers point out that if this occurs from bronchial secretion at night, or during any period when cough is absent for some time, the obstruction must remain and the imprisoned air undergo absorption if free communication does not exist between the alveoli. These workers also refer to further experiments carried out by two of them in which they showed that enough air could be absorbed in thirty minutes to render cough ineffectual in clearing the obstruction. It is thus possible that a small area of collapse in the lung may occur during sleep, particularly if a part of the lung has been strained during previous exertion /
exertion, or if a slight respiratory infection happens to be present at the time. A cough or sneeze will then be sufficient to increase the intra-alveolar pressure, and, in association with the lowered pressure of blood in the pulmonary circulation at rest or during sleep, will allow Macklin's (1937) Factors "A" and "B" both to become operative. It is while the subject is at rest or engaged in moderate activity that I have shown most of the incidents to take place. I have also noted the tendency for the onset to be in the morning (25 per cent of cases), or associated with a slight respiratory infection. Perry (1939), has also noted this tendency for a pneumothorax to occur after a period of quiet breathing.

If this is the explanation in these cases, why is spontaneous pneumothorax not more common? The reason for this is due, I believe, to the relative toughness of the pleura in comparison with the alveolar bases and interstitial tissues of the lungs. I have pointed out that the pleura is capable of withstanding pressures of up to 200 mm. of mercury. Interstitial pulmonary emphysema can be produced by pressures very much lower than this and it is for this reason that it has been customary for the pressure /
pressure in insufflation anaesthesia to be kept as a rule under 25 mm. of mercury.

Interstitial emphysema is probably much more common than is usually believed. It may be the explanation of a variety of pains in the chest for which the physician is unable to find any clinical or radiological cause, and which clear up spontaneously without treatment. I have seen not a few patients during the past four years who have been referred to the Out-Patient Department of the Royal Infirmary with a letter from their doctor stating that following "an attack of 'Flu" they have complained of a pain in the chest. Apart from occasional crepitations, there is usually no clinical abnormality to be made out, and X-Rays of the chest show little, except for perhaps some prominence of the vascular shadows in parts of the lung fields. I have noticed also among in-patients in the wards admitted with respiratory symptoms, in whom the symptoms were much more prominent than the physical signs, small areas of atelectasis both clinically and on radiograms, associated with an acute febrile illness. No specific infecting organism is usually isolated from the sputum of these patients. The conditions are certainly suitable for the production of interstitial emphysema, in the same way as this occurs in children under /
under similar circumstances of slight obstruction or infection and local atelectasis.

Since the above paragraph was written, I have had the opportunity of hearing Dr. J.G. Scadding describe similar areas in the lung fields of his cases with upper respiratory infections, and he has invented the term "aspiration pneumonia" for this condition of "collapse-consolidation".

It is probable that these patients are suffering from pleurisy which may possibly be associated with some pulmonary interstitial emphysema, but I cannot prove this. The association, however, of influenza with interstitial emphysema and pneumomediastinum is emphasised by the Macklins (1944). They refer particularly to the pathological studies carried out following the influenza epidemic of 1918, when air was a frequent finding in the vessel sheaths at autopsy, (Bullowa 1919; Berkley and Coffen, 1919).

In brief, it is probably true that pulmonary interstitial emphysema is more common than is generally believed. Spontaneous pneumothorax as a complication is less common than might be expected, because of the ability of the pleura to withstand a considerable pressure without rupturing.

In textbooks of physiology and in other publications /
publications, it is usually stated that the intra-bronchial pressure may be raised voluntarily as high as 60 cms. of water or from 50 mm. to 8 mm. of mercury, according to different authors, by forceful expiratory efforts (Kountz, Pearson and Koenig, 1932; Kountz and Alexander, 1934; Mautz, 1934; Best and Taylor, 1950). I have shown in an experiment detailed in the next section that males are capable of raising their intra-bronchial pressure to considerably higher figure than females. Although the numbers are small in my experimental series, the difference is striking between the sexes, the average for ten females being 51 mm. of mercury and that for the same number of males 130 mm. It is possible that this higher figure may be related to increased incidence of spontaneous pneumothorax in young healthy men, (and also perhaps of the higher incidence of bullous emphysema, (Ruben, 1948), in males).

I have not encountered any contribution to the literature which attempts to explain the much higher incidence of spontaneous pneumothorax in males, except for such suggestions as Ornstein and Lercher's (1942), that air is forced into the upper lobes more frequently in the muscular robust male, who is more prone to distend his upper lobes by severe exertion, when the expiratory and abdominal muscles are contracted /
contracted and the glottis closed. Draper (1948), however, states that he has found that spontaneous mediastinal emphysema is most common in the same age and sex group as spontaneous pneumothorax, and I have already shown that the latter may be a sequel to the former. Either may be associated with experimental over-inflation of the lungs. I consider that this is supportive evidence for my hypothesis. Some of the observations that have been made following blast injuries to the lung are also in favour of alterations in the relative pressures inside and outside the lungs, as a cause for this type of emphysema.

Neither Kjaergaard (1932), nor Perry (1939), while drawing attention to the disparity in incidence, make any attempt to explain the difference in incidence between the two sexes. I believe that the explanation I have offered fits the facts, and I have not found another alternative explanation which is better able to do this. It follows from the foregoing observations that if the same predisposing causes are present in the lungs of either sex for the production of interstitial emphysema, that this is more likely to occur in the young male because of his ability to produce a higher intra-alveolar pressure. There is therefore a greater risk of a pneumothorax incident /
incident in men than in women.

In attempting to reconcile the fact which I have shown to be true, that these patients are underweight for their height and age, I can only suggest that in such persons the pleura, and particularly the mediastinal pleura lacks the support which is given by fatty areolar tissue, and may therefore be subject to a greater strain than that which is cushioned, supported and protected by the latter. Other explanations might be suggested, but without experimental and other evidence, must necessarily be conjectural, and I will not attempt to offer a further solution to this problem for the present.

Much still remains to be explained about benign spontaneous pneumothorax. I have only in this Thesis touched on some points arising out of the cases which I have studied, yet it has suggested to me several other lines of investigation, which while not strictly relevant to my main theme in this instance, are worthy of further study.

It has been my intention in this Thesis to suggest the possibility of another mechanism to account for the occurrence of a benign spontaneous pneumothorax in healthy persons. This is in addition to, or more often as I believe, as an alternative to the /
the "ruptured emphysematous bulla" which has received
the credit or blame for this occurrence in the
majority of instances, since "The Captain of the Men
of Death" - tuberculosis - was relieved of the
responsibility, almost a quarter of a century ago.

The explanation I have offered, that the cause
of spontaneous pneumothorax in the apparently healthy
person is primarily the result of interstitial
emphysema of the lungs, is one which is in accordance
with the observations I have made on the cases in
this Thesis, for which I have been given clinical
responsibility, those cases I have been allowed to
study and follow up, and others I have referred to in
the literature on the subject of Benign Spontaneous
Pneumothorax.

VIII. EXPERIMENT.

COMPARISON OF INTRABRONCHIAL PRESSURES IN MALES AND
FEMALES BLOWING OUT FORCIBLY AGAINST RESISTANCE.

Mauntz (1943) states:-

"It would be interesting to know what
physiological range of pressure results
from such activities as coughing, blowing,
straining, sneezing etc. By forced blowing,
it is easy to attain pressures of 60 - 70 mm.
Hg. for short periods".

The/
The following experiment was undertaken to ascertain the difference, if any, in the height to which a column of mercury could be raised by males and females of approximately the same age, forcibly blowing out air from their lungs. No comparable figures have been found in any of the common textbooks of Physiology.

Ten male and ten female Medical Student Volunteers were asked to carry out this test. The volunteers were not selected and represented a random sample. Five of the males were ex-servicemen. The mercury column of a sphygmomanometer was connected by a rubber tube to a rubber mouthpiece. With the mouthpiece in position, the subject was asked to breathe out forcibly and gradually raise the column of mercury, attempting to reach the maximum possible height in about five seconds. The time factor was introduced in order to obviate any false readings due to over-swing. Three readings were taken after the subject had got accustomed to the apparatus. Age, height and weight were recorded on the occasion the test was carried out. The average age of the male students was 24.5 years, the youngest being 19 and the oldest 29 years, and of the female students 23.8 years, oldest 30 and youngest 20 years.

The mouthpiece employed was that commonly used on /
TABLE "U".

Mean Finding in Experimental Group of 20 Students blowing out against Resistance.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>24.5</td>
<td>176 lb.</td>
<td>165 lb.</td>
<td>130 mm/Hg.</td>
</tr>
<tr>
<td>Female</td>
<td>23.8</td>
<td>127 &quot;</td>
<td>131 &quot;</td>
<td>51 mm/Hg.</td>
</tr>
</tbody>
</table>
on the recording spirometer, and in that way it was not possible for the subject to form a valvular or narrow opening by the use of the lips and tongue. Each subject was aware of the object in view in carrying out the test, but none was aware till afterwards of the result achieved by his or her colleagues. As a rough check on each subject's effort, the pulse was taken during the time when the test was being carried out, and in each case it was found to diminish in volume and slow down in rate.

It is appreciated that the numbers are small and the test somewhat crude. All were, however, under the same conditions and anxious to co-operate, and the results show that there is a significant difference between the figures recorded by the females and the males.

In all cases a maximum or near maximum inspiration was taken prior to blowing out. It was found that the highest figures were obtained after this procedure, though there was little difference if the forceful expiration followed a moderate inspiration.

The highest individual performance was that of a young Indian student (No. 8 H.S.). He had been a sufferer from asthma in his youth, but had had no attacks for several years. He had carried out breathing exercises as part of the treatment of his asthma.
<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex</th>
<th>Height</th>
<th>Observed Weight</th>
<th>Expected Weight</th>
<th>Max. Manometer Readings mm. Hg.</th>
<th>Average</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 N.M.</td>
<td>21 M</td>
<td>5-11½</td>
<td>188 lbs.</td>
<td>162 lbs.</td>
<td>150 150 150</td>
<td>150</td>
</tr>
<tr>
<td>2 P.M.</td>
<td>21 M</td>
<td>5-10</td>
<td>173 lbs.</td>
<td>153 lbs.</td>
<td>140 120 120</td>
<td>127</td>
</tr>
<tr>
<td>3 J.M.</td>
<td>19 M</td>
<td>5-10</td>
<td>162 lbs.</td>
<td>150 lbs.</td>
<td>168 158 164</td>
<td>163</td>
</tr>
<tr>
<td>4 S.F.</td>
<td>29 M</td>
<td>5-7</td>
<td>161 lbs.</td>
<td>147 lbs.</td>
<td>138 128 130</td>
<td>133</td>
</tr>
<tr>
<td>5 M.W.</td>
<td>28 M</td>
<td>5-6½</td>
<td>159 lbs.</td>
<td>147 lbs.</td>
<td>100  90  96</td>
<td>96</td>
</tr>
<tr>
<td>6 M.L.</td>
<td>29 M</td>
<td>5-10</td>
<td>163 lbs.</td>
<td>100 lbs.</td>
<td>110 120 130</td>
<td>120</td>
</tr>
<tr>
<td>7 G.C.</td>
<td>23 M</td>
<td>6-0</td>
<td>142 lbs.</td>
<td>164 lbs.</td>
<td>70  70  75</td>
<td>73</td>
</tr>
<tr>
<td>8 H.S.</td>
<td>27 M</td>
<td>5-4½</td>
<td>132 lbs.</td>
<td>137 lbs.</td>
<td>190 170 180</td>
<td>180</td>
</tr>
<tr>
<td>9 T.T.</td>
<td>23 M</td>
<td>5-7½</td>
<td>126 lbs.</td>
<td>147 lbs.</td>
<td>110 110 120</td>
<td>113</td>
</tr>
<tr>
<td>10 M.W.</td>
<td>26 M</td>
<td>6-0</td>
<td>196 lbs.</td>
<td>168 lbs.</td>
<td>150 140 130</td>
<td>140</td>
</tr>
<tr>
<td>11 E.R.</td>
<td>26 F</td>
<td>5-7</td>
<td>121 lbs.</td>
<td>139 lbs.</td>
<td>60  50  55</td>
<td>55</td>
</tr>
<tr>
<td>12 H.M.</td>
<td>27 F</td>
<td>5-5½</td>
<td>120 lbs.</td>
<td>137 lbs.</td>
<td>70  60  60</td>
<td>70</td>
</tr>
<tr>
<td>13 A.A.</td>
<td>20 F</td>
<td>5-5½</td>
<td>125 lbs.</td>
<td>132 lbs.</td>
<td>70  75  80</td>
<td>75</td>
</tr>
<tr>
<td>14 A.D.</td>
<td>20 F</td>
<td>5-1½</td>
<td>119 lbs.</td>
<td>118 lbs.</td>
<td>60  50  60</td>
<td>57</td>
</tr>
<tr>
<td>15 J.B.</td>
<td>20 F</td>
<td>5-4</td>
<td>133 lbs.</td>
<td>125 lbs.</td>
<td>40  40  45</td>
<td>41</td>
</tr>
<tr>
<td>16 P.M.</td>
<td>30 F</td>
<td>5-3</td>
<td>136 lbs.</td>
<td>128 lbs.</td>
<td>25  30  20</td>
<td>25</td>
</tr>
<tr>
<td>17 M.A.</td>
<td>24 F</td>
<td>5-3½</td>
<td>110 lbs.</td>
<td>127 lbs.</td>
<td>35  35  35</td>
<td>35</td>
</tr>
<tr>
<td>18 R.K.</td>
<td>27 F</td>
<td>5-10</td>
<td>130 lbs.</td>
<td>153 lbs.</td>
<td>35  40  35</td>
<td>37</td>
</tr>
<tr>
<td>19 E.R.</td>
<td>22 F</td>
<td>5-4</td>
<td>140 lbs.</td>
<td>127 lbs.</td>
<td>44  50  50</td>
<td>48</td>
</tr>
<tr>
<td>20 B.G.</td>
<td>22 F</td>
<td>5-4½</td>
<td>138 lbs.</td>
<td>129 lbs.</td>
<td>65  70  75</td>
<td>70</td>
</tr>
</tbody>
</table>
asthma, and it is probable that these had increased his ability to raise his intrabronchial pressure to this high figure of 180 mm. of mercury.

No. 16 P.M. who gave unusually low readings, had some difficulty with the mouthpiece, owing to a lower dental plate carrying four incisors. Even with this removed, there was no significant difference in the readings however.

The average age, height and weight in both sexes is shown in Table "U", and from these figures it can be seen that males are capable of raising their intrabronchial pressure to a considerably higher figure than females in a similar age group.

IX. AUTOPSY REPORTS.

The records of the post-mortem examinations carried out on the following five cases are taken from the Post-Mortem Records in the Pathology Department of the Royal Infirmary. They are reproduced as they are recorded in the pathologists' report, with only slight modification such as the grouping of all the "Abstract of Record" sections at the beginning, and some minor alterations in grammar and spelling. Parts of some of the reports have been omitted which are not relevant to the subject of this Thesis, such as congestion in various organs, liver, kidney /
kidney etc. As the examinations and reports were in all except two cases carried out by a different pathologist, there is a lack of uniformity of expression, and in some instances there is insufficient detail given to be of much value for comparison. For instance such a statement as "some emphysema was present" does not help in giving an accurate view of the true pathological picture in those cases. In Case 55 (and Case A), the site of the lesion was not visualised but it was noted that there was no broncho-pleural fistula present. This I consider is supportive evidence for my hypothesis that the air in these cases may not come directly from the bronchial airways, but through the interstitium of the lung. In these cases, the patient had a tension pneumothorax, and a mechanism such as I have described would explain this.

In only one case is the Right Ventricle noted as being hypertrophied, though in every case the heart is described as being dilated.

There is a lack of uniformity in the description of the lungs, but it is not stated that the emphysema is generalised throughout the lungs. In most cases congestion is noted at the bases, with marginal and apical emphysematous bullae, largely confined to the upper lobe. In no case is there any evidence of tuberculosis.
tuberculosis.

I have been present at and observed the autopsy on three of the cases described.

ADDENDUM.

While this Thesis was in the final stages of transcription, I was given clinical charge for another patient with a spontaneous pneumothorax who was under the care of Dr. James K. Slater. This patient made a satisfactory recovery from the acute stages, and was discharged to a convalescent hospital. One afternoon, about ten days after his discharge to this hospital, he became suddenly ill again and died before medical assistance could be summoned. Since I have also had the opportunity of witnessing the autopsy on this case, I have included it in the Post-Mortem Reports, but the case is not included in the series as a whole. (Case A).


ABSTRACT OF RECORD.

1. Cardio respiratory failure, following tension pneumothorax due to rupture of an emphysematous bulla.

2. Pulmonary emphysema.

3. Atheroma of cardiac pulmonary arteries.

The /
The body was that of a thin middle-aged man of average build. Some cyanosis of head and neck was present.

**SEROUS SACS.**

On opening the thoracic cavity, it was seen that the right lung was emphysematous and adherent to the chest wall at the base, anteriorly. The heart and mediastinum showed some displacement to the right so that the apex of the heart was in line with the left border of the vertebral column. The left lung was collapsed and there were adhesions present between the pleura of the apex and base of both upper and lower lobes, and the parietal pleura. The adhesions anteriorly had not extended into the lung, but those posteriorly and at the base of the lower lobe were broad and kept the lung in fairly close apposition to the chest wall, and diaphragm. There was no blood or excess of free fluid in the pleural sacs; the diaphragm was considerably depressed to the level of the 7th rib anteriorly. Pericardium and peritoneum were normal. Pharynx, larynx, trachea and large bronchi were normal.

**LEFT LUNG** 640 gms.

Was collapsed. There were numerous fibrous adhesions over both lobes; there were emphysematous bullae.
bullae over the adhesions at the apex of the upper lobe and anteriorly at the base of the upper lobe. The latter situation appeared the most likely site of leakage of air; there was no broncho-pleural fistula.

RIGHT LUNG  880 gms.

Was emphysematous and had a few fibrous adhesions on its surface. There was a large emphysematous bulla at the apex of the right upper lobe. Mucopurulent exudate from the bronchi on pressure. The lower lobe was congested.

HEART  290 gms.

Was rather small. The right side was rather dilated; the tricuspid valve easily admitted four fingers. The musculature of the right ventricle was slightly hypertrophied. The mitral, aortic and pulmonary valves were normal. The left ventricle mostly showed a degree of brown atrophy. The coronary arteries were atheromatous and calcified, but the lumina were patent.

LUNGS Microscopic.

Section from the upper lobe of right lung shows hypertrophic emphysema. The upper lobe of the left lung is collapsed. A portion of it shows acute congestion and in this area there are small areas of broncho-pneumonic consolidation. The bronchi are acutely
acutely inflamed and the lumina are almost obliterated by a mixture of desquamated epithelium and inflammatory cells. It would appear that this might well have caused a valvular obstruction in portions of the lung, which associated with ruptured emphysematous bullae, might well have accounted for the tension pneumothorax.


ABSTRACT OF RECORD.

1. ? Tetany.
2. Generalised cardiac failure.
3. Chronic bronchitis and emphysema.
4. Bilateral pneumothorax.
5. Duodenal ulcer.

The body was that of a well nourished male.

SEROUS SACs.

Pleural, pericardial and peritoneal cavities were healthy.

RESPIRATORY SYSTEM.

Bronchi contained a small quantity of thick mucus and a quantity of slight blood-stained froth. The bronchial mucosa was slightly thickened and very slightly congested.

LUNGS /
LUNGS  Right 500 gms.  Left 640 gms.

Both lungs were almost completely collapsed. There were no pleural adhesions. On the posterior aspect of each apex there was a small rupture of the pleura through which air could be expressed from the lung. Both lungs were moderately emphysematous, particularly in the apical regions. On section both lungs were markedly congested and moderately oedematous. There were no signs of inflammatory consolidation. (No Record of Microscopic Examination).

HEART  260 gms.

It was enlarged, due to gross dilatation of all four chambers. The tricuspid orifice admitted four fingers and the mitral three. The sub-epicardial fat was normal in amount but there was slight infiltration of the right ventricle, particularly at the apex. There were a few scattered petechiae beneath the endocardium on the posterior aspect of the right ventricle. The pulmonary and aortic cusps showed fenestration. There were numerous petechiae beneath the endocardium of the left ventricle.

Heart Blood  A growth of B. Coli was obtained on culture.

STOMACH

Dilated and congested with small ulcer on the posterior /
posterior wall of the first part of duodenum. Surrounding this ulcer, the mucosa was thickened and marked fibrosis had taken place.

CASE NO. 90.  P.S. (45) M.  Joiner.

ABSTRACT OF RECORD.

1. Left sided pneumothorax.
2. Acute cardiac dilatation.

The body was that of a well developed, well nourished middle-aged male.

SEROUS CAVITIES  Pleural Sacs.

There was a left-sided pneumothorax with complete collapse of the lower lobe and partial collapse of the upper lobe. The right pleural sac was normal. Pericardial and peritoneal sacs normal.

LUNGS  Right 570 gms.  Left 350 gms.

There was extensive emphysema of both lungs in the upper lobes. Enlarged bullae were present, one of which ruptured, producing pneumothorax on the left side. The lungs were downy to feel. On section the left lung presented a fleshy appearance of a collapsed lung in the lower lobe. The upper lobe was soft with marrow congestion. The right lung on section /
section gave the typical soft feel of extensive emphysema, and also showed marked congestion. The bronchial tree in both lungs showed suppurative change extending to the medium sized bronchi. The trachea showed acute inflammatory change.

**HEART** 320 gms.

It was generally dilated. Fat and superficial vessels were normal. Dilatation was most marked on the right side. Valvular orifices, tricuspid and mitral valves were incompetent.

**MYOCARDIUM**

The right ventricular muscle was hypertrophied. Left ventricular muscle was of average thickness, pale in colour. Coronary arteries showed no atheromatous change.

**MICROSCOPIC**

Lungs show well marked hypertrophic emphysema.

**CASE NO. 93. J.C. (42) M.**

**PROVISIONAL ABSTRACT**

1. Bilateral bronchopneumonia.
2. Patchy emphysema with bullous forms in the apices and along the anterior free margins.
3. Left pneumothorax.
4. Right sided cardiac dilatation.

The /
The body was that of a middle-aged, markedly emaciated male.

**PLEURAL SACS**

There were a few fibrous adhesions between the visceral and parietal layers situated in either extreme apical region.

**RESPIRATORY SYSTEM** Larynx, trachea and bronchi.

The mucosa of the lower end of the trachea and of the bronchi was congested and there was a definite excess of muco-purulent material in the lumina.

**LUNGS** Right 600 gms. Left 440 gms.

On opening the thorax, the left lung was found to be completely retracted – a total collapse. As pneumothorax was not anticipated, it was extremely difficult to tell whether this had been present or not. However, both lungs showed numerous patchy localised superficial emphysematous bullae situated in the apical regions and along both anterior free margins. No site of rupture of one of these could be ascertained.

**LEFT LUNG**

On section the parenchyma was dark red and solid and liverlike in consistence, with a slight excess of fluid exuding from the cut surface. The bronchi did contain an excess of muco-purulent material but this /
this, in no way, seemed sufficient to produce complete bronchial blockage. The lung also contained numerous small firm nodules about 0.5 cms. in diameter, foci of broncho-pneumonia, but owing to the collapse these were most difficult to distinguish on section. In view of these findings, the diagnosis appears to be that of broncho-pneumonia together with rupture of the superficial emphysematous bulla producing pneumothorax and consequent collapse.

RIGHT LUNG

On opening the thorax, it had retracted to its usual extent. It was dark, mottled, purplish red in colour with diffuse anthracoid pigmentation and containing numerous scattered small firm nodules and on section the parenchyma was dark red and congested with an excess of frothy fluid exuding from the cut surface. One or two of these nodules were seen to be small, pale, firm indefinite areas indicative of broncho-pneumonia.

HEART 400 gms.

Was about one and a half times average size, this increase being mainly right sided. On dissection, the capacity of the right ventricle and right auricle were about one and a half times usual, but the wall was not correspondingly hypertrophied - Pure dilatation.
Left ventricular chamber and left auricle showed no significant abnormality. No abnormality was noted in the patency of the valves, in the cusps, chordae tendineae or papillary muscles. The myocardium was largely flabby in consistence.

CORONARY ARTERIES

Showed a moderate atheromatous change but the lumina were patent.

LUNG FOR CULTURE

The culture yielded growth of B. coli.

(PHOTOGRAPHS of LUNGS and SECTIONS are shown in Figures 16, 17 and 18).

CASE NO. 100. J.S. (48) M. Engineer Fitter.

ABSTRACT OF RECORD

1. Pulmonary emphysema.
2. Gross bullous formation.
3. Congestion and oedema of both lower lobes.
4. Cor pulmonale.
5. Acute right heart failure.
7. Chronic venous congestion spleen.
8. Venous congestion of kidneys.
GENERAL APPEARANCES

The body was that of a spare middle-aged man who looked considerably older than his 48 years. Post-mortem rigidity was almost fully established. Post-mortem lividity was well marked. There was very slight ankle oedema. There was no jaundice. The right pupil was larger than the left. There was an ecchymoses around the right eye.

RESPIRATORY SYSTEM

Larynx - N.A.D.

Trachea and bronchi; contained some viscid mucus. The chest was rather barrel-shaped. Costal cartilages were calcified. There was slight excess of fluid in the right pleural cavity and there were some old fibrous adhesions over the right upper lobe.

RIGHT LUNG

Was voluminous and its upper lobe was grossly emphysematous. There were large marginal emphysematous bullae and most of the remaining lung tissue was disturbed by comparatively gross emphysema. The middle lobe was similarly but less markedly affected. At the margins of the lower lobe there was some emphysema without marked bullous formation. The greater part of the lower lobe was very intensely congested, cyanotic and oedematous. The lung was kept intact for injection.

Left /
Left pleural cavity was almost entirely obliterated by fibrous adhesions of varying density, some of them being of the consistence of leather. The left lung was voluminous and showed changes similar to those in the right lung. Weight 850 gms. The upper lobe showed the same type of emphysema and the lower lobe showed some marginal emphysema and gross congestion and oedema. On sectioning the smaller bronchi stood out rather prominently and around these bronchi there were narrow pale zones which may be due to old fibrosis or perhaps more recent inflammatory consolidation. There was a slight suggestion of dilatation of the bronchi. No pus could be squeezed from the lower lobes. The pulmonary vessels appeared normal.

CARDIOVASCULAR SYSTEM

Pericardial sac contained a slight excess of fluid. It was otherwise normal. Epicardium was smooth and shining.

HEART 300 gms.

Was a little above average size. The subepicardial fat was diminished in amount. Right auricle was distended by post-mortem clot. Tricuspid valve admitted four fingers. Right ventricle was dilated. Its wall was hypertrophied and dark brown in/
in colour. Pulmonary valve was normal. Left auricle was normal. Mitral valve admitted three fingers. Its valve cusps and the cusps of the aortic valve were normal. The left ventricle was firmly contracted, dark brown in colour, of normal size and muscular thickness. Endocardium was normal. Coronary vessels appeared healthy. Aorta and great vessels showed a mild degree of atheroma.

**ALIMENTARY SYSTEM**

Tongue, Pharynx, Oesophagus, Stomach, Duodenum and remainder of the alimentary tract - N.A.D.

**LIVER 1150 gms.**

Was below average size and of roughly normal shape. The reduction in hepatic bulk was uniform both lobes being equally affected. The surface was pale and had an unusual appearance. Large areas of it up to 2 to 3 inches in diameter were slightly raised above the intervening liver substance. These areas were finely granular and yellow in colour. The intervening liver substance was rather darker in colour and of a velvety surface texture. On section the raised areas were found to be hyperplastic liver tissue and in these there appeared to be very slight fine fibrosis. The intervening darker areas were areas of subscapular atrophy, of only 2 to 3 mm. in depth. Their /
Their darker appearance indicated vascularity and that the atrophy was probably of fairly recent onset. The areas of apparent atrophy were entirely confined to the subcapsular region and none were found in the depth of the liver.

**GALL BLADDER & BILE DUCTS**  
N.A.D.

**Spleen** 160 gms.

Was slightly above average size. Its capsule was tense and the splenic substance firm in consistence and dark purple in colour. These appearances indicated some degree of chronic venous congestion.

**Pancreas**  
N.A.D.

**Urogenital System**

**Kidneys**  
R. 180 gms.  L. 210 gms.

Both kidneys were above average size and of normal shape. Their capsules stripped easily leaving a smooth congested surface. On sectioning they were red purple in colour, firm in consistence and showed no abnormalities beyond venous congestion.

**Calyces, Pelves, Ureters, Bladder and Prostate**  
N.A.D.

**Endocrines**

**Thyroid Gland**  
N.A.D.

**Suprarenals**

Both suprarenals were rather above average size. Cortical lipiod was well marked. Medulla appeared rather/
rather congested.

CENTRAL NERVOUS SYSTEM

Cranium and its contents showed no naked eye abnormalities. There was no intra-orbital haemorrhage.

MICROSCOPIC REPORT

LUNG

Sections from the lower lobe show severe intra-alveolar haemorrhage, with small areas of bronchopneumonia. One acinar branch of the pulmonary artery contained ante-mortem thrombus but no other thrombi were discovered. Re-examination of the vessels of the fixed specimen also failed to show pulmonary artery thrombosis or embolism.

HEART

Shows no microscopic abnormalities.

LIVER

There is marked distortion of the normal lobular pattern and the liver is intersected by bands of young fibrous tissue containing proliferating bile ductules and lymphocytes and plasma cells. These fibrous bands are often but not invariably associated with portal tracts. In some of the islands of liver tissue /
tissue there is no resemblance to normal lobular arrangement but in others the lobular pattern is fairly well preserved. There is no diffuse fibrosis and no fibrosis radiating from central veins. Many of the liver cells contain minute unstained — presumably fatty — vacuoles. Beneath the capsules there are areas of young highly vascular fibrous tissue containing proliferating bile ductules and round cells. The presence of several large portal tracts in this repair tissue indicates atrophy or necrosis of subscapular areas of liver of some size. No atrophic liver cells are now seen in such tissue. The microscopic appearances are similar in both lobes of the liver and suggest acute necrosis in small areas — probably anoxic during a period of acute right heart failure. The vascularity of the subcapsular tissue suggests that such an episode occurred not many weeks before death.

PANCREAS and KIDNEY show only venous congestion.

(PHOTOGRAPHS of the RIGHT LUNG and MICRO SECTIONS are shown in Figures 11, 12 and 13).

CASE NO. ADDEIDUM "A". J.R. (50) M.

ABSTRACT /
PNEUMOTHORAX WITH ADHESIONS.

Case "A". Photograph of the X-ray film of this patient showing a pneumothorax with a mid-zone adhesion between the two layers of pleura. Death was due to a superadded right spontaneous pneumothorax.
ABSTRACT OF RECORD

1. Pulmonary emphysema with marginal bullae.
2. Spontaneous pneumothorax - Right and Left.
3. Right ventricular hypertrophy.

The body was that of a well developed man of average build and nutrition. Post-mortem rigidity and lividity were well marked.

RESPIRATORY SYSTEM

LARYNX, TRACHEA and BRONCHI showed no abnormalities. On opening the peritoneal cavity the dome of the diaphragm was depressed so that its convexity pointed towards the pelvis. It was situated at the level of the sixth interspace in the mid-clavicular line on the right side and at the level of the seventh rib on the left side. On opening the pleural cavity on each side, air escaped.

Both lungs were collapsed but were attached in numerous places to the thoracic parieties by thin, string-like and fan-shaped fibrous adhesions. Before separating any of these the lung was inflated via the larynx and trachea. The hiss of escaping air was easily heard and the lungs quickly re-collapsed. After removal from the body, they were re-inflated and held under water. No escape was seen from the left lung but large bubbles of air escaped from a rent in an emphysematous /
EMPHYSEMATOUS BULLAE.

Case "A". Photograph of the lungs of this patient taken at post-mortem examination. A bulla can be seen at the antero inferior angle of the right middle lobe. There are smaller bullae visible along the lower margin of this lobe also.
emphysematous bulla at the right apex. After injection the lungs were large, voluminous and overlapped the heart anteriorly. There was some marginal emphysema and in addition there were emphysematous bullae. That at the right apex was approximately 3 cms. in diameter. Its walls were fibrous and it was attached at one point to the parietal pleura by a fibrous adhesion. It was a tear in the wall of this bulla that was responsible for the right-sided spontaneous pneumothorax. Two rather smaller thin-walled bullae were present, one on the anterior margin of the lower and one on its diaphragmatic aspect.

In the left lung there was an apical thick-walled bulla similar to that on the right lung and there was a series of smaller rather thick-walled bullae on the mediastinal aspect of the right upper lobe. Some of these were attached to pericardium by fibrous adhesions.

On sectioning the injected specimen, the lungs showed numerous small non-palpable aggregations of carbon pigment and some diffuse emphysema.

TRACHEA, BRONCHI and PULMONARY VESSELS - N.A.D.

CARDIOVASCULAR SYSTEM

PERICARDIAL SAC - N.A.D.

The epicardium was smooth and covered a normal amount /
PATCHY EMPHYSEMA.

Case "A". Section of lung from upper part of right lung showing areas of emphysema, and areas where the lung is compressed. The pleura is not thickened. (X 5)
amount of subepicardial fat. Right auricle appeared normal. Tricuspid valve admitted four fingers. The right ventricle showed no abnormality of note. Mitral and aortic valves appeared normal. All branches of the coronary arteries showed well marked atheroma and the anterior descending branch of the left was narrowed almost to the point of occlusion. Despite this, no naked eye damage to the myocardium could be detected.

The aorta and great vessels showed a moderate degree of atheroma.

MICROSCOPIC REPORT

LUNGS

Sections from right and left upper and lower lobes were examined. They are of similar appearance and show emphysematous distension and rupture of alveoli and some distension of bronchioles. The wall of the right apical emphysematous bulla is formed by fibrous tissue pigmented by carbon and infiltrated by chronic inflammatory cells. The appearance of some of the emphysematous bullae including the right apical bulla suggests direct communication with a distended bronchiole. Much of the epithelial lining of the bronchioles has, however, been lost so that identification of the smaller bronchioles is rather difficult.
difficult.

HEART

The myocardium shows no change of note. The coronary artery included in the section is virtually occluded by atheroma. In its wall granuloma has formed around an old area of haemorrhage.

X. SUMMARY.

Historical reference to pneumothorax is not common in the literature. It was probably used as a method of execution by the Greeks in early times, and Laennec's description of the condition is quoted. The earliest use of the name "pneumothorax" to describe the condition is that of Itard (1803). Different types of pneumothorax due to disease or trauma are described, and Benign Spontaneous Pneumothorax is defined as a clinical entity, according to the criteria of Kjaergaard (1932).

The method of collecting the case records of the patients in this series is described. One hundred cases have been collected who have been admitted to the Royal Infirmary as In-patients in the years 1932 to 1950. Seventeen of these were personally observed.

The incidence of Benign Spontaneous Pneumothorax in /
in relation to the total number of patients admitted to the Royal Infirmary is found to be 0.03\%, and comparison is made with figures for other published series.

The ratio of benign to pathological types is discussed, and the immediate mortality of the present series is shown to be 7\%, mostly in the older age groups.

Benign Spontaneous Pneumothorax is shown to be much more common in males than in females. In the present series, it is twenty to one, though the figure commonly recorded is something over six to one. Experimental evidence is produced which shows that men are capable of creating a greater intrapulmonary pressure than women, and it is suggested that this is concerned in the genesis of this type of pneumothorax.

The condition is shown to occur most commonly in the age group around thirty years. The average age of the patients in this series is 32.9 years, and is similar to that recorded in other comparable series.

The side affected is most commonly the left in this series, though in a general survey of recorded cases in the literature either side is probably equally /
equally liable to be affected, with a slight tendency for the right to be the more common.

The method of Follow-up of the patients is described, and the mortality, subsequent development of tuberculosis and recurrences noted. Deducting those patients who had had their pneumothorax less than six months before the Follow-up was carried out, and those who died after admission to the Infirmary, seventy-eight patients have been successfully followed-up out of a possible eighty-seven, for from six months to almost eighteen years. None has developed tuberculosis in that time, and twenty-seven patients have had more than one attack.

A familial incidence is noted in two cases and a possible third.

Three patients in this series had simultaneous bilateral pneumothoraces. One died as a result of this but the other two survived.

The incidence of Benign Spontaneous Pneumothorax is shown to be no more common in those engaged in occupations which demand heavy physical exertion, and no direct relationship of the onset to exertion has been found in most of the cases. Some patients have been wakened from sleep by the onset of the spontaneous pneumothorax, which commonly occurs in the morning.

All except five of the subjects in this series are /
are underweight for their height and age. This is an observation which has not previously been noted in any similar series.

The clinical features of spontaneous pneumothorax are described, and it is noted how the condition can simulate abdominal or heart disease. Mention is made of "Clicking Pneumothorax" in connection with the latter.

The sedimentation rate is shown to be normal in this condition, except where a respiratory infection is present.

The physical signs on clinical examination are described, and the "Anvil" or "Coin" sign discussed.

The different types of Benign Spontaneous Pneumothorax are described; closed, open, tension or valvular, bilateral and chronic, and the treatment of each discussed. The "Water Seal" method of draining a tension pneumothorax is illustrated, and a "sleeve valve" described which is formed between the ruptured alveoli and interstitial tissues of the lung in valvular pneumothoraces.

The two main types of emphysema are described and spontaneous pneumothorax is shown to be a rare complication of alveolar emphysema. It is quite commonly found in association with interstitial emphysema and with mediastinal emphysema.
Different theories that have been advanced as to the possible causes of a spontaneous pneumothorax are considered, the minimal tuberculous lesion, cystic conditions of the lung, alveolar emphysema, congenital weakness and ruptured emphysematous bullae. It is shown that a factor common to all of these conditions is that circumstances are favourable for the development of interstitial emphysema. Air can then travel along the vascular pathways in the interstitium of the lung either to the mediastinum, or to the inner surface of the visceral pleura, forming a bleb. It can then ooze or burst through into the pleural space, to produce a pneumothorax. It is suggested that emphysematous bullae, lacking the firm support of the chest wall when the initial interstitial leak has occurred, may then become overstretched and rupture. This mechanism would explain those cases which at post-mortem examination show emphysematous bullae, none of which have ruptured, and in which no cause is found for the pneumothorax.

An experiment is described in which it is shown that males are capable of raising their intrapulmonary pressure to a greater height than females of a similar age group.

Autopsy Reports are given on five cases in the series, and a sixth not included in the clinical material /
material, for whom the author had clinical responsi-

bility, and who died after transfer to a convalescent 
hospital. Four of the post-mortem examinations were 
personally observed.

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CASE RECORDS.

The following abstracts from the Case histories of the patients in this Thesis includes only the features relevant to the pneumothorax, or to other conditions in the chest which might have some bearing on the incident. If a history of previous disease in the chest is noted, this is mentioned in the abstract of the history given here. Likewise any history of tuberculosis in the patient or in near relatives has been recorded. If there is no such history recorded, and none admitted on Follow up, reference to such negative findings is omitted from the abstract.

The figures following my Case Number, are the number of the Ward to which the patient was admitted, followed by the patient's number in the Admission and Discharge Ledger for that Ward. Since each Physician has charge of one and a half wards, the main ward has been quoted in each instance in order to avoid confusion where patients have been admitted to wards which are under the charge of two different Physicians.

A list of abbreviations used in the Case History Abstracts is given overleaf.
The following abbreviations have been used in the Case Record Summaries:

AB. Apex Beat.

BP. Blood Pressure.

BS. Bell Sound, Coin Sound or Anvil Sound.

BSR. Blood or Erythrocyte Sedimentation Rate.

D.U. Duodenal Ulcer.

F. Female.

F.U. Follow Up.

H.F. Heart Failure.

Ht. Height in Feet and Inches in stocking soles.

L. Left.

M. Male.

N.F.U. Not followed Up. (Usually recent case).

N/R. Not recorded.

P. Pulse Rate per Minute.

PP. Patient under author's clinical charge.

R. Right, or Respiratory rate per minute.

Sp. Sputum.

T. Temperature in Degrees Fahrenheit.

TB. Tuberculosis or Tubercle Bacilli.

T.C. Tuberculosis Officer.

Wt. Weight in Stones and Pounds in bed garments.
Case 1. 23/13629 R.H. (54) M. Burgh Foreman.

Admitted; 22-4-47 Discharged; 12-5-47.

History: Pain in the chest 4 mths. ago.
Dyspnoea on exertion 4 mths.

While walking to his work 4 mths. prior to admission he was suddenly seized with a severe pain in the chest "like a vice". He was very breathless at the time this pain came on, but after a rest he walked on and the pain which had eased recurred. He was sent to bed by his Doctor, but as he continued to be breathless he was sent up to the R.H.E. where he was admitted. He had had a similar incident 15 yrs. before and at that time had a laparotomy for it and was found to have a haemopneumothorax. He was unconscious for 4 days at that time. In 1914 he had multiple G.S.W. which involved his left chest.

Examination: T. 97.0 P. 80 R. N/R BS. -ve
BSR 4 mm. BP 190/110 Ht. N/R Wt. N/R

Left pneumothorax, with no signs on X-ray of TB. Thoracoscopy failed to show any bullae or the site of rupture.

Follow up; Well 2 yrs. later and has had no recurrence.

Case 2. 23/13687 W.S. (42) M. Barman.

Admitted; 21-5-47 Discharged; 14-6-47.

History: Pain in L. chest 6 days.
Dyspnoea 6 days.

Just after washing and shaving he was seized with an acute stabbing pain over the lower part of L. chest. He also had acute breathlessness "as if he had been running a long distance". Symptoms eased after 10 minutes but recurred on walking to work. The following day he had the same experience of the pain easing off at his sedentary work, but recurring on exertion. No previous illness except for 'flu 2 yrs. before.

Examination: T. 98.4 P. 70 R. 20 BS. ve
BSR 2 mm. BP 100/70 Ht. N/R Wt. N/R

Left pneumothorax, with no X-ray or clinical signs of disease in the lungs.

Follow up; Well 2 yrs. later with no recurrence.
Case 3. 28/5821 J.B. (24) M. Fireman.
P.P. Admitted; 19-11-46 Discharged; 3-12-46

History; Pain in L. chest and back 1 hr.
Dyspnoea 1 hr.

An hour before admission he was seized with a severe pain in the L. side of his chest and became very breathless. He had had a cold in the head 14 days before, but no other illnesses except appendicectomy and influenza twice. One sister has asthma.

Examination; T.98.4 P.80 R. 22 BS. N/R
BSR N/R BP 120/80 Ht. 6'0" Wt. 10-9 z

Left pneumothorax. The lung fields show some emphysema on X-ray films.

Follow up; Seen by myself with a recurrence on 15th. Aug.1949, and had an earlier recurrence on 10-1-48. No X-ray evidence of lung disease, and otherwise well.


Admitted; 7-7-40 Discharged; 19-8-40.

History; Pain in R. chest a few hrs.
Slight dyspnoea – a few hrs.

While rising from his seat in the cinema on the night of admission, he was seized with a sudden pain below the R. shoulder blade. When he tried to walk it off it got worse, so he went to his Doctor who sent him into the R.I.E. Had a similar pain in L. side of his chest 1 yr. before, and was investigated for TB with negative results. 1 brother has asthma.

Examination; T.97.4 P. 68 R. 20 BS. N/R
BSR 3 mm. BP 108/72 Ht. 5'10" Wt. 8-11

Right pneumothorax, with only a few signs at the apex. A small amount of fluid seen on X-ray.
Poudrage was carried out on one side (R).

Follow up; Well 9 yrs. later and has served in Paratroops during the War. No recurrence.
Case 5. 32/8266 W.S. (54) M. Engine Driver.

P.P. Admitted; (1) 14/3/44 Discharged; 31/3/44
(2) 16/12/46 " 27/12/46

History: (2) Slight discomfort in R. chest 14 days.
Periodic attacks of breathlessness.

While driving his engine tender first he caught a slight chill. He went to bed feeling breathless but this has now improved. He was in R.I.E. in 1944 (1) with same complaint. (PP. on each occasion.)

Examination; T. 97.4 P. 88 R. 24 BS. -ve.
BSR. 5 mm. BP. 150/85 Ht. N/R Wt. "Thin" sp. TB -ve

Right pneumothorax, with typical physical signs. X-ray shows a bulla at the R. apex. Ba. Meal shows a D.U. ulcer crater.

Follow up: No recurrence after 2nd attack. 2½ yrs. F.U. Still has occasional asthmatic attacks but has not been off work.

Case 6. 25/3592. A.G. (26) M. Labourer in Rubber Wk

Admitted; 29/12/46 Discharged; 14/1/47.

History; Severe pain in R. chest - a few hrs.
Severe pain in epigastrium - a few hrs.
Dyspnoea - a few hrs.

While lying in bed he rolled over and had a sudden severe pain in the R. chest and epigastrium. The pain was also felt in the shoulder. He broke out in a cold sweat and was very breathless. Plays football regularly but has never had a similar pain. Measles and whooping cough in childhood.

Examination; T. 98.4 P. 100 R. 28 BS.+ ve.
BSR 4 mm. BP 126/80 Ht. 5'6½" Wt. 8-8½ Sp.TB -ve

Right pneumothorax with typical signs. No pathological changes in lung fields. He had some guard-in in upper R. abdomen on admission.

Follow up; Well and has had no recurrences in 2½ yrs. F.U.
Case 7. 25/2387 J.G. (52) M. Clerk.

Admitted; 9/11/44 Discharged; 27/11/44.

History: Sudden pain in L. shoulder 14 hrs. Dyspnoea 14 hrs.

He has had a cough with a purulent sputum for about 3 yrs, worse in bad weather. While walking home and smoking "the smoke went the wrong way" and he started to cough and this brought on a sudden pain in the L. shoulder and L. chest. He thought he had lost about a stone in weight in the last 6 mths.

He had no chest troubles until three years prior to admission.

Examination; T. 98. P. 128. R. 36. BS N/R
BSR 50 mm BP 126/84 Ht. N/R Wt. "Well built".
Sputum TB -ve Pneumococci WBC 11,200
Left Pneumothorax. Numerous rhonci and creps. on right side - Bronchitis.
X-ray shows left pneumothorax with chronic bronchitis and emphysema.

Follow up; No further attacks, but has occasional spasms of breathlessness, possibly asthmatic.

P.P. Outpatient MCPD August 1948.

History; Pain in R. chest for some days.
Exertion dyspnoea " " "

While walking in the street a few days ago, he was seized with a severe pain in the right side of his chest. He walked slowly home and was sent by his Doctor to MCPD a few days later. Similar attacks in 1945 and in 1947 when sitting and walking respectively Athletic, swims, boxes, etc. No other relevant previous illnesses.

Examination; BSR 4 mm. Seen after re-expansion. X-ray shows no underlying disease in the lung.

Follow up; Apart from a dull pain in the R. side of his chest for some months after when he exerted himself, has resumed all his former activities. No recurrence 10 mths. later.
Case 9. 23/12018 W.B. (41) M. Plumber.

Admitted 17/4/44 Discharged 20/5/44.

History: Pain in the L. chest 3 days.

3 days before while sitting quietly by the fire at home, he had a twinge of pain over a small area to the left of the sternum. The pain became more severe while he was sitting reading, and he collapsed and vomitted. He had a feeling as if "his chest was caught in a vice", particularly on the L. side but to a less extent on the R. He collapsed on the floor and took about 15 minutes to get up. No previous illnesses.

Examination: T. 98.4 P. 104 R. 30 BS N/R
BSR 14 mm. BP 118/74 Ht. 5'6" Wt. 9-6

Left pneumothorax.

Follow up: Own Dr. says he has had no recurrence, and in course of barium enema for abdominal symptoms recently, only slight pleural thickening was noted in the L. costo-phrenic angle. 5 yrs 2 mths F.U.

Case 10 31/2665 A.B. (34) M. Plumber.

Admitted 2/10/46 Discharged 14/10/46.

History: Pain in L. chest 1 mth.
Dyspnœa 2-3 days at onset.
Intermittent pain in L. chest 15 yrs.

For 15 years at 1 to 3 year intervals he has had pain in the left side of his chest. He has dyspnœa for 2 to 3 days at the time of onset, and the pain clears up in 10 to 14 days. At these times he is conscious of something "rubbing in his chest" and his wife has noticed "a creaking sound" when he bends in certain directions. He has no cough or collapse, palpitation or headaches, and the attacks come on when he is at rest. Tonsillectomy 17 yrs. ago. He is an enthusiast for physical culture.

Examination: T 98.4 P. 80 R. 20
BSR N/R BP 115/65 Ht. 5'6" Wt. 8-9

Left pneumothorax. Deviation of trachea to R. with medium creps. on both sides. X-ray shows some fluid in the L. costo-phrenic angle, and on re-expansion normal lung fields. Ba. meal -ve.

Follow up: Has had no recurrence 24 years later.
Case 11. 28/5492 I. McK. (22) M Occupation N/R
Admitted 9/4/46 Discharged 11/6/46

History: Pain in R. lower chest 3 days. Cough 2 years.

3 days ago he began to have a sharp pain in the lower part of his R. chest. It was not worse on coughing but worse on moving about. He has had a cough for 2 years with a little yellowish sputum. 2 years ago he had a similar attack. He has had no loss of weight. Scarlatina aged 8 yrs. Step-F. died TB?

Examination: "Rapid pulse" BS N/R Sputum TB -ve
10 BSR N/R BP 115/75 Ht. and Wt. N/R

Right hydropneumothorax. Blood stained fluid was aspirated from the chest.

Follow up: Well and has had no recurrence 3 yrs and 2 mths. later.

Case 12. 31/3122 J.T. (31) M. Baker.

Admitted: 8/7/47 Discharged: 1/8/47.

History: Pain in L. chest 2 days. While sitting by the fire, at home he became aware of a pain in the left side of his chest. The pain came on gradually. He had a spontaneous pneumothorax in 1941 and also while in the Forces in Gibraltar and Liverpool in 1944. Has occasional slight cough. One brother has "suspected TB.

Examination: T. 97 P. 100 R. N/R BS N/R
BSR 9 mm BP 140/100 Ht. 5'5½" Wt. 11-8

Left pneumothorax with a few physical signs. X-ray shows no lung pathology and rapid re-expansion.

Follow up: Traced with some difficulty. No recurrences, in 2 years F.U.
Case 13. 31/76 D.D. (40) M. Hotel Porter.

Admitted: 21/11/41 Discharged 12/12/41.

History: Pain in L.chest 3 days.
        Pain in R.side 10 days.
        Epigastric Pain with intermissions 10 yrs.

While on his way to have a Barium Meal at the Royal Infirmary, he was seized with a severe stabbing pain in the L.chest. He was very dyspnoeic, but both these symptoms eased off in about an hour leaving him with a harsh cough and tightness in his side which was worse when he moved about. The pneumothorax was noticed on preliminary screening when he went for his Barium Meal. Ten days before the sudden onset of this pain in his L.chest, he had had a similar pain associated with dyspnoea in his R.chest. This eased with rest in bed. No previous history of cough and is not subject to colds. Previous op. for D.U.

Examination: T. 97 P. 90 R. 20 BS -ve.
        BSR 15 mm. BP 120/80 Ht. 5'5½" Wt 9-3. Sp. TB -ve

Left pneumothorax, with typical signs. Ba.meal shows ulcerdeformity of duodenum but no crater and no stenosis.

Follow up: No recurrence of his pneumothorax in 7 yrs. 7 mths. F.U.


Admitted 6/9/37 Discharged 8/10/37

History: Pain in L.chest 3 days.
        Dyspnoea 3 days.
        Slight cough with frothy sputum 3 days.

While walking across the meadows after his tea, he developed a sharp pain across the front and side of his L.chest. The onset of this pain was associated with breathlessness. He had pneumonia in 1923, side not remembered, and was discharged from the army in 1932 with a spontaneous pneumothorax on the left side. No other illnesses.

Examination: T. 97.4 P. 124 R. 22 BS N/R
        BSR N/R BP 125/84 Ht. 5'9" Wt. 8-11. Sp. TB-ve
        (Sp. contained some RBC).

Left pneumothorax with typical signs. Complete re-expansion in about 2 mths.

Follow up: This patient who was in lodgings at the time of onset of his pneumothorax was not traced on F.U.

Admitted 8/5/44 Discharged 7/6/44.

History: Pain in R. chest 4 days.
Dry cough 4 weeks.

While shovelling at his work, he was seized with a fit of coughing. Then a pain came on suddenly down the R.side of his chest. Nothing eased this pain till he rested on the Sunday 2 days later, when he found that he did not feel the pain if he lay still. Following the episode he had at the time of admission a short cough with no sputum. He vomited the day (Sunday) prior to admission, and had a frontal headache. He says he sweats at night, but has not lost any weight. He has had Typhoid Fever and the usual childhood illnesses.

Examination:  T. 97.0  P. 80  R. 20  BS N/R
BSR 8 mm.  BP N/R  Ht. 5'0"  Wt. 8-12 Sp. TB -ve

Right pneumothorax, with usual clinical findings X-ray shows, (according to Radiologist) chronic bronchitis on L.side. On re-expansion "Slight pneumoconiotic changes" are noted as being present.

Follow up: 5 yrs later has had one recurrence on the same side.

Case 16. 30/1692  W.M. (50) M. Window Cleaner.

Admitted 30/5/39 Discharged 17/6/39

History: Coughing blood 11 days.
"Bronchitis" 6 weeks.

6 weeks ago he had "bronchitis" the chief symptom of which was breathlessness. 11 days prior to admission he started to bring up blood "like liver" in his sputum. No previous respiratory disease, "First illness I ever had".

Examination:  T. 97.0  P. 72  R. 22  BS N/R
BSR N/R  BP 125/93  Ht. 5'3"  Wt. 6-3

Left hydro pneumothorax. Crepitations and rhonchi noted over the other lung. It is noted that his liver is clinically reduced in size. Gross bullous emphysema is noted in the X-ray films with a hydro pneumothorax on the left.

Follow Up. Well and has had no recurrences 10 yrs. and 1 mth later.
Case 17. 28/1384 G.R. (28) M Electrical Engineer.


History: Pain in R chest 3 days. Breathlessness 1 day.

After stooping down to pick up a book he was seized with a sharp pain on straightening up again, in the R. side of his chest. The pain has remained up till the time of admission, and was worse on deep breathing. He was not breathless until the day before admission. He had pneumonia in childhood otherwise he has been healthy.

Examination: T. 97.0 P. 140; R. 20 BS N/R BSR N/R BP 115/65 Ht. 5'10½" Wt. 7-6

Right hydrothorax. 10 mls. of blood were aspirated from the R. pleural sac. Re-expansion and absorption took place, and lung was completely re-expanded in six weeks.

Follow up: Had slight recurrence 4 yrs later in the R.A.F. No further recurrences in F.U. of 10 yrs. 10 mths. Healthy.


Admitted 7/3/47 Discharged 29/3/47

History: Pain in chest 3 days. Dyspnoea 2 hrs. later, afterwards on exertion.

While walking to work 3 days prior to admission, he had a slight twinge of pain in his chest on the left side. This pain recurred at irregular intervals until 2 hrs. later, following a bout of coughing he became dyspnoeic. The pain was now worse on deep breathing and movement, so he remained still and the pain eased and the dyspnoea. In Sept. 1945 and again in May 1946 he had similar attacks which cleared on rest alone. He was discharged from the Marines after 8½ years service for "nervousness". In 1940 he was under observation in New Zealand for TB with negative results. Usual childish ailments, and is subject to winter colds which are associated usually with a cough.
Examination: T. 98.0 P. 80 R. N/R
BSR N/R BP 120/80 Ht. N/R Wt. N/R

Left pneumothorax shown on X-Ray.

Follow up: States that 2 yrs. 3 mths. later had a sudden pain in R. chest which "rendered me nearly incapable of movement and made breathing difficult", which was diagnosed as pleurodynia by his Doctor, though the patient himself at first thought it was "the pneumothorax". No other illness.

Case 19. 23/7977 E.H. (23) M. Occupation N/R


History: Sudden pain in R. chest 1 day.

A pain suddenly appeared in his R. chest without preceding exertion, 1 day before admission. He had no cough but since that time he has developed dyspnoea, a slight cough with no sputum, and he has become more ill. Doubtful history of empyema 11 years before.

Examination: T. 99.2 P. 100 R. 52 BS N/R
BSR N/R BP 140/90 Ht. N/R Wt. 9-12 Sp. TB -ve.

Right pneumothorax; usual findings on clinical examination. Consecutive X-ray films show a small effusion developing at the R. base, and 1 mth. later pneumothorax is confined to the apex, having been complete originally.

Follow up: His family Doctor, having got in touch with the patient's brother, states that the patient was killed on active service in Burma in 1945.


History: Sudden pain in R. chest 10 days. Dyspnoea 10 days.

While preparing to go to work 10 days prior to admission he was seized with a sudden severe pain in
the R. side of his chest. This was accompanied by the onset of dyspnoea, accentuated by slight exertion. He went to bed and the pain eased in 3 days, and he noticed that he was easier lying on the left side. He had had a cough for some time but no night sweats or loss of weight. Influenza and Measles in youth.

Examination  T. 99.0  P. 80  R. 20.  BS N/R.
BSR 2 mm.  BP 126/80  Ht. 5'11"  Wt. 11-2.

Right pneumothorax. Usual signs over R. side of chest. X-ray shows fairly complete collapse of the lung and later films show the lung re-expanding.

Follow up: Not traced.


P.P. (Seen as O.P. 6/1/48)

History: Pain in the L. chest 7 days.
Slight dyspnoea on walking 7 days.

While walking home from a friend's house he was suddenly seized with a sharp pain, worse on deep breathing, about the level of the third left rib just outside the mid-clavicular line. He breathed gently to ease the pain, and was able to walk home slowly. Apart from childish ailments, he had had no previous illnesses, except acute bronchitis 2 yrs. before. He had also had a gland in his back incised as a child, and his father, who died from a coronary thrombosis was found at post mortem examination to have a healed T.B. lesion in his abdomen.

Examination: T. 98.4  P.80  R.18  BS -ve
BSR 5 mm  BP. 120/70  "Tall and thin" No sp.

Left pneumothorax. Few physical signs, except a few crps. at the region of the third rib L. in the ant. axillary line. X-Ray showed a small amount of fluid at the costo-phrenic angle, and calcified glands in the neck. F.U. X-Ray 3 wks. later showed normal lung fields.

Follow up: Well and has had no recurrences 3 yrs. 6 mths. later. Spirometric tracing shown as an example of the normal in Figure " "
Case 22. 32/8731 G.N. (31) M Miner.

P.P. Admitted 25/7/47 Discharged 29/8/47.

History: Sudden pain in R. chest 1 day.
Dyspnoea 1 day.

At 7 p.m. on the day before admission he had a sudden severe pain in the upper part of his R. chest. It was not precipitated by exercise or coughing. Other symptoms were a feeling of faintness and some dyspnoea. He had had an attack of "pleurisy" on the R. side in childhood, and L. "Pleurisy" in 1943. He has had periodic attacks of asthma for some time which respond to ephedrine.

Examination: T. 99.0 P. 88 R. 20 BS +ve
BSR 2 mm. BP 110/90 Ht. 6'1" Wt. 100-10 Sp. TB -ve

Right pneumothorax with typical physical signs.
X-ray of re-expanded lung shows some emphysema only.

Follow up: No recurrences, and has put on almost a stone in weight, at time of re-examination. 2 yrs. F.U.

Case 23. 30/556 W.S. (27) M. Baker.


History: Pain of sudden onset across lower part of front of chest 16 hours.
Slight dyspnoea 16 hrs.

While sitting quietly having his tea, he was seized with a sudden severe pain in the lower part of his chest. The pain, which was associated with slight dyspnoea, eased off in about 8 hrs. He had had no previous attacks and no illnesses, but has a "smoker's cough".

Examination: T. 98.0 P. 110 R. 24 BS +ve.
BSR N/R BP 135/90 Ht. 5'4½" Wt. 83.

Left pneumothorax. Typical physical signs. X-ray shows lung expanding normally, expansion being complete in 6 wks. A trace of fluid in the early stages in L. costo-phrenic angle.

Follow up: In lodgings at onset; not traced.
Admitted 24/7/37 Discharged 3/8/37.

History: Stabbing pain in left chest 3 days. Dyspnoea 3 days.

While bending down he noticed a sudden pain in the left side of his chest in the mid-axillary line. His left arm felt cramped, and his heart beat was embarrassed. He became very dyspnoeic, and noted that the pain was worse on deep breathing and on exertion. He had an attack of the same kind 5 yrs. before, on the same side, and has had a cough for many years. He had pneumonia in childhood.

Examination: T. 97.0 P. 72 R. 26 BS N/R
BSR 5 mm. BP 116/78 Ht. 5'10" Wt. 10-1. Sp.
TB -ve

Left pneumothorax. Few signs clinically in the chest, except a few rhonchi and diminished breath sounds in region of the 8th rib in the mid-axillary line.

Follow up: In East Africa at time of follow up, but own Doctor informs that he examined him 1 yr. before and found him fit, with no apparent recurrences. Over 10 yrs. F.U.

Case 25. 31/1040 J.B. (43) M. Occupation N/R
Admitted: 18/9/43 Discharged: 16/10/43.

History: Sudden pain in upper right chest. Irritating cough, mostly at night, 10 yrs. Dyspnoea at onset of pain.

Suddenly seized with a severe attack of pain in the upper part of the right chest, radiating through to the scapula. He was very dyspnoeic even at rest when the pain came on, and the acute symptoms lasted for 3 days. He had had similar attacks 1 yr. and 10 yrs. before, but not other illnesses.

Examination: T. 97.0 P. 70 R. N/R
BSR N/R BP 160/90 Ht. N/R Wt. N/R
Right pneumothorax. Few physical signs to locate the lesion, and diagnosis was made by X-ray. Gradual re-expansion and no lung disease.

Follow up: His own Doctor states he had a recurrence 6 wks. prior to receiving the F.U. enquiry which lowered with rest in bed but he has been otherwise healthy. 5 yrs. 10 mths. F.U.

Case 26. 22/1571 W.M. (22) M. Warehouseman.


History: Pain in back and L.chest 6 wks.
Dyspnoea 6 wks.

While running for a train, 6 wks. ago he suddenly felt very breathless, so he had to walk. He got relief when he sat down in the compartment. He had no pain until 2 days later when after a heavy day he felt a pain shooting up his back. The breathlessness became more marked, and he was in bed for 10 days with complete relief of his symptoms. He returned to work for 3 wks. but the dyspnoea became worse again, and he developed a pain in the front of the L.chest which was different from the previous one. He remained in bed for a further 4 wks. and the pain and breathlessness passed off. During this time his temperature was normal, and he had no cough or other symptoms. He had a mastoid operation at the age of 3 yrs. and measles, mumps and scarlet fever as a child. One brother had TB.

Examination: T.99.0 P. 108 R.20 BS N/R
BSR 3 mm. BP 120/80 Ht. 6'3" Wt. 11-7

Left pneumothorax, with typical signs.
Relative dullness noted over the R. chest. X-ray shows some chronic inflammatory changes in the affected lung, with some pleural thickening at the costophrenic angle. Some fluid appeared in the pleural sac at a later examination, but this time the lung field is reported as normal.

Follow up: In 10 yrs. 3 mths. F.U. has had no recurrences and has remained well.
Case 27 22/2593 T.C. (32) M. Hosiery Worker

Admitted 11/11/40 Discharged 16/11/40

History: Pain in chest below R. nipple 14 days. Dyspnoea 14 days. Cough with no sputum 6 days.

While walking to work 14 days ago, he developed a cramping pain below the R. nipple. This pain became gradually more severe, and was maximal in 1½ hrs. Dyspnoea was associated with the onset of this pain, and became gradually more severe. He has had no night sweats nor loss of wt. In 1929 he was in hospital for six wks. with a "chilled stomach", and in 1934 was struck below the right nipple by the steering wheel of a motor car.

Examination: T.97.4 P. 74 R. 20 BS N/R BSR 2 mm. BP 130/80 Ht. 5'5½" Wt. 8-9

Right pneumothorax, with the typical signs. X-ray shows no evidence of lung disease, though on the F.U. films there may be some emphysema.

Follow up: 8 yrs and 7 mths later has had no recurrences, and has been in the Forces and discharged Grade 1.

Case 28 30/1496 A.Mc. (26) M. Motor Driver.


History: Pain in R. chest a few hrs. Dyspnoea - a few hrs.

While dressing in the morning he was suddenly seized with a severe pain in the R. side of his chest. No previous similar attacks. "Smoker's cough".

Examination: T.97.4 P. 90 R. 28 BS N/R BSR N/R BP 110/60 Ht. 5'7" Wt. 10-1

Right pneumothorax with typical signs. X-ray shows complete collapse with no fluid.

Follow up: Well and has had no recurrences 10 yrs. 4 mths. later.
Case 29. 32/8112 R.S. (44) M. Bus Driver.


History: Pain in L. Chest 1 mth. prior to admission. Dyspnoea 1 mth. Cough - several years.

While watching a football match he suddenly developed a pain in the left side of his chest. It eased somewhat but he was still dyspnoeic, and as it persisted he was sent in to the Infirmary for examination. No previous chest troubles.

Examination: T. 97.0 P. 72 R. 20 BS ve
BSR 10 mm. BP 160/95 Ht. 5'6" Wt. 7'-13 Sp. TB -ve

Left pneumothorax with typical findings on clinical examination. X-rays show the lung slow to expand and the development of a little fluid up to the level of the 10th rib posteriorly. Complete expansion except for a slight apical pneumothorax in 7 mths.

Follow up: No recurrence 2 yrs. 8 mths later.


Admitted: 13/5/42 Discharged: 13/6/42.

History: Dyspnoea on exertion 3 days.

5 days before admission he felt "chilly" after watching a football match. The next morning he felt a stiffness across the lower back, and he again felt cold and shivery at night while standing, and was slightly breathless on walking home. On the Monday going to his work he felt very breathless, and on climbing 3 flights of stairs, he found he was "gasping for breath" He had no cough or other symptoms. He had a perforated D.U. 10 yrs. before and in 1918 was struck by shrapnel in the chest and after this was removed from his R. chest, he was breathless for some time.

Examination: T. 97.0 P. 84 R. 20 BS -ve.
BSR N/R BP 120/70 Ht. 5'8½" Wt. 8-12

Right pneumothorax, with typical clinical signs.

Follow up: 7 yrs. 1 mth. later has had no recurrences.
Case 31. 32/8677 J.E. (53) M. Dispatch Clerk.
P.P. Admitted: 4/7/47 Discharged: 18/7/47.

History: Pain in L. Chest 6 wks.
        Dyspnoea on exertion 6 wks.
        Swelling of ankles 7 days.

While at work he suddenly had a severe pain in the L. chest. He was dyspnoeic for a short time at the onset of this pain, but the acute symptoms gradually eased off, and he was left with a dull ache in the chest which persisted until the time of admission. For 1 wk. prior to admission he has noticed that he has been increasingly breathless on exertion, and his ankles have begun to swell at night. He had "dropsy" at the age of 10 yrs. and has had a D.U. which perforated and for which he had a gastro-enterostomy in 1919.

Examination: T. 97.0  P. 80  R. 20  BS N/R
            BSR 40mm.  HP 120/85  Ht. N/R  Wt. 9-4 "Thin"
            Sp  TB -ve

Left pneumothorax, with congestive H.F. Urine Alb. Not any gross pathological changes in the lung fields, and small amount of fluid in L. costophrenic angle.

Follow up: No recurrence in 2 yrs. F.U.

Case 32. 27/199 A.G. (26) M Linoleum Worker.

Admitted 3/1/39 Discharged 16/1/39.

History: Gripping sensation in chest 1 day.
        Dyspnoea 1 day.

While dressing on the morning of admission, he experienced a sudden tightness in his chest associated with dyspnoea. He had "difficulty in getting air into his lungs". He had had 4 previous similar attacks in the past 4 yrs. all coming on at rest or at moderate activity, one having come on while he was asleep. He had noticed that the acute phase lasts about 2-3 days and gradually wears off in about 10 days. He has an occasional dry cough in the winter. The onset of the present attack was associated with a fit of coughing.
Examination: T.97.8 P.88 R.22 BS ve. BSR 5 mm. BP 144/96 Ht. 5'7" Wt. 8-1½" Sp.TB -ve

Bilateral pneumothorax, clinical signs being noted on the L.side, and the smaller pneumothorax on the R.side in the apical region being only seen on X-ray.

Follow up: 11 yrs 2 mths. Has had no recurrences, and is "only afraid of getting one when he has a cold in the winter time".

Case 33. 31/4359 A.R. (26) M. Baker.

Admitted: 9/6/49 Discharged: 21/6/49.

History: Sudden pain in R.chest 24 hrs. Dyspnoea 24 hrs.

While at work he suddenly developed a severe pain in the R.side of his chest. Similar attack 2 yrs ago.

Examination: T. 98.4 P.94 R. 26 BS N/R. BSR N/R BP 150/90 Ht. 5'11" Wt. 10-5

Right pneumothorax with typical clinical findings.

Follow up: Well 6 mths later and has had no recurrence.

Case 34. 22/1003 E.C. (26) M. Salesman.

Admitted 30/4/38 Discharged: 13/5/38.

History: Pain in L.chest 3 days. Slight dyspnoea 3 days. Cough with little sputum 2 days.

7 days before admission he had a slight cold in the head. This improved, but 4 days later while at work he was suddenly seized with a severe pain in the left side and back. He carried on with his work in spite of being slightly breathless, and went to see his Doctor. Next day he developed a cough with a little sputum, and on one occasion a small amount of blood. He states he sweats freely, but not noticeably at night, and has had a slight loss of weight over the past 2 yrs.
Examination: T.98.0 P. 90 R.24 BS +ve.
BSR 7 mm. BP 130/83 Ht. 5'10" Wt. 9-1 Sp.TB -ve.

Left pneumothorax, with classical signs, and no evidence of fluid in pleural sac.

Follow up: Not traced.

Case 35. 23/9429 A.R. (35) M. Bill-poster.
Admitted: 5/6/39 Discharged:

History: Pain in L.chest a few hrs.

While on his way to work in the morning, he was seized with a sudden severe pain in the left side of his chest, shooting through to the back. The pain was of a gripping nature and was worse on coughing and deep breathing. He collapsed at his work and was brought to the R.I.E. He had a similar but less severe attack of pain the day prior to admission, which only lasted 2½ hrs. 2 years, before this episode he had had a similar pain which was diagnosed at that time as pleurisy.

Examination: T.98.2 P. 88 R. 20 BS. +ve.
BSR 5 mm. BP 110/65 Ht. 5'2" Wt. 6-7

Left pneumothorax with typical signs. Later X-rays show re-expansion taking place.

Follow up: Not traced.

Case 36. 30/2910 W.W. (49) M. Checker.

History: Pain in L.chest 5 days.
Dyspnoea 5 days.
Cough 7 days.

While on his way to work he was suddenly seized with a severe stabbing pain in his L. side. He had had a cold in the head with some cough for 7-10 days prior to the incident, but had no previous history of chest disease.
Examination: T. 97.0 P. 70 R. 20. BS N/R
ESR 4 mm. BP 125/85 Ht. 6'1" Wt. 9-4

Left pneumothorax with few physical signs except some tympany in the L. axilla. X-ray shows loculated air in the L. pleural sac with adhesions between the two layers of pleura, and signs of chronic bronchitis and emphysema in the lung fields.

Follow up: Not traced.

Case 37. 25/1901 A.B. (42) M. Railway Fireman.

Admitted 30/10/43 Discharged 26/11/43.

History: Pain in L. chest 3 days.
Shivering attack 3 days ago.
Cough with "rusty" sputum 3 days.

14 days before admission he had a cold in the head, and 3 days before admission he had rigors, pain in the chest and a "rusty" sputum, and felt weak and tired. He was lying in bed at the onset of the pain. He had been treated by his Doctor with "M & B" prior to admission. No previous chest troubles.

Examination: T. 103.4 P. 100 R. 40 BS -ve
ESR N/R BP 126/84 Ht. 5'4" Wt. 7-0 Sp. Pneumococci.

Left pneumothorax and pneumonia. The X-ray shows partial collapse of the L. lung, but no fluid in the pleural sac. Clinical signs of consolidation were made out at the base of the L. lung.

Follow up: Well and has had no recurrence 5 yrs. 9 mths. later, and X-ray of chest is negative.

Case 38. 22/794 W.B. (16) M. Hairdresser.

Admitted 30/12/37 Discharged 26/1/38.

History: Pain in L. chest 2 days.
Cough and head cold 3 wks.

Until 3 wks. prior to admission he felt fit, but developed a cold with a cough and a little yellowish or white sputum. 2 days prior to admission he had a sudden severe pain in the L. side of his chest while
cutting hair. He became very breathless and went off work. He had no night sweats. He had what was probably acute rheumatism at the age of 6 or 8 yrs. when both legs were swollen, pneumonia and pleurisy aged 10, and is subject to bronchial and nasal catarrh. His tonsils and adenoids were removed in childhood.

BSR 18 mm. BP 120/78 Ht. N/R Wt. N/R Sp. TB -ve

Left pneumothorax, with typical signs on clinical examination. Fluid was shown to be present at the base, and pure blood was aspirated on 2 occasions, 15 fl. ozs. and 17 fl. ozs. Notified to T.O. but no TB ever found in sputum or in inoculated guinea pig.

Follow up: Traced eventually through the help of the T.O. No TB ever found, and is well and has had no recurrence in 11 yrs. 8 mths.


History: Sudden pain behind sternum 6 days.
Dyspnoea 6 days.
Spasms of dyspnoea 18 mths,
Cough 18 mths.

18 months prior to admission he began to suffer from dyspnoea of sudden onset and cessation. He would waken through the night gasping for breath, and this spasm would last for about 15 mins. Recently these attacks have become more frequent. 6 days before admission while walking along the street he was seized with sudden dyspnoea and pain over the lower part of the sternum. He went to bed and the pain eased and the dyspnoea, but returned in a few days. He has a short cough with some muco-purulent sputum, but there has never been any blood in the spit. 2 yrs. ago he had had blood poisoning, and he had malaria, in the 1914-1918 war in Egypt.

Examination: T. 98.4 P. 60 R. 22 BS. N/R
BSR 21 mm. BP 130/90 Ht. 6'0" Wt. 7-12 Sp. TB -ve 3.

Right pneumothorax with a few harsh rhonchi over both sides of the chest. X-ray shows collapse of the upper part of the R. lung, and some generalised emphysema.
Follow up: This patient was found to have died in the follow up period about 3 yrs. after the incident for which he was admitted. According to his Doctor's description it would appear that he died of acute R. heart failure.

Case 40. 22/7805 R.M. (27) M. Occupation N/R.  
Admitted: 6/1/49 Discharged: 13/1/49.  
History: Pain behind sternum 3 days.  
Exertion dyspnoea 3 days.  
He woke up one morning with a pain behind his sternum. He walked to work, and the pain became more severe and travelled through to the back. It was constantly present and unrelated to exertion. He had had a similar episode in 1948 for which he was also admitted to hospital, but has had no other illness except for an occasional sore throat.  
Examination: T. 98.8 P. 70 R. 18  
BSR N/R BP 140/92 Ht. 5'6½" Wt. 10-0  
Left pneumothorax, with no physical signs except what is described as friction at the cardiac apex, and a click in the diastolic phase of the cardiac cycle. A small apical pneumothorax was seen on the X-ray films of the chest, on the left side.  
Follow up: This patient has had one recurrence of his symptoms in the 1 yrs. follow up.

Case 41. 30/8122 W.L. (39) M. Joiner.  
History: Pain over front of chest - a few hrs. Dyspnoea a few hrs.  
While sitting at breakfast he was seized with a pain which went right across the front of his chest. This was associated with breathlessness and a slight cough but no sputum. He had a similar but less severe attack 1 mth. before. 3 yrs before he had had a tonsillectomy.  
Examination: T. 97 P. 80 R. 20 B.S. -ve.  
BSR 2 mm. BP 130/80 Ht. N/R Wt. 9-3.
Left pneumothorax with typical physical findings. X-ray shows a small effusion.

Follow up: Well and no recurrence 1 yr. later.

Case 42. 22(ML)5764. G.K. (25) M. Miner.
Admitted: 30/4/32 Discharged: 10/6/32.

History: Pain in L. chest 8 days. Dyspnoea 5 days.

8 days prior to admission he woke up with a pain in his L. chest. He stayed at home from work but 2 days later he went for a walk and the pain became more severe and was in the region of the L.axilla. At that time he first noticed the dyspnoea. He had had occasional coughs previously but had none at the time of the incident.

Examination: T. 98.0 P. 90 R. 22 BS. +ve.

Left pneumothorax with typical clinical signs. X-ray shows a small amount of fluid in the L. costophrenic angle.

Follow up: 17 yrs. 2 mths later he has had no recurrences, though he states it was 2 yrs. before he was able to work after the incident. Sister and a 2nd cousin have also had spont. pneumothoraces.

Case 43. 22/6291 W.F. (29) M. Painter.
Admitted: 4/10/46 Discharged: 30/10/46.

History: "Strained" himself 16 days before. (Transferred from Surg. Wd. after gastrectomy)

For 11 years has suffered from D.U. On admission to the surgical ward for a partial gastrectomy, he was noticed on screening prior to operation to have a partial R. pneumothorax. Thought that he had strained himself 2 days before admission to the surgical ward. After operation his pneumothorax was increased in size and he was much more dyspnoeic, cyanosed and had pain across the front of his chest. He had had pneumonia on the L. side in 1937.

Examination: T.98.4 P. 82 R. 24 BS N/R. BSR 10 mm. BP 130/75 Ht. 5'9" Wt. 6-13 Sp. TB -ve.
Right pneumothorax, with typical signs. X-ray showed a little fluid in the cost-phrenic angle.

Follow up: No recurrences in 2 yrs. 8 mths. F.U.

Case 44. 31/221. J. Mc. (13) M. Rly. Fireman.

Admitted: 21/3/42 Discharged 11.4.42.

History: Pain in back and front L. chest 1 day.
Dyspnoea 1 day.

After pushing a railway engine round on the turntable he walked about 50 yds. and felt a sudden pain in his left chest. This was associated with dyspnoea but he had no cough or spit. He had a winter cough.

Examination: T. 98.4 P. 70 R. 22 BS. N/R.
BSR N/R BP 118/74 Ht. 6'1" Wt. 10-2.

Left pneumothorax, with the usual clinical signs. X-ray shows a partial pneumothorax.

Follow Up: Is now a regular soldier in the Scots Guards, and had one slight recurrence which he thinks was on the R. side one year after. 7 yrs. 3 mths. F.U.

Case 45. 22/7143 T. Mc. (34) M. Pit Surface Worker.

Admitted: 5/2/48 Discharged: 18/2/48

History: Pain in R. chest 10 days.
Slight Dyspnoea on exertion 2-3 mths.

While asleep in bed 10 days prior to admission, he was awakened by a sharp stabbing pain which seemed to stab through from the R. nipple to the back. He had no cough and was not markedly breathless. When he attempted to rise and go to his work, the pain became severe so that he had to remain in bed. At the time of admission he had slight breathlessness on turning and moving in bed. 1 yr. before he had had a sharp pain in the R. chest which seemed to move up and down as he walked, and was transferred at that time to surface work because of headaches.

Examination: T. 98.4 P. 70 R. 22 BS. N/R
BSR 7 mm. BP 125/90 Ht. N/R Wt. N/R.

Right pneumothorax, with usual signs. X-ray
shows collapse to be mostly basal, and no effusion.

Follow up: No recurrences 1 yr. 4 mths. later.

Case 46. 22/7008 R.T. (60) M. Electrician.

Admitted: 30/10/47 Discharged: 24/12/47.

History: Pain in L chest 3 days.
Acute dyspnoea 3 days.
Cough and dyspnoea several yrs.

He has been breathless on exertion for many yrs. but this has been worse for the past 18 mths. 3 days before admission he suddenly became much more breathless, and had a sharp pain in the region of the L. shoulder and L. infra-mammary region. He had a pneumonia 20 yrs before, when he was also diagnosed as having an angina of effort and L. heart failure. At that time also he was having dizzy attacks, frontal headaches and dyspnoea.

Examination: T.97.4 P.82 R.22 BS. N/R.
BSR N/R BP 120/70 Ht. 5'6½" Wt. 7-1 Sp. TB -ve.

Left pneumothorax, with clinical signs of emphysema also noted on X-rays. A possible bulla at the base. Some fluid or pleural thickening at the costo-phrenic angle noted as the lung re-expands.

Follow up: No recurrences in 1 yr. 8 mths. F.U.

Case 47. 30/5160 D.S. (54) M. Butcher.

Admitted: 8/1/44 Discharged 12/1/44.

History: Pain in back 5 wks.
Orthopnoea 5 wks.

5 wks prior to admission he developed a severe "doubling up" pain in the back. He had no temperature at that time. He only has occasional winter colds.

Examination: T. 98.0 P.120 R.28 BS. N/R
BSR N/R BP 160/110 Ht. 5'8" Wt. 8-8
Left pneumothorax with displacement of the tracheas on X-ray.

Follow up: No recurrence in 5 yrs. 5 mths. F.U.

Case 43. 23/9562 P. Mc. (26) M. Joiner.


History: Heavy feeling in R. chest 2 days.

While walking the day prior to admission, he became conscious of a heaviness and a dull feeling in his R. chest, which became gradually worse. He could not sleep well that night because of this feeling, which was worse when he lay on his back. He had previously been well apart from whooping cough and measles in childhood.

Examination: T. 99.2 P. 88 R. 24 BS. ve. ESR 2 mm. BP 148/95 Ht. 5'4" Wt. 7-11.

Right pneumothorax with typical physical findings, complete collapse shown on X-ray.

Follow up: 9 yrs. 10 mths. later has had no recurrence.

Case 49. 22(ML)7907 D.P. (40) M. Paper Mill Wkr.

Admitted: 18/2/36 Discharged: 24/3/36.

History: Pain in R. hypochondrium 5 days. Cough 5 days. Dyspnæa 5 days.

While sitting by the fire he was seized with a severe pain below the R. costal margin, associated with dyspnæa. The acute symptoms passed off in about 20 mins. and only a dull ache remained thereafter. He was sent up to a surgical ward first, but was seen and transferred to the medical side. He has an occasional dry cough and had measles in childhood.

Examination: T. 97.0 P. 80 R.20 BS N/R. ESR N/R BP 125/85 Ht. N/R Wt. N/R.

Right pneumothorax with typical clinical signs. No X-ray evidence of disease.

Follow up: 18 yrs.4 mths. has had no recurrence.
Case 50.  22/7180  J.L. (21) M. Railway Fireman.


History:  Pain in L. Shoulder 1 day.
          Head cold 10 days.

10 days prior to admission he developed a cold in the head after a soaking in the rain. He had a cough with a greenish spit. On rising after lighting the fire in the morning of admission, he felt a sudden pain in the region of his L. nipple. This pain passed to his L. shoulder and down the back of his arm to the elbow. He was very breathless and had to sit down. The dyspnoea passed off, but the pain remained. He managed however to walk to his work and was sent to hospital in an ambulance. He also noted the presence of a "clapping sound" which was synchronous with his heart beat. No previous chest disease.

Examination:  T. N/R  P. N/R  R. N/R  BS. N/R.
            BSR 2 mm.  BP 150/90  Ht. 5'1½"  Wt. 110.  Sp. TB -ve.

Left pneumothorax, with practically no clinical signs at all. L. lung noted to be in about 1-2 cms from the chest wall on X-ray.

Follow up:  Now in Rhodesia. After he returned to work for two or 3 days after his convalescence was completed, his R. lung collapsed, but he did not stay off work long, and was thereafter on a light job for 3 mths. He was recently X-rayed in Bulawayo and the film was normal, and he has not had any further incidents in 1 yr. 5 mths F.U.

Case 51.  27/6241  Mrs. M.D. (28) F. H/Wife.

Admitted:  1/2/49  Discharged 22/2/49.

History:  Pain in R. chest - a few hrs.
          Dyspnoea - a few hrs.

While still in bed at 7 a.m. she felt an acute pain in the R. chest and R. shoulder. She had no cough or sputum. 2 wks. before she had a ligation of veins as an O.P. but was ambulant. No previous history of chest disease, but had "threatened mastoid" aged 10 yrs. and appendicectomy aged 17 yrs.

Examination:  T. 97.0  P. 84  R. 22  BS. N/R
            BSR 5 mm.  BP. 108/64  Ht. 5'2½"  Wt. 82.
Right pneumothorax, with typical signs. Pallor but no cyanosis noted on admission. X-ray shows no disease in the lungs.

Follow up: 1 recurrence on the same side in 6 mths.

Case 52. 15/16 Miss M.S. (68) F. Receptionist.

History: Sudden pain over back and R. side 3 hrs.

While playing dominoes in the evening she was seized with a sudden severe pain over the region of mid-dorsal spine. The pain remained in this site for about 1 1/2 hrs. and then moved round into the right lower chest. She vomited and was short of breath within 5 mins. of the onset of the pain. She had had attacks of "indigestion" for some time, with an unproven history of peptic ulcer. Apparently had had a "heart attack" about 5 yrs. ago.


Rather overweight woman who looks younger than her years. Restless, moaning with pain, cold clammy sweat, with cold clammy cyanosed extremities.

Abdomen: Guarded and almost board-like, with no movement on respiration. No apparent loss of increase of liver dullness in the abdomen.

Chest: Movements poor but apparently equal, with some diminution of air entry at the R. base. A few coarse creps. or friction at the R. base.

Heart: Pulse regular rather fast - c 120 - but regular and of good volume. BP 150/80. Apex beat in the 6th space at anterior axillary line, with systolic thrill and rough systolic murmur at the apex.

Progress: Treated conservatively with heroin and "Aminophylline".
9-9-49: Much more cyanosed at 8.30 a.m. and Oxygen given by B.L.B. mask. Signs of R. pneumothorax now apparent. A needle was introduced into the right pleural sac in the 6th interspace and air came out under pressure. Drainage with a tube under water was instituted.
Later in the day the patient's pulse became gradually more weak in spite of these measures, and she died at 6.30 p.m. In the later stages a blood stained fluid was being expelled through the tube, as well as air. Approximately 5 cc. of air was expelled with each respiration, but this increased to about 15 - 20 cc. on coughing.

Permission was not granted for an autopsy.

Case 53. 25/4927 C.H. (54) M. House Painter.


History: Pain across chest 6 days before admission.
Increasing dyspnoea 6 days.

While at his work as a painter 6 days prior to admission he was suddenly seized by a severe pain across the front of his chest. The pain eased off after a few minutes but he has noticed that he has become increasingly breathless since that time. He has had a cough with no sputum for many years (he smokes 20 cigarettes per day) and has had a pain in his chest before associated with an increase in his cough. Otherwise he has never been ill before.

Examination: T. 93.0 P. 90 R. 26 BS N/R.
B.S.R. 5 mm. BP 120/78 Mt. 5'10" Wt. 8-6' Sp. TB -ve 9

Left pneumothorax, with typical signs. X-ray shows no fluid but some fibrosis at the R. apex suggestive of a TB lesion. Later films show some fibrosis of the re-expanding lung, with some emphysema and emphysematous bullae. The A. apex remains as before. A Mantoux test was -ve to 1/10,000 dilution.

Follow up: This patient was seen again 8 mths. later and was well and had no recurrences.

Case 54. 28/6520 M.C. (15) Schoolboy.


History: Pain in R. chest 2 days.
Dyspnoea 2 days.

2 days before admission this boy had pain in the front of his chest going through to the back and worse on deep breathing. Only other recorded illness was
tonsillitis.

Examination: T.98.4 P. 110 R. 24 BS. N/R. BSR 2 mm. "Thin".

Right pneumothorax, with few physical signs except deviation of the apex beat and trachea. X-ray shows a partial pneumothorax and on re-expansion no pathological changes in the lungs.

Follow up: No recurrence in 16 mths. F.U.

Case 55. 25/4450 J.S. (54) M. Brickwork Labourer.

History: Pain in L. lower chest 3 wks.
Dyspnoea on slight exertion 3 wks.
Cough, worse in winter, 10 yrs.

3 wks prior to admission he developed a severe pain in the lower part of the L. chest. He has been increasingly more breathless on exertion for the past 5 yrs. No other history of chest disease, apart from a slight winter cough for the past 10 yrs.

Examination: T.97.2 P.104 R.38. BS. N/R. BSR 5 mm. BP 138/92 Ht. N/R. Wt. 6-7 Sp. TB -ve.

Left pneumothorax with typical signs. X-ray shows apical adhesion preventing complete collapse. Slight cardiac displacement. Aspiration of air from the L. pleural sac showed this to be a tension pneumothorax, and in spite of treatment the patient died.

A post mortem examination was carried out, and the report is given in detail in the Section of this Thesis where these are described.

Case 56. 32/0P/7371 Miss M.D. (33) F. Farm Worker
P.P. Seen first 20/4/49.

History: Stabbing pain in L. chest 8 wks. ago.
Dyspnoea at onset, now easier.

While working at the potato pits 8 wks. before she was seized with a sudden pain in the L. side below the L. breast in the ant.axillary line. The pain was worse on deep breathing, and though she was
dyspnoeic at the onset of the pain the breathlessness has now passed off. She has no cough. She had bronchitis in 1945 but has otherwise been fit. No pain at time of examination.

Examination: T. R. and R. normal. BS -ve. BSR 11 mm. BP 130/80 Ht. 5'5" Wt. 10-7½

Left pneumothorax, with few signs except increased resonance and diminished breath sounds and a few crepitations. X-ray shows no evidence of lung disease but possibly very slight amount of fluid. Re-expansion shows normal lung fields.

Follow up: Well and has no recurrence 8 mths. later. X-ray clear.

Case 57. MOPD/41 G.R. (21) M. Medical Student.

Outpatient MOPD Oct. 1941

History: Pain in L. chest 3 days.
"Clicking Sound" in chest 1 day.
Slight breathlessness 1 day.

While asleep in bed he suddenly got a severe pain in the L. side of his chest which woke him up. This pain passed off and he went to sleep again. 2 days later however he developed a "clicking sound" in the chest synchronous with his heart beat, which alarmed him. The sound was clearly audible across the room in which he slept, and appeared in cardiac systole, and the intensity of it could be altered by alterations in his position, such as leaning forward or bending to the left.

Examination: T. P. R. etc. not recorded.
BP 125/80 Ht. 6'0" Wt. 11-0.

Left pneumothorax with no physical signs except this clicking sound. Diagnosis made on X-ray which showed the pneumothorax to be a small one.

Follow up: Well and has been active in the R.A.F. in sport and other enterprises. 8 yrs. F.U.

Case 58. 30/7256 Mrs.M.G. (42) F. H/Wife.

History: Pain in epigastrium 14 days.
Pain in L. hypochondrium 12 days.
Acute breathlessness 12 days.

This patient was admitted to a surgical ward 14 days before with a history suggestive of a perforated D.U. She had a 20 yrs history of D.U. but at operation she showed only scarring of the duodenum and nil else. Apparently during the night she had a severe pain also in the L. lower chest, and had had a slight head cold preceding this. She had had asthma as a child, and a minor gynaecological operation. Seen by a physician and transferred to medical ward.

Examination: (on admission to medical ward)
T. 98.0 P. 110. R. 28 BS N/R

Left pneumothorax, on clinical examination.
Died day following admission from R. heart failure.

No post mortem examination carried out.

Case 59. 31/H) 7329 A.C. (28) M. Clerk.
Admitted: 28/9/39 Discharged 6/12/39

History: Pain in L. chest 4 days.
Shivering at onset.
Cough with sticky sputum 4 days.

4 days prior to admission he had a fit of shivering, followed by a pain in his L. chest. He had a slight cold and cough with sticky sputum. He attempted to carry on with his work but felt so ill he had to go to bed. No previous illnesses except childish ailments.

Examination: T. 96.0 P. 152. R. 48 BS N/R.
ESR N/R BP 110/60 Ht. 5'11 3/4" Wt. 10-0 Sp.TB -ve x6

Left hydropneumothorax, with bloodstained fluid in the pleural sac, which contained no organisms.

Follow up: Well and has had no recurrence in the 10 yrs. 2 mths. F.U. period.

Case 60. 32/4246 W.N. (30 M. Engineer (Turner)
Admitted: 15/11/40 Discharged: 14/12/40.

History: Pain in L. chest 1 day.
Dyspnoea 1 day.

Examination: T. 97.0 P. 120. R. 24 BS N/R.
ESR N/R BP 110/60 Ht. 5'7" Wt. 127 Sp.TB -ve x6

Left hydropneumothorax, with bloodstained fluid in the pleural sac, which contained no organisms.

Follow up: Well and has had no recurrence in the 10 yrs. 2 mths. F.U. period.
He was suddenly awakened from sleep by the onset of an acute pain in the L. side of his chest. He had had a cough with a small amount of white frothy sputum for one wk.

Examination:  T. 98.2  P. 84.  R. 24.  BS +ve.  
BSR N/R  BP 138/96  Ht. N/R  Wt. 10-11

Left pneumothorax with usual signs.

Follow up: Well and has had no recurrences 9 yrs. later but own Doctor states that the patient's sister showed clinical signs of TB. in 1943.

Case 61.  MOPD/47  J.F.  (42)  M. Miner.

P.P. (Seen as O.P. May 1947).

History:  Pain in region of R. Nipple - a few days.  
Haemoptysis - a few days.  
Cough with little sputum many yrs.  
Dyspnoea on exertion some yrs.

A few days before he was seen as an O.P. he had a pain in the R. chest associated with a slight amount of blood in the sputum. He has had a cough for many years, and suffers from breathlessness on exertion.

Examination:  T.  P. and R. Normal.

Right pneumothorax, the only clinical signs being a few scattered crepitations and an occasional rhoncus. X-ray shows marked emphysema of the R. lung with a small apical pneumothorax.

Follow up: No recurrence in 2 yrs. 2 mths. F.U.


P.P. (Seen as O.P. 22/1/48).

History:  Sudden pain in L. chest 1 day.  
Slight dyspnoea on exertion 1 day.

While walking down the road for lunch the day before he was seen as an O.P. he was suddenly seized with a severe stabbing pain in the L. side of his chest. The pain eased when he rested but came on again when he exerted himself. He had had no previous illnesses except childish ailments. Maternal grandmother has asthma.
Examination: T. Normal, P. 90. R. normal. BS N/R

Left pneumothorax, without marked physical signs but diagnosis confirmed by X-ray.

Follow up: 18 mths later has had no recurrences, and plays football and tennis regularly without ill effects.

Case 63. 31/3680 D.G. (24) M. Plumber.


History: Sudden pain in R. chest 2 days ago, Dyspnoea on exertion 2 days ago.

While having his breakfast 2 days prior to admission he was seized with a sudden severe pain in the R. upper chest. The pain was worse on coughing and deep breathing. He had no cough or sputum only tonsillitis and no chest trouble previously.

Examination: T. 98.2. P. 80 R. 24 BS. +ve
BSR 5 mm. BP 144/100 Ht. 5'10" Wt. 11-4

Right pneumothorax with typical signs. X-ray shows some displacement of the mediastinum.

Follow up: His family Doctor states he has had no recurrences and there is no TB history in the family or near relatives. 19 mths. F.U.

Case 64. 31/4180 A.M. (30) M. Rly. Engine Driver.


History: Sudden pain over L. lower chest and shoulder for 24 hrs., Dyspnoea - 24 hrs.

While painting in his house he was seized with a sudden severe pain in the L. lower chest and the L. shoulder. Breathlessness followed the onset of the pain. He had had a cough for a few months with a little frothy sputum, but had been discharged from the Forces Cat. A.1.

Examination: T. 97.8 P. 104 R. 28. BS. N/R

Left pneumothorax, with typical signs clinically.
Progress: He settled down in the ward comfortably, but apparently turned over in bed and expired suddenly without any warning.

Permission was not given for a post mortem examination.

Case 65. 32/8632 L.R. (24) M. Medical Student.
P.P. Admitted 4/7/47 Discharged: 8/7/47.

History: Pain in R. chest 1 day.
Dyspnœa 1 day.
Head cold and cough 2-3 days.

He had had a cold in the head for 2 or 3 days prior to admission, and on the morning of admission while coughing, he felt a sudden pain in the R. side of his chest followed by a feeling of tightness in the chest, which gradually changed to a dull aching pain made worse by coughing and deep breathing. He managed to attend for the last of his clinical examinations in his Final Examination and was admitted to the Ward thereafter when he came for advice about this pain in his chest.

Examination: T. 101 P. 86 R. 20 BS. -ve

Right pneumothorax, with usual clinical signs.
No disease in the lungs at the time or after re-expansion. Bifid R. 4 rib.

Follow up: Well and has had no recurrences 2 yrs. later. 3 mthly. X-rays have been consistently -ve.


Admitted 5/4/43 Discharged 7/6/43

History: Pain in chest 14 days.
Dyspnœa 7 mths.

In Sept. 1942 she "caught a chill" which was associated with a cough and a copious greenish spit. She was in bed at that time for 9 wks, and has lost about 1 stone in weight in the past yr. Since she caught the chill she has had a tight feeling in the chest and has been "short of breath". 4 mths prior to admission she noticed a slight swelling at the root
of her neck, and began to feel nervous and feel the heat much more. These symptoms were worse during the attacks of breathlessness. She had measles and whooping cough in childhood and a spontaneous pneumothorax 8 yrs. before the present incident. She has a brother who has had a spontaneous pneumothorax, and she has two children the younger of whom (aged 2½ yrs) suffers from infantile dermatitis and asthma.

Examination:  T. 93.4  P. 78  R. 28  BS. N/R  
BSR N/R  BP N/R  Ht. 5'0¾  Wt. 10-10  Sp Mucopurulent.

Right pneumothorax, with displacement of the mediastinum to the left side. X-ray shows some fluid in the pleural space.

Progress: Air was aspirated from the pleural space but the lung failed to expand, and she was therefore placed on the W.L. for Bangour Hospital where her R. lung was eventually removed by Mr. W. Mercer.

Follow Up: 7 yrs. later was seen and had spirometric test. Some fairly severe dyspnoea on exertion, and weight has increased by 1½ stone. No further trouble with her chest apart from the dyspnoea, though her ankles tend to swell at night if she walks a lot.

Case 67. 22/7599  T.T. (27) M. Publisher's Agent.  

History: Pain in the R. shoulder and chest 6 days. 
Dyspnoea 6 days.

While rising from bed in the morning 6 days prior to admission he was seized with a severe pain in the R. shoulder and R. chest. He had no previous cough or sputum. In 1940 while in the Forces he had a gunshot wound of the L. chest with collapse of the lung and the development of an effusion.

Examination:  T. 97.0  P. 86.  R. 22  BS. N/R  
BSR 8 mm.  BP. 110/80  Ht. 5'10¼  Wt. 9-7

Right pneumothorax with typical signs. Cyanosis was noted. A shadow at the R. apex was noted in the X-ray films and the pneumothorax was maintained artificially up to the time of the F.U. No TB apparently ever been recognised in the sputum.

Follow up: Well and still attending for weekly refills of the pneumothorax 16 mths. later.
**Case 68.** 32(19) 8494 T.M. (29) Medical Proctnr.

P.P. Admitted: 25/1/47 Discharged 12/2/47.

**History:** Sudden pain in R. chest 2 hrs. Acute dyspnoea 2 hrs.

While lifting his case down off the rack prior to leaving a train he was suddenly seized with a severe pain in the R. side of his chest. He managed with difficulty to reach the ward in the Infirmary, and became much more dyspnoeic suddenly while removing his jacket for examination of his chest. While in the RAFVR before 1939 he had had a small haemoptysis and X-ray at that time revealed a small cavity at the L. apex. He was in a sanatorium for 9 mths. where TB was isolated from his sputum, and a L. artificial pneumothorax was induced. This pneumothorax was kept up during his student career until 1945, when the lung was allowed to re-expand. His health otherwise had been good and he had had no previous episodes of spontaneous pneumothorax.

**Examination:** T., P. and R. charts missing BS ve. BSR 3 mm. BP N/R Ht. N/R Wt. N/R Sp TB. ve x2

Right pneumothorax, with typical signs in the chest. Tension developed and rapidly recurred after aspiration of air with a syringe. Under water drainage was instituted with success. X-rays of chest showed only healed apical focus of TB at L. apex. after recovery and no lesion in the R. lung.

**Follow up:** Well and in active General Practice in the Midlands of England 3 yrs. 3 mths. later having had 1 slight recurrence 2 mths. after discharge.

**Case 69.** 26/1184 F.B., (30) M. Window-Cleaner.

Admitted 26/3/38 Discharged 18/4/38.

**History:** Pain in L. chest 1 day. Dyspnoea 1 day. Dry cough with no sputum 1 day.

On the day before admission while walking to his work he was seized with a severe pain between the shoulder blades associated with sudden breathlessness. The pain moved round to the left side of his chest anteriorly. 1 mth. before he had had a similar attack, which also came on when he was walking. As a child he had had double pneumonia, and middle ear
disease since the age of 12 yrs.

Examination:  T. 97.0  P. 60  R. 20  BS -ve.
BSR N/R  BP 125/80  Ht. 5'11"  Wt. 9-12.

Left pneumothorax with typical clinical findings.

Follow up:  Was found to have died during the F.U. Period from a perforated D.U. in the Western General Hospital.  Post mortem confirmed the diagnosis, and showed pleural adhesions at the apex of the L. lung with fibrosis.  No pneumothorax.

Case 70.  23/9868  E.M.  (31)  M.  Labourer in Mill.

History:  Stabbing pain in R. chest 6 hrs.
Dyspnoea 6 hrs.

After lifting a bag of flour, he began to feel a pain in the R. side of his chest. This pain became gradually more severe but he had no cough or spit. The pain was worse on deep breathing coughing or moving. He had pyelitis in 1939.

Examination:  T. 97.0  P. 80  R.  BS. -ve
BSR N/R  BP 118/72  Ht.  N/R  Wt.  N/R.

Right pneumothorax, with few signs.  X-ray shows some fluid which later absorbed leaving some pleural thickening.  Some basal emphysema is noted.

Follow up:  Not traced.

Case 71.  27/2926  A.S.  (47)  M.  Grocer.

Admitted 2/9/43  Discharged 9/10/43
(Seen MOPD 1/11/41 for first time).

History:  Dyspnoea on exertion and lying down.
Cough with whitish sputum.

When seen in 1941 this patient had a history of dyspnoea especially when lying down in bed at night, and some pain in the L. chest. Later this dyspnoea became worse and he complained at the time of admission of a "numbness due to his lung pressing on his heart". He was often subject to heavy colds, and has had measles, pleurisy and bronchitis.
Examination: (After admission 2/9/43)
T. 97.0 P. 80 R. 26
BSR N/R BP 138/98 Ht. 5'3" Wt. 6-12 Sp. TB -ve x2

Left pneumothorax, with no physical signs, at first examination (1941). Localised apical emphysema later became fibrosed and he was admitted (1943) to exclude tumour. Calcified or fibrosed area noted at R. apex also. Emphysema.

Follow up: Not traced after 1947, but well then.

P.P. Admitted 21/7/44 DIED: 21/7/44.

History: Dyspnoea becoming more acute 2 days.
Vomited 2 days.
Feeling sick 3 days.

About 3 days prior to admission he felt unwell and stayed off work. The day before admission he vomited, and became slightly breathless, and the day of admission he went over to the sink to be sick and after retching became much more breathless.

Examination: T. subnormal P. impalpable at the wrist and inaudible at the apex. gasping with the chest in the position of inspiration. Unconscious, with muscular spasms in limbs, and opisthotonos. Died in less than half an hour after admission.

Bilateral pneumothorax, diagnosed at post mortem examination.

(Autopsy report given in the Section of this Thesis where these are recorded.)

Case 73. 23/C/3401 P.M. (23) M. Rubber Worker.


History: Stabbing pain in front of chest 3 days.
Dyspnoea.
Slight cough with "slaty" sputum 3 days.

He was suddenly seized with a pain in the front of his chest, associated with dyspnoea and with a cough and a "slaty" sputum. No previous illnesses except "Influenza".
Right pneumothorax with usual physical findings. X-ray shows some deviation of the mediastinum but no pathological change in the lung.

Follow up: Not traced.

Case 74. 28/0/5431 F.S. (39) M. Electrician.

Admitted: 16/11/34 Discharged: 18/11/34

History: Pain in L. chest 3 days.

While riding a motor cycle after lunch 3 days before admission, he suddenly felt a pain over the 3rd, 4th, and 5th ribs just outside the mid-clavicular line. The pain eased off and then recurred and was then also felt below the L. scapula. 4 years before he had had a similar attack which came on at rest, and 15 years before he had had pneumonia.

Examination: T. 97.4 P. 84 R. 30 BS. N/R.
BSR N/R BP N/R Ht. 5'10" Wt. 310

Left pneumothorax with usual clinical signs. X-ray show slight deviation of the mediastinum and some fluid in the costo-phrenic angle. Expansion uneventful and showed normal lung fields.

Follow up: Well and has had no recurrences in the 14 yrs. 9 mths. P.U. period.

Case 75. 26/E/7788 J.T. (37) M. Miner.

Admitted: 9/12/32 Discharged: 5/1/33.

History: Pain in back of R. chest 1 day. Slight dyspnœa.

While at work he was suddenly seized with a severe pain in the R. side of the back of his chest.

Examination: T. 98.2 P. 104 R. 30 BS. ve
BSR N/R BP 110/70 Ht. 6'0" Wt. 10-10½

Right pneumothorax with typical signs on clinical examination. X-ray confirms, and shows an adhesion to the chest wall. Re-expansion shows no disease in the lungs.
Follow up: Well and has had no recurrence 16 yrs. 9 mths. later.

Case 76. 31/G/1468  J. Mc. (23) M. Motor Driver.

Admitted: 13/10/32  Discharged 8/11/32.

History: Slight pain in L. chest 8 days. Severe pain in L. chest 1 day.

While standing still 8 days ago he was suddenly seized with a severe pain in the L. side and front of his chest. This severe pain eased off but on the morning of admission he was again seized with a severe pain in his chest which also came on when he was standing still. He went slowly home and went to bed and was later sent up to the Infirmary. His only previous illnesses have been measles, mumps and diphtheria.

BSR N/R  BP 110/75  Ht. 5'9"  Wt. 105

Left pneumothorax with typical signs. Some rigidity was noted over the upper part of the L. rectus abdominis. A considerable amount of fluid was noted in the L. pleural sac on X-ray examination. A total of 30 fl. ozs. of bloodstained fluid was aspirated, which contained many RBC's and numerous lymphocytes with some polymorphs many of which were eosinophils.

Follow up: Not traced. (In lodgings).

Case 77. 31/G/2487  A.B. (20) M. Student.

Admitted 1/7/32  Discharged 4/7/32.

History: Sudden pain in R. side - a few hrs.

While at a party he was suddenly seized with a severe pain in his R. side and collapsed. He had had a slight cold in the head for a few days previous to the incident.

Examination:  T. 97.4  P. 80  R.20.

Right pneumothorax. (The physical findings are not detailed, but the apex beat is noted as being displaced) The diagnosis was confirmed by X-ray.
Follow up: Now in Ontario, Canada, whence he writes that he has had one recurrence, and that two chest X-rays within the last 2 yrs. have been reported as "negative". 17 yrs. 1 mth. F.U.

Case 78.  31/G/3333  G.S. (26)  Bottle Worker.
Admitted: 27/12/33  Discharged: 1/2/34

History: Pain in R. lower chest and upper abdomen for 5 hrs.

He had just risen from his bed when he was seized with this severe pain in the lower part of his R. chest, and the upper part of his abdomen. He had had "pleurisy" on the R. side 10 yrs. before, and measles in childhood. In the June prior to admission he had had muscular rheumatism in his arms and legs.

Examination: T. 98.4  P. 104  R. 48  BS. N/R
BSR N/R  BP 148/56  Ht. 5'8"  Wt. 9-12.

Right pneumothorax with typical physical findings. X-ray shows an adhesion at the apex of the lung, but on re-expansion there is no evidence of any lung disease.

Follow up: No recurrence in 15 yrs. 8 mths. F.U.

Case 79.  31/G/6294  J.D. (69)  M. Retd. Clerk.
Admitted: 22/5/38  Discharged: 21/6/38

History: Pain in R. chest - a few hrs.
Dyspnoea - a few hrs.

While straining at stool in the morning, he felt a sudden pain in the back of his R. chest. This pain was associated with dyspnoea, and he has had a slight cough since he had bronchitis some 5 mths. before. He had been perfectly healthy until he had this bronchitis.

Examination: T. 96.4  P. 108  R. 26  BS. -ve.
BSR N/R  BP 140/80  Ht. 5'7"  Wt. 12-92

Right pneumothorax, with usual signs. The lung on X-ray shows chronic inflammatory changes.

Follow up: His son writes that he was told at the
time of his father's discharge from the Infirmary he was told that he would have "a cough for the rest of his life". This is not the case for he had no further cough after leaving the ward. He is still alive aged 81, and though in poor health, has had no further trouble with his chest. 11 years. 3 mths. F.U.

**Case 80. MOPD/49 R. M. (27) M. Rubber Worker.**

**P.P.** Seen in MOPD 15/8/49

**History:** Pain in L axilla 1 day.

While walking about his house about 4 p.m., he was suddenly seized with a severe pain in the L. axilla and side of the chest. He was not breathless at the time of onset of this pain, and had no cough. He had had no previous similar attacks and no other chest troubles except measles in childhood.

**Examination:** T. 97.8 P. 72 R. 18 BS. -ve.

BSR 6 mm. BP 105/70 Ht. 5'7" Wt. 7-11½

Left pneumothorax with no physical signs except diminished vocal resonance in the region of the L. upper lobe. X-ray shows partial collapse of the L. upper lobe and no disease in the lung.

**Follow up:** No recurrences 6 mths. later.

**Case 81. 31/3279 R.D. (17) M. Miner.**

Admitted: 2/10/47 Discharged: 18/10/47.

**History:** Pain in R. chest 1 wk.

As he was going in to a shop he was suddenly seized with a severe pain in the R. side of his chest. The pain was felt over the front and the back. He sat down, sweated and vomited and was very breathless. He had had a similar but less severe pain 10 wks. before. He had no cough or sputum. In February 1947 he had "pleurisy", when he was in bed 3 wks. and he had measles in childhood.

**Examination:** T. 98.0 P. 80 R. 22 BS. -ve.

BSR 6 mm. BP 136/70 Ht. 5'7" Wt. 8-0½ Sp TB -ve.

Right pneumothorax with usual findings. No evidence of lung disease and lung re-expanded
satisfactorily.

Follow up: This boy has had 4 recurrences since the one for which he was admitted, in the 1 yr. 9 mths. F.U. Each side has been affected but not at the same time.

Case 82. 10/8089 J. Mc. (25) M. Solicitor.

Admitted: 8/1/49 Discharged:

History: Pain in epigastrium 3 hrs.
Slight cough 3 hrs.

While sitting quietly by the fire he was suddenly seized with a severe pain in the epigastrium. The pain became more severe when he stood up and he was sent in to the R.I.E. He had had a mastoid operation at the age of 10 yrs. whooping cough aged 4 or 5 yrs. and his tonsils were removed in 1930. He had no previous chest illnesses and no dyspepsia and had been discharged from the Forces Category A.

Examinations: (Ward charts missing).

Right pneumothorax, which was not diagnosed till a laparotomy had been performed, the case being mistaken for a perforated peptic ulcer.

Follow up: Well and has had no recurrences. The X-ray appearances of the lungs are normal 1 yr. later when seen at F.U.


Seen at MOPD 20/8/48.

History: Sudden pain in R. chest 10 days ago.
Dyspnoea 10 days.

While in bed at 3 a.m. he was suddenly awakened by the onset of a severe pain in the R. chest. The pain was worse on deep breathing. No previous illnesses of note, but one brother was in Bangour Hospital for observation as ? TB. Wife has asthma.

Examination: T., P. and R. normal.
BSR 2 mm. BP N/R Ht. 6'2" Wt. 10-10
Right pneumothorax with slight physical signs. X-ray shows apical pneumothorax with some generalised emphysema.

Follow up: Well and has had no recurrences when seen 1 yr. 3 mths later.

Case 84. 22/8441 W.D. (41) M. Civil Engineer.

Admitted 22/12/49 Discharged 27/12/49.

History: Sudden pain in L. chest 4 hrs. Dyspnoea 4 hrs.

While going to his office in the morning he started to sneeze and was seized with a severe pain in the L.side of his chest posteriorly. The pain was worse on deep breathing and he was unable to drive his car and was brought in to the Infirmary in an ambulance. He had "pleurisy" when at school, and was discharged from the Royal Marines because of a D.U.

Examination: T.98.4 P. 66 R.22 BS. N/R
BSR 5 mm. BP 110/70 Ht. N/R Wt. 10-10.5

Left pneumothorax, which showed only slight clinical evidence, and none on X-ray taken 4 days later. A systolic clicking sound was noted at the apex beat.

Follow up: N.F.U.

Case 85. 32/10252 A.S. (46) M. Miner.

P.P. Admitted: 27/7/49 Discharged 19/8/49.

History: Tight band across chest 10 wks. Dyspnoea 10 wks.

He began to have a cough 7 mths. before admission, and was seen as an OP. 10 wks before admission when he had signs of bullous emphysema and a L. pneumothorax and large bullae. Since that time he has had a gripping sensation in the chest and dyspnoea on exertion. He had pneumonia in 1930.

Examination: T.97.0 P.96. R.22 BS -ve.
BSR N/R BP 120/80 Ht. 5'6" Wt. 10-5

Left pneumothorax with marked bullous emphysema.
Follow up:— Has had a slight recurrence on the L. side on F.U. over 7 mths.

Case 86. 27/5641 G.F. (28) M. Police Constable.


History: Pain in R. chest 2 hrs.

He gave a short cough and was seized with a pain - "like a knife" - in the R. side of his chest in the axillary region. He had had a similar type of incident while in the Army 2 yrs. before. He had whooping cough and measles in childhood and had his appendix removed while in the Army. He seldom has a cold in the head.

Examination: (Charts missing) P.70 BS. -ve. BSR 2 mm. BP N/R Ht. 6'1½'' Wt. 12-0 Sp. TB -ve.

Right pneumothorax, with typical signs. X-ray shows a small amount of fluid as the lung re-expands.

Follow up: Has had one recurrence 14 mths later while walking, 2 yrs. F.U.

Case 87. 23/15397 J.H. (33) M. Plumber.

Admitted 5/12/49 Discharged: 6/1/50.

History: Pain in R. chest 1½ hrs.

While having a cup of tea in the course of his work he was suddenly seized with a sudden pain in the R. side of his chest. He became very breathless. He had not been doing any strenuous work or heavy lifting prior to the incident. He had had no previous trouble with his chest, and was discharged from the Navy Category A.1. 1 brother discharged from The Forces with "chest" trouble.

Examination: T.97.0 P.98 R.20 BS. +ve. BSR 12 mm. BP 110/70 Ht. 5'7'' Wt. 10-2½

Right pneumothorax with typical clinical features. X-ray shows complete collapse of the lung and no underlying disease.

Follow up: Well and has had no further trouble with his chest when seen 4 mths later.
Case 88. 28/7842 J.K. (29) M. Occupation N/R

Admitted: 20/12/49 Discharged 9/1/50.

History: Pain in front of chest 1 day.
Choking sensation in neck 1 day.

While getting dressed in the morning, he was suddenly seized with a severe pain in the front of his chest behind the sternum, and a tightness in the neck. He was also dyspnoeic. 8 weeks before he had bronchitis.

Examination: T. 96.8 P. 90 R. 28 BS. +ve.
BSR N/R BP 110/56 Ht. N/R Wt. N/R.

Right pneumothorax with typical clinical signs. X-ray shows complete collapse of the lung, with no evidence of underlying disease.

Follow up: N.F.U.

Case 89. 32/9400 P.H. (27) M. Factory Foreman.


History: Acute pain in L. chest 2 hrs.
Dyspnoea 2 hrs.

While sitting in a bus 2 hrs. before admission he was seized with a severe pain which radiated from the L. iliac fossa to the chest and up to the shoulder. The pain at the time of admission is now localised to a region round the L. mid-chest anteriorly. He has shoulder pain on the L. side on inspiration, and the pain is eased by sitting up. He has no cough or sputum. He has had "pleurisy" 7 times in 7 years. His mother suffers from bronchitis, and a sister has "recurrent pleurisy".

Examination: T.98.4 P. 70 R.24 BS. N/R
BSR 2 mm. BP 108/62 Ht. 5'10" Wt. 9-10

Left pneumothorax with faint but apparently definite signs. X-ray shows small apical pneumothorax with congestive changes at both bases.

Follow up: Has had one recurrence since he was in the Royal Infirmary. His sister was also traced and the Radiologist at the Hospital where she was X-rayed on the occasion of her last incident reports only "No focal lesion seen".
Case 90. 23/11412 P.S. (45) M. Joiner.

Admitted: 31/1/43  DIED 31/1/43.

History: "Asthmatic attack" 2 days.
"Asthmatic attacks" since 1918
Cough with purulent sputum 1 mth.

He had an attack of acute bronchitis which started 1 mth. before admission. Since being gassed in the 1914-1918 War he has suffered from periodic attacks of acute dyspnoea. He was sent in with what appeared to be one of his periodic attacks and had a sudden spontaneous pneumothorax when the House Physician was not present and died almost at once.

Examination:  T. 100.2  P. 120  R. 44.

Left pneumothorax diagnosed at post mortem examination.

Post mortem report is recorded in the section where these are recorded.

Case 91. 23/15275 A.C. (27) M. Miner.

Admitted 22/9/49  Discharged 1/10/49.

History: Pain in R. side of chest 15 days.

15 days ago while lifting a heavy weight he felt a pain over the lower part of his R. chest. He was not breathless at that time and after a short rest he finished his shift. The following morning at 3 a.m. he was awakened by the increased severity of the pain and felt nauseated. Moving about in bed made the pain appear and it was worse on deep breathing. He had had pneumonia 2 yrs. before.

Examination:  T. 97.0  P. 86  R.22  BS. N/R
BSR 3 mm.  BP N/R  Ht. 5'11½  Wt. 10-6

Right pneumothorax with typical signs. X-ray shows no disease as the lung expands.

Follow Up:  Well and has had no recurrences 5 mths. later.
Case 92. 27/3245 A.Mc. (47) M. Motor Driver.

Admitted 27/3/44 Discharged 2/5/44.

History: Pain in R. chest 6 hrs.

When stepping down from his lorry he was seized with a severe pain in the R. side of his chest extending from the sternum to the axilla. It was a "strangling type of pain", and was associated with a slight cough with a frothy sputum.

Examination: T. 97.0 P. 80. R.30 BS. N/R
BSR N/R BP 96/70 Ht. 5'8½" Wt. 12-10.

Right pneumothorax which required aspiration of 800 ccs. of air. Typical signs and X-ray showed complete collapse of the lung.

Follow up: Seen 5 yrs 11 mths. later. Is well and has had no recurrences.

Case 93. 28/7969 J.C. (42) M. Occupation N/R

Admitted 28/2/50 DIED: 28/2/50

History: Cough with sputum 1 wk.

Shortness of breath 3-4 years.

5 days before admission he set out for work but collapsed and was very breathless. No history of pain in the chest, but he has been getting gradually more breathless on exertion for the past 3 or 4 yrs.

Examination: T.97.0 P.140 R.50 BP 124/90.

Left pneumothorax, the diagnosis being made at autopsy. He was pale and slightly cyanosed on admission, with numerous rhonci and crepitations throughout the chest. He was given morphine after admission but he collapsed and died suddenly shortly after.

Autopsy report is recorded in the Section devoted to post mortem examinations in this Thesis.
Case 94. 28/ H.G. (20) M. Commercial Traveller.

Admitted: 7/3/50  Discharged:

History: "Pressing feeling" in R. axilla and R. side of neck 12 hrs.

While playing cards quietly he gradually became conscious of a pressing feeling in his chest, mostly on the R. side. He was not breathless at rest with this feeling. On previous occasions he has been conscious of a slight "stitch" in his side, and always on the same side. He used to play a lot of football and tennis. He had had whooping cough and pneumonia in childhood and was discharged from the Army with a "threatened D.U."

Examination: T.97.0  P.74  R.20  BS. -ve.
BSR 3 mm. BP 118/68  Ht. 5'6½"  Wt. 9-4

Right pneumothorax with typical physical signs. X-ray shows no lung disease, and normal re-expansion.

Follow up: N.F.U.

Case 95. 31/S/271 J.S. (27) M. Plumber.

P.P. Admitted: 8/2/50  Discharged 18/2/50.

History: Pain in L. Chest 7 days before admission.

While walking across the room in his house, after rising from a chair in the morning he was suddenly seized with a severe pain in the L. chest. He had had a slight cold in the head for a few days before.

Examination: T.97.4  P.90  R.20  BS. -ve.
BSR 5 mm. BP 124/80  Ht. 5-10  Wt. 10-6  Sp.TB -ve.

Right pneumothorax with typical signs. When seen first as an O.P. he had a "clicking" sound synchronous with his cardiac systole. This gradually cleared but could still be heard if the patient leaned forward and to the left. X-ray showed no lung disease, and follow up films showed normal re-expansion.

Follow-Up.  N.F.U.
Case 96. 27/5488 P.B. (3) M. Electrical Engineer.

Admitted 29/11/47 Discharged: 16/12/47

History: Pain in L. chest 30 hrs.
Breathlessness 30 hrs.
Cough 3 days.

At 3 a.m. on the day prior to admission he developed a sudden severe pain in the L. chest. He found he was "gasping for breath", and the pain spread up the centre of his chest and "nearly choked" him. He had had "pleurisy" in 1938 and pneumonia in 1944.

Examination: T.97.2 P.96 R.24 BS. N/R
BSR 4 mm. BP 150/90 Ht. 6-12 Wt.10-74 Sp. TB -ve

Left pneumothorax with typical signs. "Friction" sounds were noted over the precordium, and an X-ray film shows evidence of air in the mediastinum.

Follow up: Now is "100% fit" and has had no further trouble with his chest. Vital capacity when seen on F.U. 2 yrs. 5 mths. later is 4 litres.

Case 97. 30/9135 N.P. (23) M. Police Constable.

Admitted: 4/2/50 Discharged 7/3/50.

History: Pain in R. chest - some hrs.
Dyspnoea - some hrs.

While asleep in bed, he was suddenly awakened at 1.30 a.m. by a severe pain in the R. side of his chest. On previous occasions he has had attacks of pain in the region of the L. shoulder, which he has put down to "rheumatism". He had "dry pleurisy" on the R. side of his chest in 1944.

Examination: T.97.0 P.80 R.20 BS. +ve.
BSR 2 mm. BP 126/60 Ht. 5'10" Wt. 9-7

Bilateral pneumothorax, but signs clinically were only obvious on the R. side. The L. apical pneumothorax was only seen on X-ray films of the chest.

Follow up: N.F.U. Was expanding satisfactorily when seen 6 wks. after the incident.
Case 98. 23/8156 W.M. (24) M. Occupation N/R.
Admitted: 28/7/36 Discharged 27/8/36.

History: Pain at the lower angle of R.scapula 14 days prior to admission.

While standing 14 days ago he suddenly felt a pain at the inferior angle of his R. scapula. The pain was of a stabbing nature and spread over the whole of the R.chest and upper R. abdomen. The pain was worse on deep breathing and exercising, and was eased by lying flat. He was dyspnoeic on slight exertion but he had no cough. He has had 9 similar attacks in the last 4 yrs., 4 on R. side and 5 on the L. Attacks come on regardless of his state of physical activity. He has never had asthma, but often has had tonsilitis and quinsy, and never had any trouble with his chest until 4 yrs. before admission.

Examination: T.98.4 P.70 R.20 BS N/R.
BSR N/R BP 140/85 Ht. 5'3" Wt. 8-12.

Right pneumothorax, with typical signs. X-ray shows R. pneumothorax with fluid and gross bullous emphysema, and Lipiodol shows bronchial tree ending in dilated sacs.

Follow up: Not traced.

Admitted 21/7/41 Discharged 5/8/41.

History: Cutting pain in R. chest 6 wks. before. Dyspnoea, severe at onset, now less so. Cough 10 mths.

Immediately after getting out of bed 6 wks. prior to admission was seized with a severe pain in the R. side of the chest. He was very dyspnoeic at the time of onset of the pain. He has had a cough for the past 10 mths. and is working amidst hydrochloric acid fumes. He has lost a stone in weight over the past year. He has had dyspepsia for many years.

Examination T.98.0 P.100 R.24 BS. N/R
BSR 4 mm. BP 130/80 Ht. 5'4" Wt. 8-12 Sp. TB. -ve

Right pneumothorax with typical signs. X-ray shows signs of chronic bronchitis and emphysema.
Follow up: Was admitted to Wa. 32 R.I.E. in 1947 with a haematemesis. Seen again 8 yrs. 8 mths. after the incident, has remained well and has had no recurrences.

Case 100. 31/3/331 J.S. (48) Tramcar Fitter.


History: Swelling of ankles 1 wk.
Severe dyspnoea 1 wk.
Attacks of dyspnoea 6 yrs.

1 wk. prior to admission he had to give up work on account of severe dyspnoea. This breathlessness has been waking him up at night, and he has a frothy white sputum. The attacks of breathlessness last about an hour at a time, and the attacks date from a spontaneous pneumothorax he had 6 or 7 yrs. ago. He often has a tight "vice-like" feeling over the front of the chest.

Examination: T.98.0 P.100 R.34 BS. N/R
BSR BSR BP Ht. N/R Wt. N/R.

Acute right heart failure. This patient had a L. spontaneous pneumothorax 6 years before. X-rays of the chest show bullous emphysema on this occasion, with thickened pleura.

Autopsy report is given in the Section of this Thesis in which post mortem reports are recorded.