M.D. THESIS

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on

IDIOPATHIC HAEMOPNEUMOTHORAX.

by

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PREFACE.

This study, extending over the past four years, was mainly conducted at Whipps Cross Hospital, London, E.11. It is a pleasure to express my appreciation to Dr. Eric Smith, Consultant Physician, for permission to supervise cases under his care. I am also indebted to: Dr. J. Fawcett, Consultant Physician St. Andrew's Hospital, Billericay, Essex for allowing me to record Case 6; Mr. Geoffrey Flavell (London Hospital) for authority to refer to the operative findings in Cases 5 and 6; to Dr. Keith Simpson (Guy's Hospital) for the autopsy-report on Case 3, which Dr. H. H. Kenshole, H.M. Coroner permitted me to record.

It appeared opportune to the author to prepare a monograph on this subject, since existing accounts are widely scattered, and not readily amenable to the general physician.

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"There is no reference to Spontaneous Haemopneumothorax in the Index Catalogue of the U. S. Library, nor is it discussed in any of the standard treatises on medicine, in either English, French or German."

Newton Pitt (1900).

IDIOPATHIC HAEMOPNEUMOTHORAX.

Introduction.

The entity, idiopathic haemopneumothorax, also known as benign spontaneous haemopneumothorax (Hyde and Hyde, 1951) and spontaneous idiopathic haemopneumothorax (Fry et al., 1955) is gaining wider recognition. Two recent publications (Fry et al., 1955; Calvert and Smith, 1955) have outlined many of its salient features. The limited scope of these accounts, however, indicated the need for a re-analysis to include previous omissions and more recent contributions.

This study purports:

(1) to collate the entire literature up to the end of 1955, since no accurate and detailed analysis is available for ready reference. Indeed, standard treatises on pulmonary diseases, with the notable exception of Davidson's (1935), merely recognise that this condition exists. In Davidson's textbook, two of his three fatal cases of spontaneous haemothorax are described.

(2) to present eight illustrative case-reports, which are collectively representative of the varied problems. Three of these cases, and a fourth in postscript, have been reported (Calvert and Smith, 1955), when it was stated that the relevant literature comprised about 150 case-reports. It is now apparent that this was a distinct under-estimation, a criticism also valid to the total of 174 cases cited by Fry and colleagues. This discrepancy was due to lack of an exhaustive exploration of all indirect sources of information. For instance, cases have been reported as complications of spontaneous pneumothorax, in

discussions on such topics as haemothorax, haemorrhagic pleural effusions, and the therapeutic applications of pulmonary decortication and of the intrapleural injection of fibrinolytic enzymes. Moreover, additional well-documented cases have been recorded in foreign literature, which is neither listed in the standard indices of references nor readily acquired.

(3) to ascertain the aetio-pathogenesis by scrutinising the available data from autopsy and operation, and by examining the evidence for an aetiological identity or distinction between idiopathic haemopneumothorax and a/ idiopathic pneumothorax, b/ idiopathic haemothorax, c/ the tuberculous types of pneumothorax and haemopneumothorax and d/ the haemothorax, which occasionally follows artificial pneumothorax.

For convenience, this presentation comprises four sections, which necessarily overlap.

Section I is devoted to a brief elucidation of the nomenclature and classification, prior to a rather lengthy historical survey. Then the author's 8 patients are described and, in turn, 240 cases of idiopathic haemopneumothorax are summarised (Table I). The 12 cases of idiopathic haemothorax are presented elsewhere (Table 13).

Section 2 provides a statistical analysis of the more important aspects of the cases recorded in Table I.

Section 3 deals with the elusive problem of aetio-pathogenesis. For this purpose, the available knowledge derived from autopsies, operations and relevant investigations is concisely recorded and integrated as a preliminary step.

Section 4 consists of a fairly detailed description of the clinical picture, its simulations and complications, as well as a full

discussion on the hitherto controversial question of treatment. Its re-evaluation is imperative in view of the earlier reported mortality-rates of 25 to 33 per cent, and the recent promising therapeutic developments.

SECTION I.

Definition and Nomenclature.

Haemopneumothorax signifies the association of blood and air in the pleural cavity, a rare event of varied causation. The present study, however, is restricted to the "idiopathic" or"non-specific" variety. Therefore, haemopneumothorax due to pulmonary trauma, tuberculosis, neoplasm or infarct (Rawson and Cocke, 1947; Masson and Hartman, 1949) will receive little attention. The related and, indeed, popular term, spontaneous haemopneumothorax invites clarification. The qualification, "spontaneous," denotes an intrinsic and unspecified cause and so does not strictly exclude the tuberculous type. By analogy with the connotation, "pneumothorax simplex" (Kjaegaard, 1932) for the non-tuberculous type of spontaneous pneumothorax in apparently healthy subjects, the term, "haemopneumothorax simplex" merits consideration, but not acceptance. Unfortunately, any haemopneumothorax is so potentially productive of complications that such a title, which conveys the opposite impression, is inappropriate. Fry and colleagues (1955) refer to spontaneous idiopathic haemopneumothorax, although the word "spontaneous" is virtually redundant. This idiopathic type has even been termed "benign" spontaneous haemopneumothorax (Cardenas et al., 1937; Castex and Mazzei, 1940; Hyde and Hyde, 1951) to indicate the non-tuberculous type, but the fatality-rate precludes the concept of benignity. Again, the term, haemopneumothorax has been viewed (Boland, 1900; Hansen, 1949) as less

accurate than pneumohaemothorax since the primary event is often a pneumothorax, with subsequent intrapleural haemorrhage. However, as the haemothorax constitutes the chief threat, it merits priority in description. Furthermore, it indicates more readily and correctly, as this study re-affirms, that the entity is a haemorrhagic complication of a pneumothorax. To eliminate these sources of confusion, the term, idiopathic haemopneumothorax, will be rigidly adhered to in this presentation, whilst acknowledging the clinical usefulness of the title, spontaneous haemopneumothorax, for provisional diagnosis.

Classification.

In attempting to classify patients with haemopneumothorax, confusion occasionally arises over borderline cases. A few brief illustrations will suffice. For instance, haemopneumothorax may present one or two weeks after recognised trauma (Kiaer, 1923; Morlock, 1933; Cosgriff, 1950). Of these authors' cases, only Morlock's is excluded. This patient, a boy of 14, was run over by a horse and cart one week before the recognition of his haemopneumothorax.

Again, haemopneumothorax may supervene in a patient with established pulmonary tuberculosis, which is apparently limited to the opposite side of the chest (Beatty and Frelick, case 7, 1952). The reversed sequence, but still including contralateral tuberculosis (Dorendorf, 1932; Beatty, 1939), especially when the interval is extended to several years (Myers, 1954), provides a reasonable basis for dissociating the two illnesses. These two conditions may co-exist in the same hemithorax and appear to be discrete at autopsy (Birch, 1936). Similarly, the latent pulmonary tuberculosis, discovered radiologically in the two patients of Allan (1925) might have

been fortuitous. However, none of these patients, in whom pulmonary tuberculosis was recorded, are included in this analysis.

The same arguments apply to the possible causal rôle of artificial pneumothorax when the haemopneumothorax appears several days after a refill, (Hurst and Epstein, 1937). Whereas, if the interval is several months from cessation of this treatment, other explanations are clearly necessary (Diamond, 1952).

Again, a small intrapleural haemorrhage, complicating a spontaneous pneumothorax may, unaided by thoracentesis, be misinterpreted as an ordinary pleural effusion, i.e. a hydropneumothorax. During the emergency deflation of a tension pneumothorax a blood vessel may be traumatised and lead to a factitious haemopneumothorax. Furthermore, a haemopneumothorax may be simply diagnosed as a haemothorax or pneumothorax if the complementary medium, air or blood, is only minimal radiologically. Indeed, cases of haemopneumothorax have been reported (Yater and Rodis, 1933; Korol, 1936), in which a "supernatant" air-bubble was only detectable radiographically when the patient was lying on the unaffected side. Conversely, in a patient with a true haemothorax, air may be unwittingly introduced into the pleural cavity during thoracentesis. Therefore, if the first radiograph is only taken later, haemopneumothorax will be diagnosed (Birch, 1938). Again, the air in a genuine haemopneumothorax may have been completely absorbed by the time that the patient presents for investigation (Crimm, 1948). Similarly, in haemopneumothorax with brisk haemorrhage, death may prevent the opportunity for radiography. If, therefore, the enclosed air is not readily demonstrable at autopsy, the appropriate diagnosis would be haemothorax. Whereas, if autopsy revealed a ruptured bulla, a revised diagnosis of haemopneumothorax

would be justified (Davidson and Simpson, 1940).

Due critical consideration has been directed to both nomenclature and classification as these are closely linked with the implied aetiology. The variant, idiopathic or so-called spontaneous haemothorax, which was for years entitled "haemorrhagic pleurisy" (Korol, 1936) is also considered in this study.

Historicial Survey.

In the preparation of this historical review it was intended to accentuate two points: (1) the unusual natural history of a potentially fatal condition, arising rather unheralded in otherwise previously healthy young male adults. (2) the difficulty in establishing either a clear-cut aetiology or the source of haemorrhage, even with the results of numerous autopsies readily available. The risk of monotony incurred by undue repetition of such morbid details is perhaps outweighed by their accessibility in chronological order and completeness to facilitate their close study. Besides, for ready reference a synoptic presentation (Table 9) is included later.

Although the term, pneumothorax was introduced by Itard in his thesis in 1803, the first clear account of spontaneous pneumothorax in a young adult without evidence of co-existent tuberculosis was provided by McDowell in 1856. His patient recovered and the incident was attributed to the accidental rupture of an air-vesicle.

The first clinical accounts of idiopathic haemopneumothorax were, however, as recent as the beginning of this century. Laennec (1827) had already recorded his surprise encounter of an escape of air from a

haemothorax, which was opened at autopsy, but this patient had pulmonary and pleural tuberculosis and so cannot receive uncritical acceptance for present purposes.

The first record of the diagnostic and therapeutic applications of thoracentesis to haemothorax is accredited to Whittaker (1876).

The ensuing annals of idiopathic haemopneumothorax partly depict a "procession" of deaths in young male adults and the autopsy-findings are provided in some detail to emphasize the pathological patterns. Clinical features, where considered important, are incorporated. For comparative purposes, five fatal cases reported as idiopathic haemothorax (Davidson, 2 cases, 1935; Davidson and Simpson, 1940; Cuningham, 1950, Irwin, 1951), have been conveniently included here.

Priority in the documentation of idiopathic haemopneumothorax is assigned to Pitt (1900). Indeed, the first three contributions (Pitt; Rolleston; Boland) all appeared in 1900. Collectively, these provide an instructive picture.

Pitt's patient, aged 18 years, died within 8 hours of admission to hospital. Autopsy revealed an apical emphysematous bulla as a clue to the cause of the pneumothorax and a ruptured pleural adhesion, attached to the bulla, as the presumed source of intrapleural haemorrhage. Thus, Pitt is credited with publicising the potential hazard and the possible pathogenesis of this entity.

In Rolleston's patient, aged 21, the feigned features of a perforated peptic ulcer suggested the need for laparotomy, which was only deferred by the patient's critical state. At autopsy, surprisingly, there were no signs of the pathological basis, despite a meticulous search. Although signs of a

pneumothorax were elicited during life, at necropsy there was neither an obvious escape of air on opening the affected pleural cavity nor any overt perforation of the lung. However, it is pertinent to point that these "negative" observations are quite consistent with the patient's survival for one week. It is noteworthy that in neither of these two patients was there any evidence of concurrent pulmonary tuberculosis.

Boland's patient, an American grocer aged 34, recovered uneventfully in three months, following initial repeated thoracenteses, which yielded a total of just over two litres of haemorrhagic fluid.

The next report was that of Ness and Allan (1910), whose patient was a clerk aged 31. The interesting, but by no means unique, feature was the rather long latent period of one week until admission to hospital, as compared with periods not exceeding one day in the three preceding cases. Ultra-conservative treatment, i.e. diagnostic aspiration alone, was adopted. Recovery ensued in two months. Again, there was no evidence of a tuberculous aetiology. In common, all these four patients had a history of sudden onset of simultaneous thoracic and upper abdominal pain, followed by respiratory distress and well-marked signs of shock.

Bushby's patient (1913), a clerk aged 17, recovered in three months, following the aspiration of one pint of sanguineous pleural fluid.

The patient of Williamson (1917) merits recall as, despite the total aspiration of ten litres of haemorrhagic pleural fluid, recovery was incomplete. This patient, the first reported case of idiopathic haemo-pneumothorax to undergo radiography, and of residual, and indeed marked, deformity from fibrothorax, provides one of the best illustrations of the latter (Fry et al., 1955).

The next patient, described by Fischer (1922), warrants more detailed description in view of the numerous important features recorded. This patient, a German cinematograph-operator aged 22, was subjected erroneously to laparotomy for a well-simulated perforated peptic ulcer, one week after the onset of symptoms. Death ensued during the next day. The observations at autopsy and the subsequent pulmonary histology are still frequently quoted to illustrate the aetio-pathogenesis of the idiopathic types of pneumothorax and haemopneumothorax. The right pleural cavity contained over $4\frac{1}{2}$ litres of blood, half of which was clotted. The corresponding lung-apex was studded with several prominent bullae, one of which was ruptured and its edges were coated with haematoma. Yet, in contradistinction to the observations of Pitt (1900) and many other authors (Table 9), no pleural adhesions were visible. Fischer, therefore, first described one of the accepted mechanisms to explain the onset of idiopathic haemopneumothorax, i.e. the eruption of both air and blood from a torn apical bulla. Furthermore, he demonstrated histologically that this ruptured bulla had developed from scar-tissue as a "valve-vesicle" in an otherwise healthy lung. These so-called "scar-tissue vesicles," which permit the ingress but impede the egress of inspired air, progressively inflate and rupture. This mechanism, first described by Fischer's pupil, Hayaski (1915), is the popular explanation of the pathogenesis of benign spontaneous pneumothorax.

The case-legend of Kaier (1923) outlines the fatal outcome, 3 days after the onset of symptoms, and the autopsy-findings in a 30-year old Danish rifleman. At necropsy, precisely as in Rolleston's patient (1900), there were no informative features to correlate with the haemopneumothorax.

None of the triad of standard lesions, i.e. bullae, adhesions or apical scarring, was found.

The next landmark was the report of Housden and Piggot (1931), whose patient, a 40-year old plumber, died 4 days after the onset of symptoms. Autopsy exhibited, at the apex of the affected lung, a puckered and presumably tuberculous scar, which was surrounded by two sub-pleural emphysematous blebs, to one of which two torn cord-like adhesions were attached. It was concluded, although unproven, that a perforated bleb had caused the pneumothorax, whilst the haemorrhage had arisen from the severed end of a pleural adhesion. Since, however, the bullae were intact, the site of the escaped air remains speculative while the source of haemorrhage, as in so many other instances, was not conclusively determined. Nevertheless, these post-mortem observations duplicate those of Pitt's (1900) and provide a distinct, and not uncommon, variety.

By 1932, about 200 cases of idiopathic pneumothorax (Kjaergaard, 1932) and 20 cases of idiopathic haemopneumothorax were on record and the clear-cut clinical pattern of the latter was emerging. All were young men of previous good health. All previous reports, except for the two patients of Palmer and Taft (1931) were based on individual cases.

Then, in 1932, Kjaergaard published his classical monograph on spontaneous pneumothorax in apparently healthy subjects. This detailed dissertation provided an aetiological landmark, which was also relevant to idiopathic haemopneumothorax. He advanced convincing evidence, from a study of 49 patients with spontaneous pneumothorax, that pulmonary tuberculosis was not the immediate cause in patients of previous good health.

To avoid unnecessary confusion on this point, it is important to stress that his view does not conflict with the theoretical possibility of old tuberculous apical scars producing "check-valved" bullae, the precursors of idiopathic pneumothorax.

In 1935, four fatalities were recorded. Two of these cases featured in Davidson's textbook, as illustrations of spontaneous haemothorax. Davidson's first patient, a clerk aged 28, died three weeks after the commencement of symptoms. Autopsy-examination was non-contributory, except indirectly by the demonstration of apical scars and adhesions on the side opposite to the haemothorax. Davidson's second patient, identifiable as Case I of Perry (1938), was a tailor aged 26, who died within two days of the onset of symptoms. Autopsy disclosed several apical bullae and adhesions. The latter were shown microscopically to be very vascular. The unusual site of the adhesions, i.e. between the lung and the pericardium, deserves special mention.

In the patient described by Tait and Wakely (1935), a man aged 32, the only significant autopsy-finding was a torn bulla filled with fresh blood and accompanied by eleven other bullae at the affected lung-apex. These observations, and their implied interpretation, are reminiscent of those described by Fischer (1922).

Rossel's case (1935), a French soldier aged 20, had a hitherto unique clinical picture. He developed, within six days, a spontaneous pneumothorax on the opposite side to the haemopneumothorax. Autopsy revealed multiple bilateral bullae but no definite site of haemorrhage.

The next important contribution was that of Jones and Gilbert (1936). Their patient was a law-student aged 23, whose presentation resembled acute appendicitis. The added interest in this patient was that death occurred late,

i.e. on the 38th day, and was not due to haemorrhage. Autopsy disclosed masses of intrapleural fibrin, constituting a fibrothorax. This is the only case of lethal fibrothorax recorded in the relevant literature. Death was attributed to the mechanical effects of the fibrin on the circulatory system. Neither the site of the rupture of the lung nor of the haemorrhage could be ascertained. This is not at all surprising in view of the long interval following these events, coupled with the obscuring effect of the fibrothorax. However, one bulla but no adhesion was detected on the affected side, while scarring was observed at the opposite lung-apex.

Louria (1938) reported the fatality, after 4 days illness, of an Italian barber aged 22. Autopsy showed a ruptured apical bulla, apical scarring and adhesions on the affected side. Several bulla were observed on the opposite lung. The source of the pneumothorax, but not of the haemorrhage, was discovered. Louria concluded, after scrutiny of the literature, that the aetiology and pathogenesis of idiopathic pneumo-haemothorax remained obscure, although tuberculosis apparently was not responsible. In the same year, Beaumont recorded the case-summary of a man aged 28 who, following a 3 week's illness, died on the day of admission to hospital. The results of autopsy, except for two pints of fresh blood in the affected pleural cavity, were entirely negative.

In 1940, Davidson reported his third fatality (Davidson and Simpson) from idiopathic haemothorax. However, it is apparent from the demonstration at autopsy, of a ruptured bulla, that this was really a case of haemo-pneumothorax. The patient, a soldier aged 26, died within two days of the onset of symptoms. The results of autopsy included the unequivocal identification of the source of haemorrhage. Apart from the circumstantial

evidence of a ruptured adhesion, as described by some other authors, it was witnessed in this case that blood issued from the free end of a small torn basal adhesion. Other findings included a ruptured bulla and a small "tuberculous" scar at the apex of the involved lung. The authors concluded that the ruptured bulla had produced the pneumothorax, and thereby subjacent pulmonary collapse, which caused traction on, and rupture of, the fragile vascular basal adhesion.

Lorge (1940) described his observations on one of the most rapidly fatal cases recorded. The total illness did not exceed 24 hours. At autopsy, there was no evident source of haemorrhage, but intact bullae and scarring were revealed at both apices.

Hartzell (1942) directed attention to this entity by reporting four non-fatal cases, which he had observed in a decade. He also referred to the literature where, excluding tuberculosis and malignancy, 40 cases were on record, with a 30 per cent mortality-rate. He prophetically realised, as did earlier observers (Hopkins, 1937; Wilson, 1937), the need for judicious surgical intervention to identify and ligate the bleeding point. His forthright view was that this was a task for a "hardy surgeon." Helwig and Schmidt (1947) recorded the fatality within 24 hours of the onset of illness, of a 30 year old naval officer. Autopsy revealed well-vascularized adhesions as the undoubted source of haemorrhage. Indeed, histologically these adhesions were found to contain small interstitial haemorrhages. Neither bullae nor other possible source of escape of air from the lung could be identified. Apical scarring was detected.

McMyn, also in 1947, reported the death, despite orthodox medical treatment, of a 28 year old clerk. The patient expired just within one

week of the onset of an idiopathic tension pneumothorax, which was later complicated by haemorrhage. At necropsy, several small emphysematous bullae were seen at the apex and free border of the affected lung. One bulla was subjacent to a breach in the pleural apex where blood could be expressed. There was no mention of pleural adhesions or other possible source of haemorrhage. At this juncture, it is expedient to recapitulate and enlarge on the age and sex distribution of the earlier recorded cases. Hitherto only two female patients (Hopkins, 1937; Waring, 1945) had been reported. A further five (Harrell, 2 cases; Knight and Oelrich; Dorset and Terry; Hansen), however, were recorded in 1949, and two more (Kertesz; Deucher) in 1950. With the exception of Deucher's case, aged 51, the ages of the female patients conformed to the same fixed limits, i.e. 15 to 50 years, of all the previously reported male patients. This revised sex-incidence rapidly rescinded the erroneous earlier assertion that the occurrence of this condition in females was quite exceptional.

Hansen (1949) described three cases of unusual interest. The first, a ship's cook aged 30, was primarily admitted to a surgical ward with the diagnosis of a perforated peptic ulcer, and then transferred to a medical department with a revised diagnosis of coronary thrombosis. Two days after admission the correct diagnosis of idiopathic haemopneumothorax was made. The other two patients had underlying cystic disease of the lung as a departure from previously recorded cases. His second patient, a housewife aged 42 years, presented with a tension haemopneumothorax. Three weeks later she underwent an exploratory thoracotomy but the exposed pulmonary rent was too extensive for successful repair. She died several days later. The third patient, a welder aged 21, was successfully subjected

to thoracotomy for induction chemically of pleurodesis.

Arst and colleagues (1950) recorded the death, after a four day's illness, of a 22 year old postman. Post-mortem examination revealed a scarred lung-apex, where a torn adhesion was unequivocally defined as the source of haemorrhage. No mention was made of bullae.

At this juncture, it is convenient to review the results of numerous and varied investigations on the physical state and fate of blood in the pleural cavity. Cosgriff (1950) published his observations on the clotting capacity of extravasated intrapleural blood of a patient with traumatic haemothorax, while Harold (1951) reviewed the natural history of haemothorax.

Most observers, from the time of the earlier reports (Trousseau, 1870; Nelaton, 1880; Pagenstrecher, 1895; Sacquepee, 1902) agree that in cases of uninfected haemothorax, the intrapleural "blood" is mainly fluid in situ and that thoracentesis yields a dark sanguineous fluid which frequently fails to clot in vitro. Already in 1870, Trousseau taught that the extravasated intra-pleural blood clotted rapidly, but that it was defibrinated by the churning action of cardio-respiratory movements, before the red cells had time to sediment. This view has received subsequent support (Nelaton, 1880; Denny and Minot, 1915; Rietz, 1922; Melick and Spooner, 1945; Sellors, 1945), and has certainly not been disproved. On the contrary, it is popular explanation. Indeed, Sellors (1951) pointed out that, although the patient with a haemothorax has loss of movement of the chest-wall, this contrasts with the vigorous intrathoracic movements seen during thoracotomy.

Cosgriff (1950) demonstrated that such coagulation factors as

prothrombin, thrombin, and fibrinogen were absent from the haemorrhagic pleural fluid. He found no unusual anticoagulant activity, although this possibility had recently been entertained (Christian's textbook, 1947). Cosgriff concluded that intrapleural blood does clot as earlier experimental investigation, mainly involving the intrapleural introduction of blood, had suggested or shown (Trousseau, 1870; Nelaton, 1880; Sacquepee, 1902; Denny and Minot, 1913; Dorlencourt and Paycheres, 1918; Melick and Spooner, 1945). Indeed, within a few hours of its introduction, this clotting process occurs and leave a haemorrhagic fluid which is incoagulable and resembles pure blood. The fluid obtained by aspiration is therefore serum with a variable suspension of the formed elements of the blood. Earlier investigators (Zahn and Walker, 1914; Denny and Minot, 1915; Rietz, 1922; Filatoff, 1928; Melick and Spooner, 1945; Sellors, 1945) had found that this fluid has virtually no fibrinogen, at least in the first few days (Sellors, 1945). Zahn and Walter (1914) concluded from their observations that fibrinogen was altered or destroyed by the pleural surfaces but Denny and Minot (1915), introducing pure fibrinogen intrapleurally were unable to confirm this view.

It is therefore generally agreed that intrapleural clotting of blood occurs but there is no absolute agreement on whether this process is followed by defibrination or fibrinolysis or both. For instance, Quick (1942) summarised the evidence for, and admitted the possible existence of, a fibrinolytic enzyme in the pleural cavity. More recent study (Favre-Gilly, 1954) on patients with haemothorax suggests that the pleural fluid does not possess fibrinolytic properties, a view which is supported by the fact that fibrinolytic solutions are occasionally indicated and effective.

in the pleural débridement of fibrin deposits. Whilst, the exact natural fate of the fibrin is obscure, masses of fibrin-deposit have occasionally been observed radiographically or at operation or autopsy. Sellors (1945) showed that intrapleural blood provokes a large serous effusion, rich in fibrinogen, which may produce secondary clotting.

Chapman (1955) reviewed the reaction of serous cavities to blood. This response is manifest by pain, fever, leucocytosis and sometimes shock. He also discussed the occasional development of eosinophilia in both the haemorrhagic extravasation and the peripheral blood. The mechanism of this cellular reaction remains obscure.

Reverting to further clinical and autopsy observations, Cuningham (1950) and Irwin (1951) each reported a fatal case of idiopathic haemothorax. These will now be described to emphasize that the clinical course and autopsy-findings do not differ from those reported for patients with idiopathic haemopneumothorax. Cuningham's patient, a previously healthy male aged 17, was admitted to hospital 24 hours after the onset of severe thoracic pain. Despite therapeutic thoracentesis he expired within the next 24 hours. At necropsy, $3\frac{1}{2}$ litres of blood occupied the right pleural cavity, at the apex of which there was a roughened fibrous area with adherent clot. The corresponding lung-apex was puckered and beneath the adherent blood-clot there was a small, blood-infiltrated fibrous tag, marking the only probable site of haemorrhage. The opposite lung-apex was slightly scarred. These post-mortem observations, even without sub-pleural blebs, closely resemble those reported in fatal cases of idiopathic haemopneumothorax.

Irwin's patient, an army major aged 45, gave a history of previous

excellent health. During the afternoon of the day of admission, he was observed to be extremely pale, although he did not complain of pain. A provisional diagnosis of bleeding peptic ulcer was made. He expired 9 hours after the onset of illness, immediately on arrival in hospital. The following post-mortem findings are in all respects identical with those described in idiopathic haemopneumothorax. The right pleural cavity contained about $2\frac{1}{2}$ litres of blood. Fibrosis was detected at the right lung-apex, which was capped by four intact emphysematous blebs. From one of these blebs, however, a single fibrous band, 3 mm. in diameter, protruded for about 2 cm.. Both its free distal end and the adjacent parietal pleura had ragged surfaces, which were covered with blood. The torn fibrous band, on histological examination, contained four small arteries. No other lesion or bleeding point was discovered. There were, however, many pleural adhesions on the opposite side of the chest.

Finally, from 1951 onwards, a remarkable transformation in the history of idiopathic haemopneumothorax was manifest. During this new era only two deaths (Grundi, 1953; Author's case 3) have been reported in one hundred cases. This improved mortality-rate coincided with the introduction of three therapeutic measures, which will only receive preliminary mention at this stage.

Firstly the operation, emergency thoracotomy, has been brilliantly successful, (Myers et al., 1951; Ross, 1952; Beatty and Frelick, 1952; Holloway et al., 1952; Borrie, 1953; Ross et al., 1953; Williams et al., 1954; Fry et al., 1955; Clyne and Hutter, 1955). Despite the fact that several of these patients were in extremis pre-operatively, all of the 19 recorded patients (Table 11) who underwent this operation recovered.

Among the 12 cases with definite mention, all except 3 (Holloway et al., 1952; Borrie, Case I, 1953; Clyne and Hutter, Case I, 1955) were discharged from hospital within a fortnight after the operation.

Secondly, the adoption of the intrapleural instillation of fibrinolytic enzymes (usually the combined solution of streptodornase and streptokinase) to liquefy blood-clots and to debride fibrinous pleural thickening, has met with a measure of success (Read and Berry, 1950; Carr and Robbins, 1951; Jones and Bigham, 1953; Calvert and Smith, 1955).

Thirdly, the operation, "late" thoracotomy with pulmonary decortication has frequently been valuable in improving the pulmonary function of patients with clotted haemothorax, organising haemothorax, fibrothorax, and empyema consequent on haemothorax (Harrington and Frelick, 1947; Leahy, 1947; Elrod and Murphy, 1948; Nalls and Matthews, 1949; Kelly, 1949; Mario et al., 1949; Wright et al., 1949; Williams, 1950; Arst et al., 1950; Deiss et al., 1950; Seley and Neuhof, 1951; Hyde and Hyde, 1951; Carroll et al., 1951; Beatty and Frelick, 1952; Ross, 1952; Fusia and Cook, 1952; Kastl, 1952; Kiekens, 1952; Eidinger and Rubin, 1952; Garcia et al., 1952; Biancanala, 1953; Mezzera and Aquiar, 1954; McGown, 1954; Shefts et al., 1954; Fry et al., 1955; Agnew, 1955; Weiner, 1955).

TABLE I. IDIOPATHIC HAEMOPNEUMOTHORAX.

¥ D = Died
R = Recovery

YEAR		AUTHORS	AGE SEX	SIDE OF CHEST	NATIONALITY and OCCUPATION	PREVIOUS LUNG INFECTION	FAMILY T.B.	ACTION AT ONSET	LATENT PERIOD	TOTAL PERIOD OF ILL.	OUTCOME ¥	HB. %
1900	1	Pitt	18 M.	R.	Brit. -	Nil	No	Walking	16 hrs.	24 hrs.	D.	-
	2	Rolleston	21 M.	R.	Brit. -	Nil	No	-	25 hrs.	7 days	D.	-
	3	Boland	34 M.	R.	Amer. Grocer	Nil	No	No Exercise	Few hrs.	11 wks.	R.	-
1910	4	Ness and Allan	31 M.	L.	Brit. Clerk	Nil	No	Arising from bed	7 days	7 wks.	R.	-
1913	5	Bushby	17 M.	L.	Brit. Clerk	Nil	No	Running	-	3 mths.	R.	-
1917	6	Williamson	- M.	L.	Amer. Broker	Nil	No	Bending	-	10 wks.	R.	-
1922	7	Fischer	22 M.	R.	Germ. Cine-operator	Nil	No	No Exercise	7 days	7 days	D.	-
1923	8	Kiaer	30 M.	L.	Dane Rifle-man	Nil	-	-	2 days	3 days	D.	-
1926	9	Bouchut & Beaupere	21 M.	R.	French -	Nil	No	-	-	7 wks.	R.	-
1928	10	Hurxthal	29 M.	R.	Amer. -	Colds	No	Moving car seat	Few hrs.	10 days	R.	-
	11	Doria	26 M.	R.	Ital. Clerk	Nil	No	Walking	18hrs.	3 mths.	R.	-
1930	12	Terry	34 M.	L.	Amer. -	Dry pleurisy	No	Walking	90hrs.	16 days	R.	-
1931	13	Housden & Piggot	44 M.	L.	Brit. Plumber	Bronchitis	No	Cough	60hrs.	24 days	D.	-
	14	Milhorat	21 M.	L.	Cuban Clerk	Colds	No	Riding in subway	24hrs.	3½ mths.	R.	-
	15	Palmer and Taft	26 M.	L.	Amer. Clerk	Nil	No	No Exercise	12hrs.	5 wks.	R.	-
	16		25 M.	R.	Amer. Gas-fitter	Nil	No	Vomiting	-	-	R.	-

IDIOPATHIC HAEMOPNEUMOTHORAX.

1932	17	Kjaergaard	20 M.	L.	Dane	Student	Nil	No	No Exercise	12 hrs.	2 mths.	R.	53
	18		25 M.	L.	Dane	Clerk	Nil	No	No Exercise	24 hrs.	4 mths.	R.	-
1933	19	Woll	43 M.	R.	Germ.	Farmer	-	-	Walking Uphill	24 hrs.	2 mths.	R.	-
1934	20	Patino Mayer and Pataro	32 M.	R.	Arab	Labourer	Nil	-	Walking	12 hrs.	3 mths.	R.	-
1934	21	Bellon	21 M.	R.	French	Soldier	Nil	No	Climbing Stairs	15 hrs.	3½mths.	R.	60
	22	Leggett et al.	- M.	R.	Amer.	-	Nil	-	-	-	-	R.	-
	23		- M.	-	Amer.	-	Nil	-	-	-	-	R.	-
1935	24	Holden	32 M.	L.	Amer.	Mechanic	Pneumonia	-	Awakened 4 a.m.	36 hrs.	1 mth.	R.	80
* see next page	25		25 M.	R.	Amer.	Labourer	-	-	Carrying a sack	24 hrs.	1 mth.	R.	77
1936	26	Castex & Mazzei	35 M.	R.	Argent.	Valet	Nil	-	Cleaning	15days	2½mths.	R.	-
	27		21 M.	L.	Argent.	Fruit Vendor	Nil	-	Awoke with pain	6 hrs.	2½mths.	R.	45
	28	Wilson	31 M.	L.	Amer.	Baker	Nil	No	Driving van	4days	11 wks.	R.	77
	29	Jones & Gilbert	23 M.	R.	Amer.	Student	Pneumonia & pleurisy	No	Riding in Subway	7 hrs.	5 wks.	D.	-
	30	Jacob	23 M.	L.	French	Soldier	Cough Sputum	-	Lifting a sack	24hrs.	1 mth.	R.	-
	31	Vio	20 M.	R.	Ital.	Student	-	-	-	3days	3 wks.	R.	75
	32	Centeno et al.	27 M.	R.	Argent.	-	Nil	No	Working with veh.	13days	7 wks.	R.	74
	33	Poli	35 M.	L.	Ital.	shopkeeper	Nil	No	No Exercise	4days	2½mths.	R.	91
	34	Munos-Monatorio	26 M.	L.	Urug.	Soldier	Nil	No	-	Few Hrs.	2 mths.	R.	

INTERPOLATION

IDIOPATHIC HAEMOPNEUMOTHORAX.

1935	25a	Frey	30 M.	R.	Amer.	Upholsterer	Pneumonia	-	In bed	36 hrs.	6 wks.	R.	82
	25b	Rossel	20 M.	L.	French	Soldier	Pneumonia	No	No Exercise	Few hrs.	6 days	D.	63
	25c	Tait & Wakely	32 M.	R.	Brit.	Clerk	-	-	Lifting a bed	4 days	8 days	D.	-
	25d	Aguilar and Ferradas	37 M.	L.	Argent.	-	Nil	-	-	9 days	5 wks.	R.	-
	25e		29 M.	L.	Argent.	-	Bronchitis	-	-	5 days	8 wks.	R.	-
	25f	Catuogno	27 M.	R.	Argent.	-	Nil	No	Walking into room	2 days	$2\frac{1}{2}$mths.	R.	-
	25g		27 M.	R.	Argent.	Chauffeur	-	-	Working with car	-	5 wks.	R.	-
	25h	Staffieri	24 M.	R.	Argent.	Businessman	Nil	No	-	12 hrs.	1 mth.	R.	78

¤ This group (25a - 25h) of patients was inadvertently omitted during the preparation of the typescript.

IDIOPATHIC HAEMOPNEUMOTHORAX.

1936	35	Munos-Monatorio	22 M.	R.	Urug. Student	Nil	No	Climbing Stairs	4 days	2 mths.	R.	-
	36	Korol	35 M.	R.	Amer. -	-	-	-	11days	Over 2 mths.	R.	-
	37		33 M	L.	Amer. -	Nil	-	Bending	Few hours	4 mths.	R.	60
	38	Vesin and Bobek	20 M.	R.	Czech. Student	-	-	Cycling	5 days	1 mth.	R.	-
1937	39	Hopkins	30 M.	L.	Negro -	-	-	No Exercise	Few hours	7 wks.	R.	80
	40		30 F.	L.	Amer. Clerk	-	-	No Exercise	5 days	3 wks.	R.	-
	41		30 M.	L.	Amer. Cafe Manager	-	-	Shaving	30hrs.	2 wks.	R.	-
	42	Hees	22 M.	L.	Dutch Student	-	-	Just played Clarinet	1 day	3 wks.	R.	62
	43	Troisier	34 M.	L.	French Plumber	Nil	No	Walking	5days	6 wks.	R.	-
	44	Wilson	24 M.	L.	Amer. Student	Nil	Nil	Bending	24hrs.	2 wks.	R.	80
	45	Rist	17 M.	R.	Chinese Student	-	-	Climbing tower	8days	2 wks.	R.	-
	46	Landron & Irwin	22 M.	L.	Peurto Rican Labourer	Nil	No	Awoke with pain	1 day	6 wks.	R.	65
	47	Catuogno et al.	28 M.	L.	Argent. Businessman	Nil	No	Getting off train	1 day	5 wks.	R.	-
	48	Cardenas et al.	21 M.	L.	Havana Clerk	Nil	No	Arising from bed	2days	6 wks.	R.	60
1938	49	Salaris	25 M.	L.	Ital. Clerk	Nil	No	Arising from bed	36hrs.	1 mth.	R.	28
	50		27 M.	L.	Ital. Clerk	Nil	No	Walking	Few Hrs.	1 mth.	R.	86
	51	Beaumont	25 M.	R.	Brit. -	Nil	No	Divesting coat	2days	Not stated	R.	55
	52		28 M.	R.	Brit. -	-	-	-	3 wks.	3 wks.	D.	-

IDIOPATHIC HAEMOPNEUMOTHORAX.

1938	53	Perry	32 M.	L.	Brit. Tailor	-	-	Awoke with pain	Few Hrs.	6 wks.	R.	70
	54	Louria	32 M.	R.	Amer. Plumber	L.Pleurisy	-	-	1 wk.	2 mths.	R.	-
	55		22 M.	L.	Ital. Barber	Chronic cough	-	Awoke with pain	16hrs.	4 days	D.	40
	56		31 M.	R.	Amer. Chauffer	Nil	No	-	24hrs.	7 wks.	R.	-
	57		28 M.	R.	Negro Taxi-driver	Nil	-	-	Few hrs.	4 mths.	R.	63
	58		45 M.	R.	Amer. Labourer	Nil	No	-	2 days	4½mths.	R.	55
	59	Egues	38 M.	L.	Argent. -	Nil	Brother had T.B.	Gymnastics	1 day	7 mths.	R.	-
	60		31 M.	L.	Argent. Clerk	Nil	2 brothers had pleurisy	-	1 day	2 wks.	R.	-
1939	61	Sloer	29 M.	L.	Argent. Clerk	Nil	Father suspect T.B.	No Exercise	1 day	5 wks.	R.	63
1940	62	Davidson & Simpson	26 M.	R.	Brit. Soldier	-	-	Following parade	10hrs.	37hrs.	D.	
	63	Lorge	34 M.	L.	Amer. Messenger	Nil	-	Working	Few hrs.	About 12hrs.	D.	-
	64	Repetti	20 M.	R.	Argent. Student	-	-	-	Few hrs.	6.wks.	R.	-
	65	Rist and Worms	36 M.	L.	French Economist	Nil	-	Conversation	2 days	4 mths.	R.	-
1941	66	Arroyo	37 M.	L.	Argent. Labourer	Nil	No	-	2 days	3 mths.	R.	-
	67	Roderiquez Pastor and Arruza	24 M.	R.	Peurto Rican -	Nil	No	-	-	2 mths.	R.	60
	68		19 M.	L.	Peurto Rican Student	Nil	No	Rising from seat	Few hrs.	Several weeks	R.	48
	69	Jennings	41 M.	L.	Amer. Bank-teller	Cough 1 yr.	No	Reclining in aeroplane	1 day	5 mths.	R.	88

IDIOPATHIC HAEMOPNEUMOTHORAX.

1942	70	Hartzell	26 M.	L.	Amer.	Machinist	Nil	-	Arising from bed	1 wk.	26 dys.	R.	55
	71		17 M.	L.	Amer.	-	Nil	-	-	3days	26 dys.	R.	
	72		21 M.	L.	Amer.	Truck Driver	Cough & haemop. 2 wks.	-	Arising from bed before	12 hrs.	18 dys.	R.	80
	73		22 M.	R.	Amer.	-	Nil	-	-	-	-	R.	-
	74	Lea	26 M.	L.	Canad.	Army clerk	Nil	No	Sitting in car	12 hrs.	6 wks.	R.	54
	75	Tannenbaum	24 M.	L.	Amer.	Clerk	Nil	No	Returned from walk	1 day	1 mth.	R.	60
1943	76	Staffieri et al.	45 M.	L.	Argent.	Ironmonger	Nil	-	Stretching	A few days	2½mths.	R.	-
	77		28 M.	R.	Argent.	Electrician	Nil	-	Climbing Stairs	1 day	6 wks.	R.	-
	78	Raimondi & Boffi	38 M.	L.	Argent.	Clerk	Nil	No	Standing up	Few hrs.	5 wks.	R.	67
	79	Leston & Lamberti	26 M.	L.	Argent.	Businessman	Nil	-	Returning from work	Few hrs.	3 mths.	R.	50
1944	80	Goldman & Roth	28 M.	R.	Amer.	Engineer	Nil	-	-	1 day	3 wks.	R.	78
1945	81	Waring	"Young" M.	R.	Amer.	-	-	-	-	-	5 mths.	R.	
	82		"Young" F.	R.	Amer.	-	-	-	-	2days	9 wks.	R.	-
	83	Franklin	23 M.	R.	Amer.	Soldier	Nil	Yes	-	1 wk.	1 mth.	R.	-
	84	Schneider and Reissman	33 M.	R.	Amer.	Soldier	Nil	-	Eating breakfast	Few hrs.	6 mths.	R.	-
	85	Payn and Lief	19 M.	L.	Amer.	Soldier	-	-	-	Few hrs.	2 mths.	R.	92
1946	86	Bernstein et al.	31 M.	L.	Amer.	-	-	-	Getting off bus	2 days	2 wks.	R.	42
	87	Van Der Meer	26 M.	L.	Amer.	Soldier	Nil	No	Filling in Trench	1 day	25 dys.	R.	-

IDIOPATHIC HAEMOPNEUMOTHORAX.

1947	88	Harrington and Frelick	27 M.	R.	Amer. Clerk	-	-	Rising from bed	1 day	9 wks.	R.	-
	89	Helwig and Schmidt	30 M.	L.	Amer. Naval Off.	-	-	Carrying bag	5days	6days	D.	-
	90	McMyn	28 M.	R.	Brit. Clerk	-	-	-	36hrs.	6days	D.	-
	91		19 M.	R.	Brit. Airman	-	-	-	Few hrs.	2mths.	R.	-
	92	Stockler	44 M.	-	Braz. -	-	-	-	1 mth.	2mths.	R.	-
	93		39 M.	L.	Braz. -	-	-	-	1 day	5 wks.	R.	-
	94		30 M.	R.	Braz. -	-	-	-	1 day	3 wks.	R.	-
	95		19 M.	L.	Brazil. -	-	-	-	-	-	R.	-
	96	Groen & Godfried	38 M.	L.	Dutch Waiter	Nil	No	-	5days	2mths.	R.	70
1948	97	Thomas & Beerens	35 M.	R.	Belg. Wharf-man	-	-	Weight-lifting	1 day	34hrs.	D.	-
	98	Orsi	28 M.	R.	Argent. Paint-er	-	-	-	25days		R.	-
	99		36 M.	L.	Argent. -	-	-	-	2 days	4½mths.	R.	60
	100	Elrod & Murphy	31 M.	L.	Amer. -	-	-	Driving car	10days	7½wks.	R.	-
	101	Dardet	21 M.	L.	Spanish -	Nil	-	-	3 hrs.	2 mths.	R.	-
	102	Nightingale	27 M.	L.	Amer. Chief torpedo-man	Nil	Yes	Reaching over head	1 day	11 wks.	R.	-
1949	103	Wright et al Case 3	21 M.	R.	Amer. -	Nil	-	Sawing wood	2 days	2 mths.	R.	-
	104	Myers et al.	22 M.	R.	Amer. -	-	-	After base-ball	2 days	2 wks.	R.	70
	105	Knight & Oelrich	16 F.	L.	Amer. Textile worker	-	-	Working in mill	12 hrs.	2 wks.	R.	48

IDIOPATHIC HAEMOPNEUMOTHORAX.

1949 106	Nalls & Matthews	41 M.	L.	Amer. Bus Driver	-	-	Driving	24hrs.	3 wks.	R.	70
107		19 M.	L.	Amer. -	-	-	Awoke with pain	24 hrs.	4½mths.	R.	77
108	Dorset & Terry	27 F.	L.	Amer. Clerk	Nil	No	Sleeping	2 days	1 mth.	R.	-
109	Kelly	42 M.	R.	Amer. Book-keeper	Nil	No	Eating Dinner	1 day	5 wks.	R.	50
110	Hansen	30 M.	R.	Dane Ship's cook	Nil	-	Exertion	Few Hrs.	2 wks.	R.	58
111		42 F.	L.	Dane -	Nil	-	No Exercise	3days	3 wks.	D.	-
112		21 M.	R.	Dane Welder	Nil	-	Walking home	Few hrs.	5wks.	R.	
113	Solovay	32 M.	R.	Amer. Clerk	-	Yes	Standing still	6 days	7 wks.	R.	40
114	Walton (Case 2)	26 M.	L.	Brit. Joiner	Nil	No	-	3 wks.	-	R.	90
115	Harrell	42 F.	L.	Amer. Housewife	Nil	-	Shovelling snow	1 day	8 dys.	R.	81
116		35 M.	R.	Amer. Upholst-erer	Nil	-	Shaving	-	4½mths.	R.	-
117		29 F.	R.	Amer. Housewife	-	-	Washing dishes	6 hrs.	-	R.	-
118	Nario	25 M.	L.	Uruguay Painter	Nil	-	Turning	16 dys.	6 wks.	R.	47
1950 119	Arst et al.	22 M.	R.	Amer. Post-man	Nil	No	Sitting	Few hrs.	4 days	D.	58
120		21 M.	L.	Amer. Student	Nil	-	Sitting	2 days	5 wks.	R.	57
121	Cuningham	35 M.	L.	N.Z. -	-	-	-	24hrs.	7 wks.	R.	73
122	Deiss et al.	20 M.	R.	Iranian Stu-dent	Nil	No	Reaching	1 day	6 wks.	R.	-
123		23 M.	L.	Amer. Farmer	-	-	Awoke with pain	Few hrs.	2mths.	R.	-

IDIOPATHIC HAEMOPNEUMOTHORAX.

1950 124	Cosgriff	42 M.	R.	Amer. -	Nil	No	-	24 hrs.	2 wks.	R.	87
125	Kertèsz	17 F.	R.	Amer. School	-	-	Boarding bus	1 wk.	5 wks.	R.	75
126	Read and Berry	21 M.	L.	Negro -	-	-	-	2 hrs.	5 wks.	R.	-
127	Williams	14 M.	L.	Amer. School	-	-	Sledging	Few hrs.	5 wks.	R.	-
128	Deucher	51 F.	R.	Swiss Housewife	Nil	No	Beating a carpet	24hrs.	1 mth.	R.	-
129		27 M.	L.	Swiss Railway Official	Nil	-	On waking	12hrs.	7 wks.	R.	-
130	Leston and Pilheu	19 M.	L.	Argent. Labourer	Nil	No	-	Few hrs.	5 mths.	R.	35
131		23 M.	R.	Argent. Bookkeeper	-	Yes	-	8 dys.	6 wks.	R.	70
1951 132	Moser	17 M.	R.	Amer. Clerk	Pneumonia	-	Sitting	36hrs.	3 wks.	R.	-
133		35 M.	L.	Amer. -	Broncho-pneumonia	-	Eating dinner	-	2 wks.	R.	-
134		22 M.	L.	Amer. Metal Polisher	-	-	Walking home	20hrs.	10 dys.	R.	-
135		18 M.	L.	Amer. -	Periodic pneumonia	-	-	6 hrs.	2 wks.	R.	-
136	Beatty & Frelick	33 M.	L.	Amer. -	-	-	Turning at desk	2 hrs.	17 dys.	R.	66
137	Carroll et al.	38 M.	R.	Amer. -	-	-	Sitting	-	2 mths.	R.	-
138		24 M.	L.	Amer. Student	Nil	-	Walking	-	2 mths.	R.	-
139	Grimaldi	34 M.	L.	Ital. Goldsmith	Nil	No	-	-	5 mths.	R.	-
140	Myers et al.	22 M.	R.	Amer. -	-	-	After baseball	2 dys.	2 wks.	R.	70
141	Seley and Neuhof	23 M.	R.	Amer. Clerk	-	-	-	5 wks.	7 wks.	R.	78

IDIOPATHIC HAEMOPNEUMOTHORAX.

1951 142	Lewis	44 M.	L.	Brit.	Photo-grapher	-	-	Walking upstairs	12 hrs.	6 wks.	R.	-
143	Joselevich	43 M.	L.	Argent.	Elec-trician	Nil	-	No exercise	3 dys.	1 mth.	R.	-
144	Borroni	37 M.	L.	Ital.	Chauf-feur	Nil	-	-	Few hrs.	2 mths.	R.	73
145	Hyde and Hyde	44 M.	L.	Amer.	-	-	-	In bed	-	11 wks.	R.	100
146		31 M.	L.	Amer.	-	-	-	-	-	7 wks.	R.	99
147		36 M.	L.	Amer.	-	-	-	At rest	-	6 wks.	R.	100
148		27 M.	R.	Amer.	-	-	No	-	3 dys.	1 mth.	R.	79
149		38 M.	R.	Amer.	-	-	Yes	-	6 wks.	11 wks.	R.	-
150		30 M.	L.	Amer.	-	-	-	-	Few hrs.	5 wks.	R.	100
151		21 M.	R.	Amer.	elec-trician	-	-	-	10dys.	3 wks.	R.	-
152		23 M.	R.	Amer.	-	Periodic pneumonia	-	-	-	-	R.	65
153		20 M.	L.	Japanese	-	-	-	-	-	9 wks.	R.	-
154		20 M.	L.	Amer.	-	-	-	-	-	1 mth.	R.	-
155		27 M.	R.	Amer.	-	-	-	Exertion	2 dys.	2 wks.	R.	-
156		21 M.	R.	Amer.	-	-	Yes	No effort	3 dys.	1 mth.	R.	74
1952 157	Beatty and Frelick	26 M.	L.	Amer.	-	-	-	-	-	-	R.	-
158		21 F.	L.	Amer.	-	Nil	No	-	1 day	2 wks.	R.	70
159		33 M.	R.	Amer.	-	-	-	-	1 day	2 wks.	R.	-

IDIOPATHIC HAEMOPNEUMOTHORAX.

1952 160	Beatty and Frelick	30 M.	L.	Amer. -	-	-	In bed	1 day	1 wk.	R.	73
161	Fusia and Cook	23 M.	L.	Amer. Insurance Agent	Nil	-	-	Few hrs.	1 mth.	R.	64
162		25 M.	L.	Amer. Carpenter	Nil	-	-	9 dys.	6 wks.	R.	63
163	Kastl	27 M.	L.	Amer. -	Nil	-	Awoke with pain	36hrs.	2 wks.	R.	68
164		32 M.	L.	Amer. -	Nil	-	Carpentry at home	3 dys.	5 mths.	R.	68
165	Holloway	19 M.	R.	Amer. Sailor	Nil	No	-	8 hrs.	1 wk.	R.	55
166	Kiekens	24 M.	L.	Belg. -	Nil	-	Rising from bed	Few hrs.	1 mth.	R.	61
167	Eidinger and Rubin	28 M.	L.	Canad. -	Brenchitis	-	-	1 day	2 wks.	R.	-
168		42 M.	L.	Canad. -	Pleurisy	-	-	1 day	-	R.	-
169		34 M.	R.	Canad. -	Pneumonia	-	Standing on steps	1 day	1 mth.	R.	-
170	Ross	34 M.	R.	Brit. Machinist	-	-	-	24hrs.	1 wk.	R.	-
171		26 M.	L.	Brit. Labourer	-	-	-	3 wks.	3 mths.	R.	-
172		29 M.	L.	Brit. Engineer	-	-	Sitting at desk	3 wks.	9 wks.	R.	-
173		22 M.	L.	Brit. Toolmaker	-	-	-	1 day	1 mth.	R.	-
174		25 M.	L.	Norweg. Sailor	-	-	Pulling a rope	Few hrs.	3 wks.	R.	-
175		26 M.	R.	Brit. Teacher	-	-	-	3 mths.	-	R.	-
1953 176	Freund and Hicks	24 F.	L.	Amer. Salesgirl	-	-	-	1 wk.	6 wks.	R.	50
177	Desjardins et al.	26 M.	R.	Amer. Innkeeper	Nil	-	Awoke with pain	2 dys.	9 dys.	R.	80

IDIOPATHIC HAEMOPNEUMOTHORAX.

1953 178	Desjardins et al.	22 M.	R.	Amer. Student	Nil	-	-	2 dys.	3 wks.	R.	40
179	Huss	20 M.	L.	Amer. Maid	Nil	-	-	36hrs.	2 wks.	R.	-
180	Biancanala	40 M.	R.	Ital. -	Nil	No	-	-	-	R.	-
181	Ross et al.	27 M.	L.	Amer. Newspressman	Nil	No	Sneezing	1 day	12 dys.	R.	-
182	Garcia et al	29 M.	L.	Chinese -	-	-	In Vehicle	Few hrs.	5 mths.	R.	-
183		28 M.	L.	Phillipine Clerk	-	-	No exertion In office	9 days	6 mths.	R.	-
184	Ingrassia	18 M.	R.	Sicil. student	-	-	No exertion	Few hrs.	10 days	R.	-
185	Grundi	21 M.	R.	Canad. -	Nil	No	No exertion	3 days	7 wks.	D.	68
186	Borrie	21 M.	R.	N.Z. Sailor	-	-	Duties	Few hrs.	16 days	R.	-
187		26 M.	R.	N.Z. Stevedore	-	-	In Cinema	1 day	15 days	R.	-
188		18 M.	R.	N.Z. School	-	-	Rowing	2 wks.	-	R.	-
189	Stensrud	30 M.	L.	Norway -	Pneumonia & bronchitis	-	-	-	3 mths.	R.	101
190	Jones and Bigham	22 M.	L.	Amer. Airman	Nil	-	Eating	3 days	6 wks.	R.	70
1954 191	Moraes	22 M.	L.	Braz. Businessman	-	-	-	10 days	3 wks.	R.	-
192	Monti	29 M.	L.	Ital. -	Nil	No	-	4 hrs.	9 wks.	R.	-
193	Towson	27 F.	L.	Canad. Housewife	Nil	-	-	2 wks.	7 wks.	R.	58
194	McGown	23 M.	L.	Brit. Shop Assist.	Nil	No	-	1 day	6 wks.	R.	29
195	Williams et al.	33 M.	R.	Amer. Truck-driver	-	-	-	3 days	2 wks.	R.	-

IDIOPATHIC HAEMOPNEUMOTHORAX.

1954 196	Meyer et al.	52 M.	L.	French	Antique-dealer	Nil	No	At breakfast	1 day	1 mth.	R.	-
197	Myers	24 M.	R.	Amer.	-	-	-	-	-	1 mth.	R.	-
198		34 M.	R.	Amer.	-	-	-	-	-	-	R.	-
199		23 M.	R.	Amer.	-	-	-	-	-	-	R.	-
200		39 M.	R.	Amer.	-	-	-	-	-	1 mth.	R.	-
201		31 M.	R.	Amer.	-	-	-	-	-	-	R.	-
202		20 M.	L.	Amer.	-	-	-	-	-	2 wks.	R.	-
203		34 M.	R.	Amer.	-	-	-	-	-	18 days	R.	-
204		31 M.	R.	Amer.	-	-	-	-	-	-	R.	-
205	Mezzera & Aguiar	30 M.	L.	Urug.	Student	Nil	No	Duty	Few hrs.	4 mths.	R.	-
206	Meo & Tallone	23 M.	L.	Ital.	Gold-smith	Nil	No	Sitting in theatre	3 hrs.	6 wks.	R.	68
1955 207	Fry et al.	27 M.	Not Stated	Amer.	-	-	-	-	-	-	R.	-
208		20 M.	"	Amer.	-	-	-	-	-	-	R.	-
209		30 M.	"	Amer.	-	-	-	-	-	-	R.	-
210		23 M.	"	Amer.	-	-	-	-	-	-	R.	-
211		19 M.	"	Amer.	-	-	-	-	-	-	R.	-
212		19 M.	R.	Amer.	-	-	-	-	-	-	R.	-
213		21 F.	Not Stated	Amer.	-	-	-	-	-	-	R.	-

IDIOPATHIC HAEMOPNEUMOTHORAX.

1955	214	Fry et al.	27 M.	Not Stated	Amer.	-	-	-	-	-	-	R.	-
	215		51 M.	"	Amer.	-	-	-	-	-	-	R.	-
	216		27 M.	"	Amer.	-	-	-	-	-	-	R.	-
	217		31 M.	"	Amer.	-	-	-	-	-	-	R.	-
	218		33 M.	"	Negro	-	-	-	-	-	-	R.	-
	219		19 M.	"	Amer.	-	-	-	-	-	-	R.	-
	220	Author's Cases	26 M.	L.	Brit.	Clerk	Nil	No	Walking home	24 hrs.	18 days	R.	56
	221	Calvert & Smith	29 M.	L.	Brit.	Foundry worker	Nil	Yes	Working	4 days	9 wks.	R.	88
	222		40 F.	L.	Brit.	House-wife	Nil	Yes	In bed	Few hours	1 day	D.	73
	223		27 M.	L.	Brit.	Mech-anic	Whoop.Cough Pleurisy	No	Working	Few hours	2 wks.	R.	72
	224		36 M.	L.	Brit.	Gas-fitter	Nil	No	Working	Few hours	2 wks.	R.	60
	225		33 M.	L.	Brit.	Driver's mate	Whoop.Cough Bronchitis	No	Lifting	2 days	3 wks	R.	52
	226		22 M.	L.	Brit.	Com-positor	Nil	No	No exercise	1 day	1 mth.	R.	84
	227		23 M.	L.	Brit.	Bus-driver	Nil	No	Driving	1 day	3 wks.	R.	96
	228	Clyne & Hutter	28 M.	L.	Brit.	Air-man	-	-	-	8 hrs.	1 mth.	R.	-
	229		32 M.	L.	Brit.	Air-man	-	-	-	6 hrs.	3 wks.	R.	-
	230		19 M.	L.	Brit.	Sol-dier	-	-	-	6 hrs.	3 wks.	R.	58

IDIOPATHIC HAEMOPNEUMOTHORAX.

1955 231	Agnew	41 M.	R.	Brit.	Shop-manager	Nil	-	Tying Shoe lace	5 hrs.	5 wks.	R.	80
232	Weiner	35 M.	L.	Amer.	-	Nil	No	-	-	1 mth.	R.	-

N.B: The case numbering ends here with 232 instead of 240 as eight cases relating to 1935 were inadvertently omitted during the preparation of the typescript.

Postscript: Sinclair (1955) described 5 cases of idiopathic haemopneumothorax, 3 of which Borrie (1953) had previously reported in his article on emergency thoracotomy. The 2 remaining patients of Sinclair are not included here, but it is interest that one of these was a woman aged 32 who developed a late elevated intrathoracic tension. Both patients recovered.

Presentation of Author's Cases.

The eight patients, whose case-reports follow, collectively provide a remarkably clear picture of the entire subject of idiopathic haemopneumothorax. This series was collected during a four-year's study of this entity. During that period, no acceptable case of idiopathic haemothorax was observed.

By strange coincidence, all the patients had left-sided haemopneumothoraces. Except for Case 3, all were male subjects. The age-range was 22 to 40. One fatality (Case 3) occurred. Two patients, Cases 5 and 6, underwent operation, i.e. emergency thoracotomy for haemostasis and pulmonary decortication for early fibrothorax respectively.

CASE I.

J.H., a previously healthy clerk aged 26, was admitted on May 28, 1952, with moderate breathlessness and a sharp inspiratory pain, radiating from the left lower chest behind the left shoulder. This pain had appeared abruptly on the previous evening while he was walking home from work. Despite distress, he reached home unaided and immediately went to bed. He experienced discomfort on attempting to lie on either side. Next morning he had several syncopal attacks, during one of which he was unconscious for five minutes. As movement increased his pain, he remained stock-still. His doctor diagnosed a left sided pneumothorax, but considered that there was associated acute intercostal fibrositis as the intercostal muscles splinted the left hemithorax. The past history disclosed no respiratory disease. Indeed, a routine chest radiograph carried out three months previously was normal. There was no family history of tuberculosis.

Examination revealed a pale, anxious patient of fairly good physique

with minimal venous congestion. The respirations were shallow and their rate was 30 per minute. The salient signs were confined to the respiratory and cardiovascular systems. The trachea was displaced to the right. The remaining respiratory signs were on the left side, where there was fullness, greatly restricted movement, and a very dull percussion note in the base, mid-zone and axilla. In these sites tactile vocal fremitus, breath-sounds, and vocal resonance were absent, but aegophony was heard both in the scapular region and anteriorly. The vascular signs included tachycardia (120/minute), an easily compressible but regular pulse, and hypotension (115/70 mm. Hg.). The apex beat could not be located, while the cardiac sounds were pure but faint.

Immediate investigation showed a haemoglobin level of 56% with a normal total and differential white cell count, and a chest radiograph (Fig. I) revealing a left-sided pneumothorax with effusion and ipsilateral apical pleural thickening. The electrocardiogram was normal apart from ST segment depression in the limb leads, suggesting a relative coronary insufficiency. An hourly pulse chart was begun and blood was cross-matched for emergency. Morphine sulphate ($\frac{1}{4}$ gr.) was injected before thoracentesis, which yielded $5\frac{1}{4}$ pints of sanguineous fluid with a haemoglobin value of 62%. Almost complete re-expansion of the lung promptly followed. The next day the patient was very comfortable with a pulse rate of 90 per minute, blood pressure of 115/75 mm. Hg. and the trachea central. Progress was now uneventful and the chest radiograph on June 16, 1952 (Fig. 2) was almost normal. Five weeks after admission he was discharged home feeling fit, having gained 8 lb. in weight, and with a haemoglobin value of 84% following treatment with ferrous sulphate

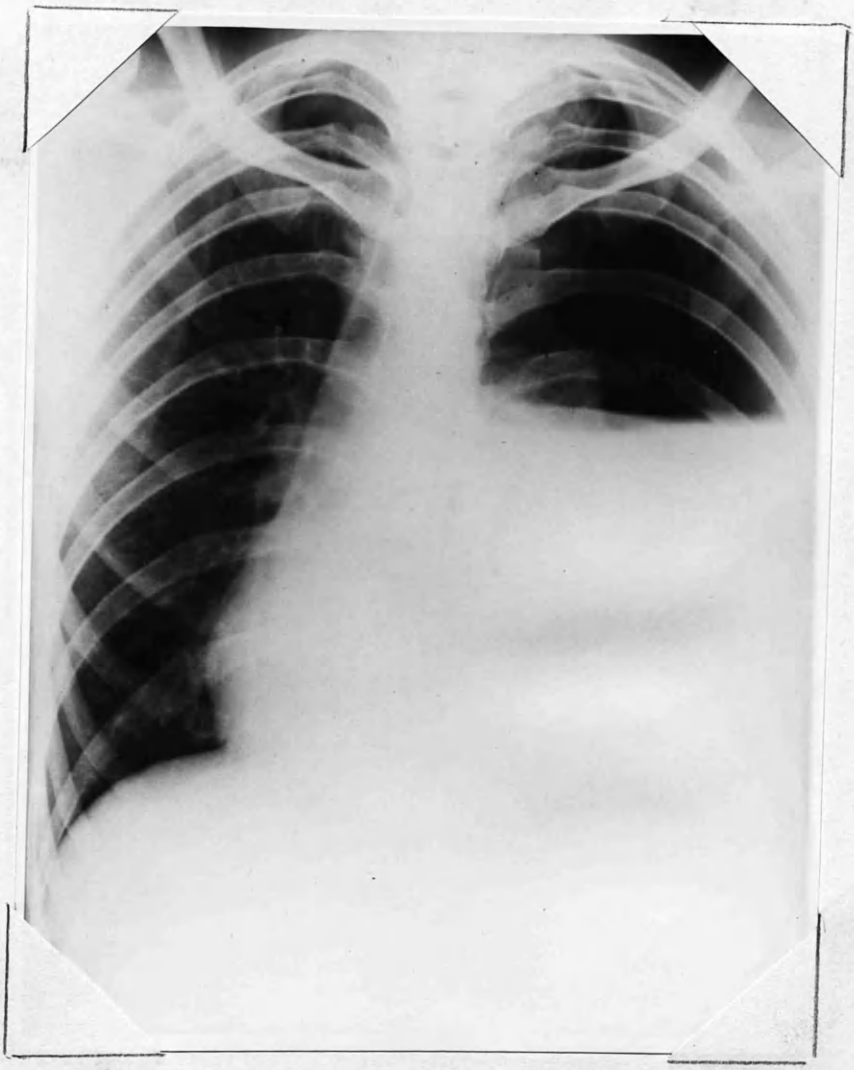

FIG. I. Case I. Radiograph on May 28, 1952, on admission to hospital. Large left-sided pleural effusion before thoracentesis.

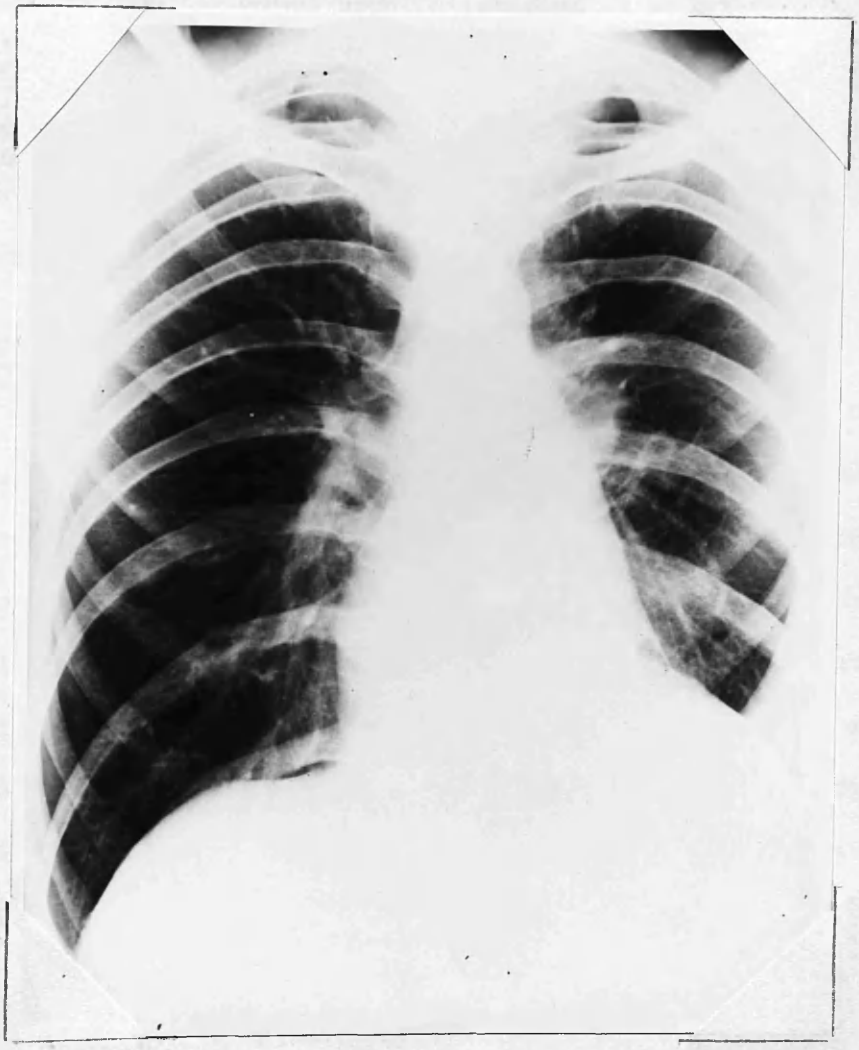

FIG. 2. Case I. Radiograph on June 16, 1952. Almost three weeks after the single aspiration of 5¼ pints of haemorrhagic effusion. The left dome of the diaphragm is raised and a small effusion persists.

(0.2 g.), ascorbic acid (50 mg.), and "casilan" (15 g.) each thrice daily. No blood transfusion was given throughout.

Two months later he returned to work, having gained a further 7 lb. in weight. He has since been followed up as an out-patient at three-monthly intervals. The E.S.Rs. and chest radiographs, apart from cystic disease in the left mid-zone (Fig. 3), have remained normal. There has been no evidence of tuberculosis, including examination of the sanguineous fluid directly and on culture.

CASE 2.

J.G., a robust foundry-worker, aged 29, was admitted on August 8, 1952, with a history that 10 days previously he experienced a sudden severe pain in the left side of the chest, especially posteriorly, but also felt in the left upper abdomen. He remained in bed under his doctor's supervision. The pain subsided after one day, but 4 days before admission it recurred with its previous severity. It was aggravated by deep breathing and coughing. He now felt listless and breathless and had an irritating non-productive cough. His doctor stated that there had been no pyrexia.

The past history included no illness of note, and, in particular, no chest illness or symptoms, but the family history revealed that five years ago a sister had spent a year in a sanatorium.

On physical examination he was pallid, but showed no venous congestion or distress. The respiratory system showed the classical signs of a moderate left-sided pleural effusion, including displacement of the trachea to the right. The pulse was regular at a rate of 82 per minute and of moderate volume, while the rest of the cardiovascular system was

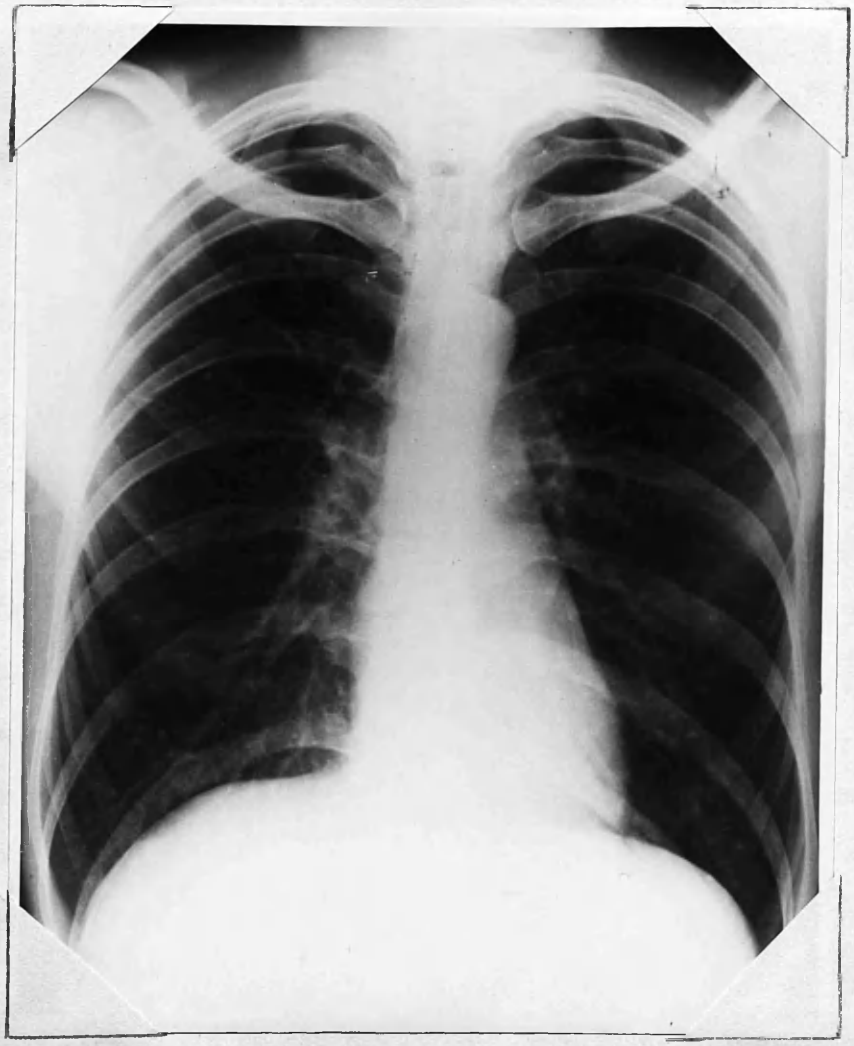

FIG. 3. Case I. Radiograph on January 8, 1953, six months after leaving hospital. Lung fields now normal apart from cystic disease in the left mid-zone.

also normal, apart from a rather low diastolic pressure (130/60 mm. Hg.). The other systems were normal.

Initial investigations revealed a haemoglobin level of 88%, a normal total and differential white blood count, and an E.S.R. of 42 mm. (Westergren) in one hour. The radiograph of the chest showed a left-sided pneumothorax with effusion and associated partial collapse of that lung. Immediate diagnostic thoracentesis produced 8 oz. of fluid resembling venous blood. This showed 59% haemoglobin, an erythrocyte count of 2,760,000 per cu.mm., and a leucocyte count of 3,600 per cu. mm. (polymorphonuclears, 17%; lymphocytes 70%; eosinophils 13%) compared with the concurrent peripheral blood count of 89% haemoglobin, an erythrocyte count of 4,200,000 per cu. mm., and a leucocyte count of 7,900 per cu. mm. (polymorphonuclears 60%; lymphocytes 33%; eosinophils 5%; monocytes 2%). The radiographic appearances of the chest remained unchanged. Next day (August 9), despite frequent attempts at aspiration, only 2 oz. of fluid was removed, but four days later 1 pint of haemorrhagic fluid was withdrawn and the radiograph now showed that the left lung had almost completely re-expanded, although a small effusion persisted. The aspirated fluid contained 22% haemoglobin and 830,000 per cu. mm. of red cells.

Up to August 26 there was evidence of recurrence of the effusion (Fig. 4). This refractory phase, coupled with evening pyrexia to about 100° F., prompted further aspiration, when 30 oz. of xanthochromic, relatively acellular fluid was withdrawn. This fluid had a protein concentration of 4.9 g. % and a bilirubin level of 1.7 mg. %. This aspiration was immediately followed by the instillation of "varidase" (200,000 units of streptokinase and 50,000 units of streptodornase,

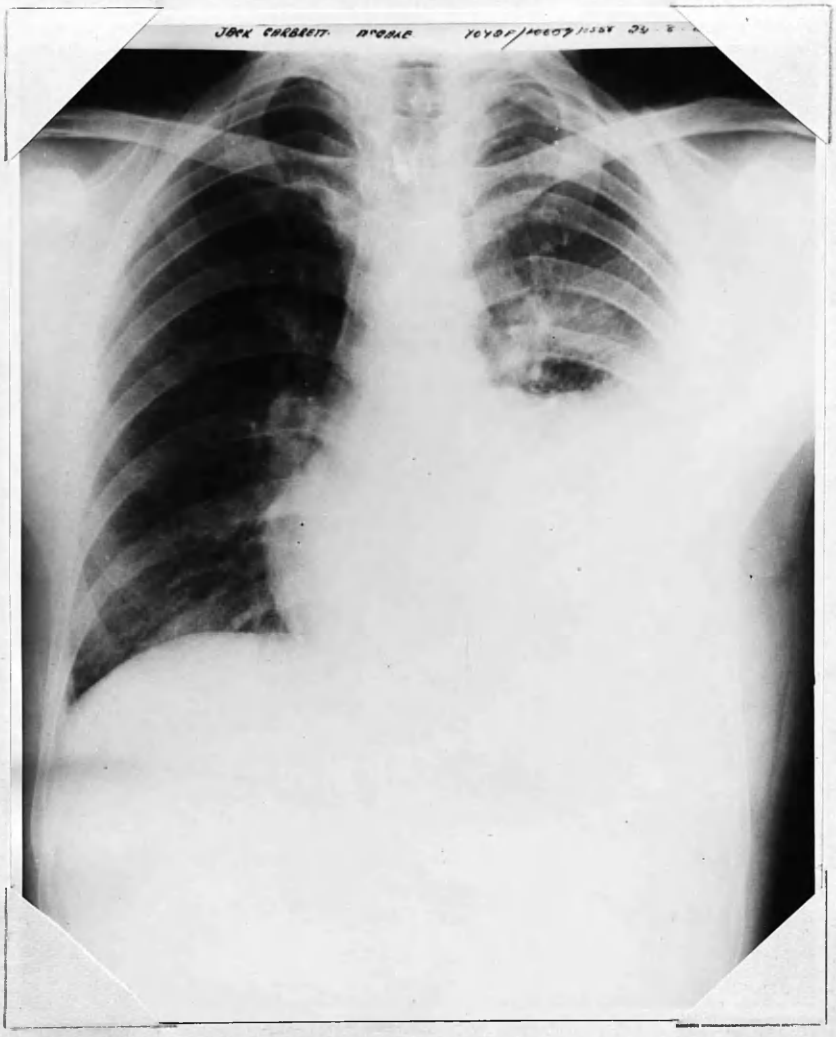

FIG. 4. Case 2. Radiograph on August 26, 1952, showing recurrence of left-sided effusion preceding the aspiration of a further 1½ pints of haemorrhagic fluid and the instillation of "varidase."

dissolved in 10 ml. of physiological saline) into the pleural cavity. During the next 48 hours the patient felt further distress. There was a recurrence of chest pain, breathlessness, perspiration, pyrexia (100-102° F.), a tachycardia (90-110 per minute), a leucocytosis of 13,100 per cu. mm. (polymorphs 90%, lymphocytes 10%), and increased effusion. On August 27 a pint of clear, relatively cell-free fluid was aspirated. Its bilirubin level was 0.15 mg. %. Uneventful progress ensued. Further thoracenteses were unproductive. By September 17 the chest radiograph revealed only partial collapse of the left lung, associated with a small effusion.

On October 3 he was discharged home, feeling fit and regaining weight. Follow-up has shown virtually a complete recovery, for the serial chest radiographs have appeared normal since the film of October 15 (Fig. 5). He resumed work one month after discharge. He had then gained a further 7 lb. in weight. No evidence of pulmonary tuberculosis was obtained from the radiological investigations or sputum (four separate specimens were examined). The Mantoux test was positive to a 1 in 1,000 dilution.

CASE 3.

F. C., a housewife, aged 40, was admitted on June 22, 1953, for the treatment of a recurrent compound ganglion on the dorsum of the wrist with a course of combined streptomycin and P.A.S.. An identically located ganglion had been excised two years previously and it revealed typical melon-seed bodies with the histological appearance of tuberculosis.

Apart from three attacks of rheumatic fever in childhood, producing no subsequent evidence of cardiac disease, she had no previous illnesses of note. Although there was a strong family history of tuberculosis, her

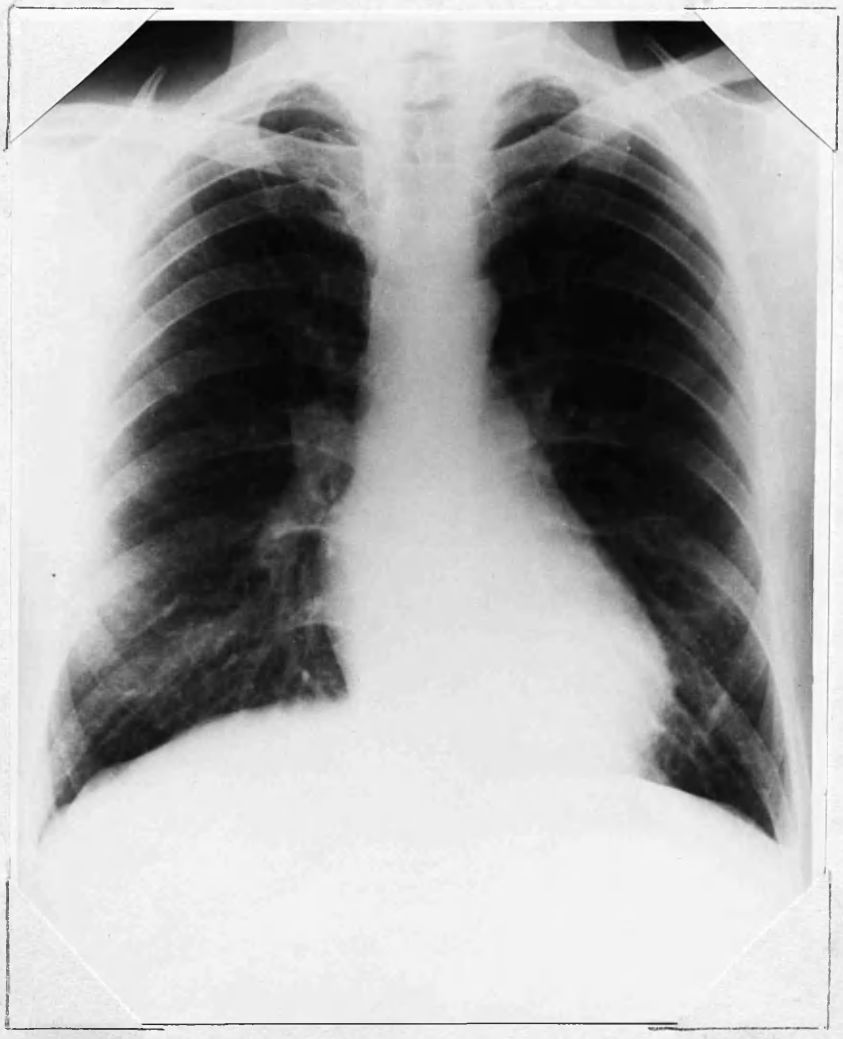

FIG. 5. Case 2. Radiograph on October 15, 1952, a fortnight after discharge from hospital. No residual effusion.

frequent serial chest radiographs and erythrocyte sedimentation rates had always been normal. In particular, the lung apices remained clear.

Physical examination showed no striking signs apart from facial pallor, koilonychia, and the ganglion. Her blood pressure was 140/80 mm. Hg.. A blood count revealed haemoglobin 73%, erythrocyte count of 3,600,000 per cu. mm., mean cell diameter 7.2 μ and a leucocyte count of 14,000 per cu. mm. with a normal differential count. The mean E.S.R. was 9 mm. in one hour. On August 10, 1953, the ganglion was excised and again it contained typical melon-seed bodies.

On August 13 her temperature rose to 99° F., while she experienced left-sided pleuritic pain, unaccompanied by a pleural rub or an effusion. The following afternoon she suddenly became shocked and dyspnoeic with a moderate left-sided pleural effusion. The mediastinum was not displaced (Fig. 6). The relatively sudden onset of a large pleural effusion and pallor immediately suggested a diagnosis of haemothorax. This was promptly confirmed by the aspiration of apparently pure blood with a haemoglobin value of 62%. Moderate shock was shown by free perspiration, a pulse rate of 120 per minute, and hypotension (80/40 mm. Hg.). A half-hourly pulse and blood pressure chart was now begun and a blood transfusion set up. By 10 a.m. next day (August 14) her haemoglobin level was only 64%, despite the administration of 4 pints of blood and 1 pint of plasma. The blood pressure was now 120/80 mm. Hg., but the pulse rate had increased to 140 per minute. Therapeutic thoracentesis was now begun, but after the aspiration of 2 pints of sanguineous fluid the patient demanded a respite. Meanwhile blood transfusion was continued; a further 5 pints of blood were at hand.

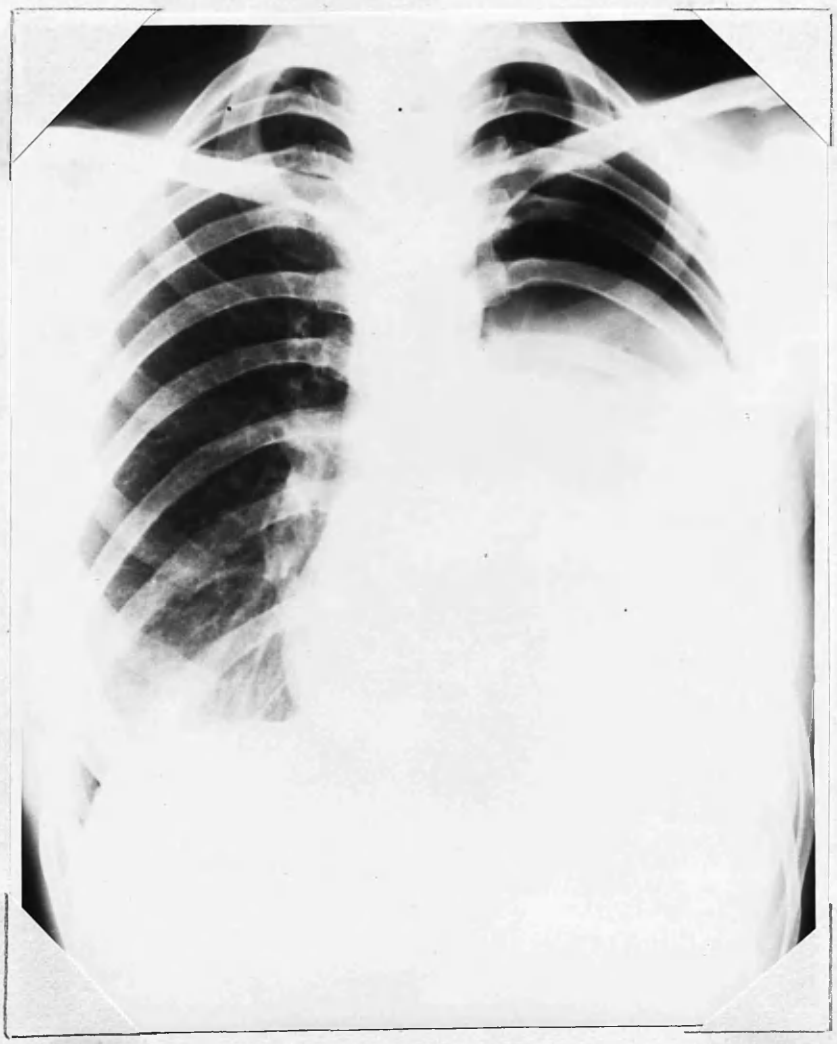

FIG. 6. Case 3. Radiograph on August 14, 1953, a large initial left-sided pleural effusion before thoracentesis was begun, but no mediastinal displacement.

By 2.30 p.m. the blood pressure was 140/85 mm. Hg., with the pulse rate still 140 per minute, but rose to 150 per minute by 4 p.m.. The blood pressure was unchanged. The chest radiographs (Fig. 7) now showed mediastinal shift to the right with a persistent large pleural effusion. Respiratory distress continued without signs of cerebral anoxia. Just before further aspiration at 4.30 p.m. there was a sudden general deterioration. The blood pressure dropped abruptly to 110/60 mm. Hg. and the pulse was thready and its rate was uncountable. A further 2 pints of the haemorrhagic fluid were promptly aspirated, but a syncopal attack precluded further immediate thoracentesis. At 5 p.m. she lapsed into extreme respiratory distress, accompanied by restlessness, disorientation, and delirium. The pupils were now large and reacted only sluggishly to light. As an arterial transfusion was being set up the patient died at 5.30 p.m..

Necropsy revealed $2\frac{1}{2}$ pints of partly clotted blood in the left pleural sac, while the underlying lung was completely collapsed. A stretched and torn apical pleural adhesion with a bleeding point was located between the necks of the first and second ribs. The lungs showed no evidence of tuberculosis. It was concluded that the haemopneumothorax had resulted from the spontaneous tear of an old tuberculous pleural adhesion.

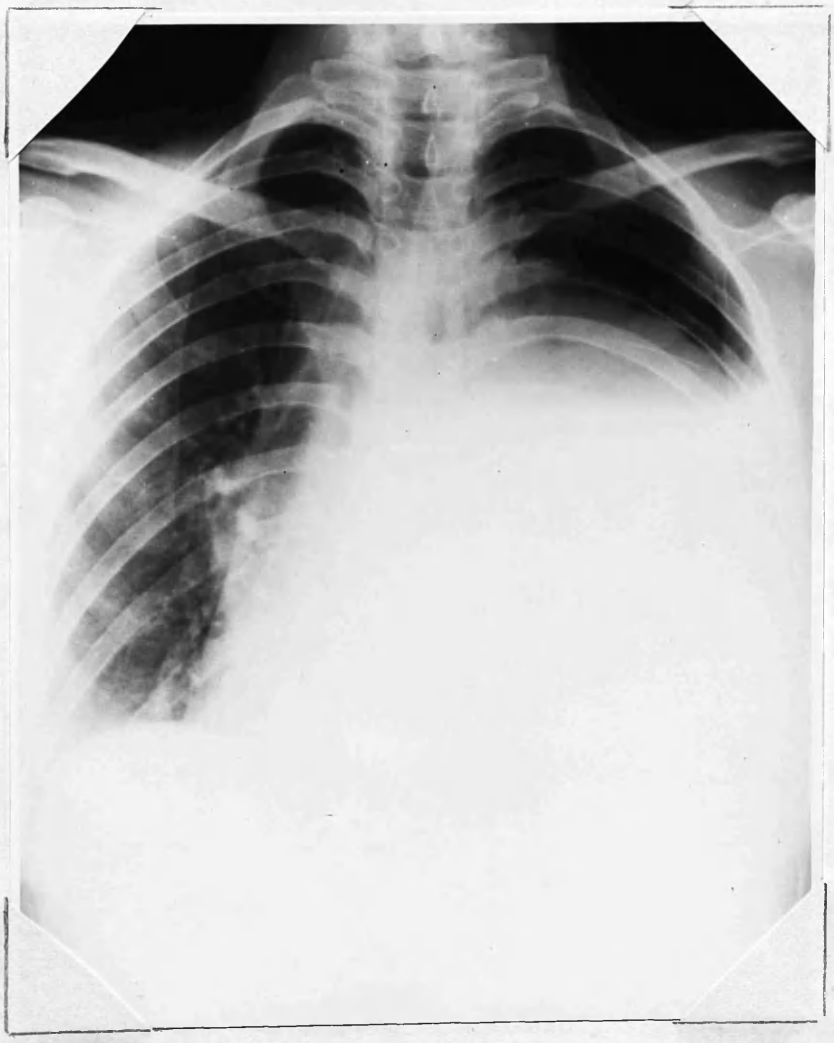

FIG. 7. Case 3. Radiograph on August 15, 1953, three hours before death. The radiograph resembles Fig. 6, but there is now mediastinal shift.

CASE 4.

J.W., a motor mechanic aged 27, was admitted to hospital on October 2, 1954, with a provisional diagnosis of spontaneous pneumothorax. At 10.30 a.m. that morning, while working, he was suddenly seized with a stabbing pain in the left mammary region. The pain, pleuritic in type, was aggravated by movement and radiated to the neck, left shoulder and scapular region. The pain persisted, with slightly increased intensity, despite the injection of morphine (gr. $\frac{1}{4}$) from his doctor. A further gr. 1/6 was injected on arrival at hospital, early in the afternoon and the dose was repeated half an hour later.

His previous history was exclusively related to his chest. He had whooping-cough when aged 13, dry "pleurisy" at 20, and an injury to the right side of the chest, necessitating observation in hospital for 3 days, two years prior to the present admission. This injury followed a fall at work and resulted in the fracture of two ribs on the right side. There was no family history of tuberculosis.

Examination, in hospital, disclosed angular build, slight orthopnoea without cyanosis, moderate pallor, and disquietude. The pulse was normal; its rate, 78 per minute. The apex-beat was impalpable and the cardiac sounds were pure but distant and were best heard in the sternal region. The blood-pressure was 125/80. Respiratory signs included rapid (32 per minute) and shallow breathing, tracheal shift to the right and classical signs of a left-sided hydropneumothorax. The pleural effusion was of moderate extent, radiologically.

Immediate thoracentesis produced 3 pints of fluid, resembling blood. Its haemoglobin value was 76 per cent (Sahli). Following aspiration, the radiograph showed virtually complete re-expansion of the left lung, no

fluid and normal lung-fields. The initial blood count, just prior to aspiration, showed:- haemoglobin 73 per cent; leucocytes, 6,300 per cu. mm. (P., 68%; L., 23%; E., 6% and M., 3%), while the E.S.R. was 32 mm. (Wintrobe) in one hour.

Further progress was rapid. Blood transfusion was not required. Apart from occasional twinges of pain at the left base, on certain movements, he was now fit. Pyrexia was restricted to the first three days, but readings did not exceed 100° F. Clinically, there was equivocal deficient movement at the left base, which was still impaired to percussion. However, these signs had cleared at the end of the first week, when the X-ray of chest was normal. Investigation of sputum for the acid-fast bacillus was negative for three specimens of sputum. Two weeks after admission, he was allowed up and transferred to a convalescent ward.

On October 30th, exactly one month from the onset, he was discharged from hospital. His haemoglobin level was now 88 per cent and the E.S.R. 11 mm.. He resumed work one month later. Surveillance, now for one year, has shown no clinical nor radiological abnormality.

CASE 5.

R.W., a gas fitter aged 36, was admitted on August 22, 1955. Three days previously, during work, he was seized with a sharp pain in the left lower thoracic region. This pain was pleuritic and lasted for several hours. Progressive breathlessness ensued. On arrival in hospital, he was orthopnoeic.

There was no family history of tuberculosis and no personal history of respiratory disease. He had always enjoyed good health, apart from a

brief episode of haematemesis from a peptic ulcer four years previously.

Physical examination revealed restlessness, extreme pallor and moderate cyanosis. Signs were otherwise restricted to the respiratory and cardiovascular systems. The trachea was deviated to the right. The respiratory rate was 32 per minute and the respiratory excursion was diminished at the left base, where the percussion note was flat and vocal fremitus, vocal resonance and breath-sounds were all absent. The percussion-note in the left upper anterior zone was hyper-resonant. A succussion-splash was elicited. The pulse was rapid (130 per minute), regular and thready, the blood-pressure was 80/40 mm. Hg. and the apex-beat was in the fifth left interspace in the parasternal region. The cardiac sounds were faint, but pure.

As preliminary treatment, the foot of the bed was slightly elevated, morphine sulphate (gr. 1/6) was injected hypodermically and 2 ml. of coramine intravenously, followed by immediate thoracentesis. When, however, two pints of almost pure blood had been aspirated, the patient felt faint and was granted a respite. The haemoglobin level of the aspirated fluid was 68 per cent, Haldane (10.0 G %) while the concurrent value for the peripheral blood was 60% (8.9 G %).

One hour later (7 p.m.) profuse perspiration occurred and the pulse was very feeble, with unchanged rate. Morphine (gr. 1/6) was injected immediately and at 8 p.m. a dextrose-saline intravenous drip was commenced (pulse 130 per minute; blood-pressure 80/60) and followed by four pints of blood overnight.

At 9 p.m. the intra-pleural pressure was recorded and the aspiration of air commenced (see small Table). A total of 1,350 cc. was withdrawn in half an hour.

Intra-pleural Pressure (cm. H_2O)		Volume of Air Removed (cc.)	Pulse Rate
Insp.	Exp.		
+5	+10		130
-1	+ 5	500	130
-3	+ 3	300	108
-5	0	550	100

Re-examination at 9.30 p.m. showed little improvement. As the general condition and the signs in the left hemithorax were virtually unchanged, it was concluded that haemorrhage was still occurring. The pulse rate was 110 per minute.

Next day (23.8.55) shock persisted and a radiograph of the chest showed a massive left-sided hydrothorax (Fig. 8). At 8.30 a.m. the pulse-rate was 130 per minute and the blood-pressure 90/80. Blood transfusion was now recommenced, with six pints of blood, available. The thoracic surgeon was promptly informed and emergency thoracotomy arranged. By 12.45 p.m., the dyspnoea was increased, the pulse-rate, 160 per minute and the blood-pressure 95/55. At 2 p.m., in view of urgent dyspnoea, under-water seal drainage was instituted to reduce the intra-pleural pressure prior to thoracotomy.

At 3.45 p.m., following a total of 10 pints of blood transfused, the operation was begun. Per-operative inspection revealed a collapsed left lung, copious blood-clots in the pleural cavity and a few apical adhesions, from one of which blood was oozing. No emphysematous blebs were seen.

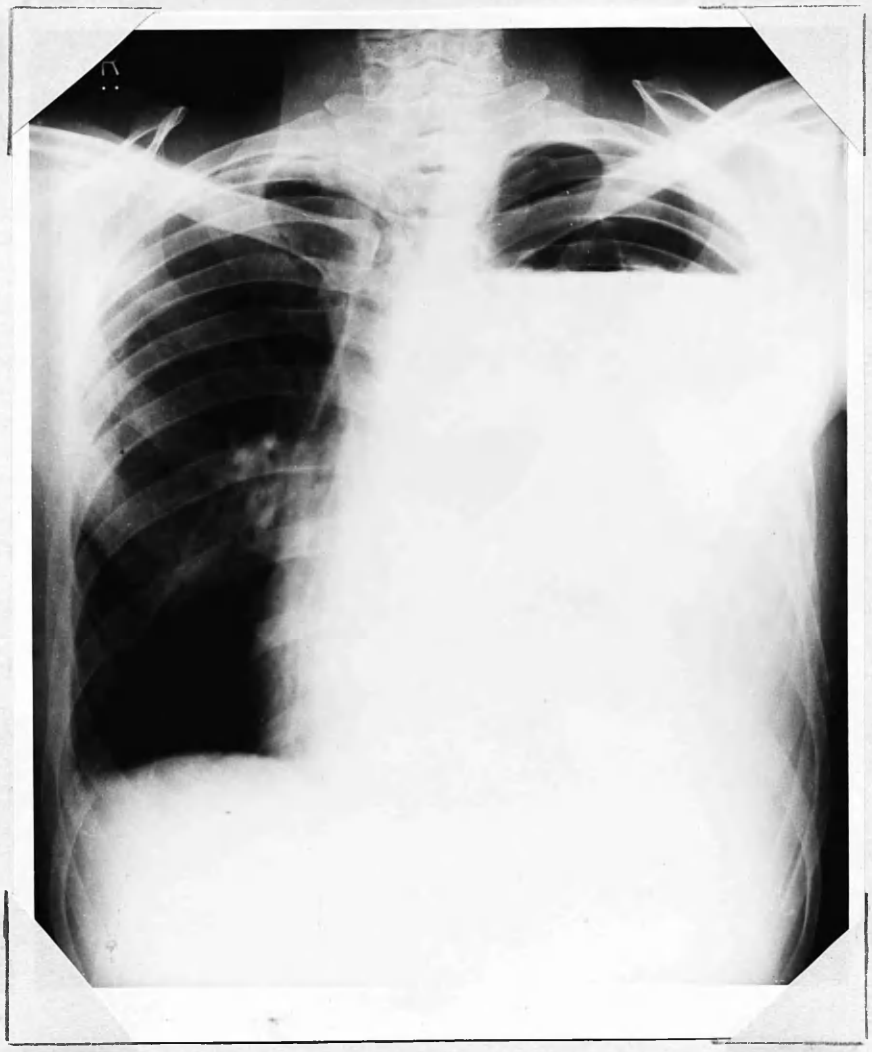

FIG. 8. Case 5. Radiograph on 23rd August, 1955 displaying a massive left-sided hydropneumothorax, despite the aspiration of 2 pints of sanguineous fluid shortly after admission the previous afternoon.

The clot was evacuated, the pleural cavity cleared of several pints of blood and the bleeding point was diathermised. A thoracotomy-tube was inserted and further under-water seal drainage set up. The immediate post-operative blood-pressure was 95/55. The pulse volume had improved, but tachycardia (120 per minute) persisted. Crystalline penicillin, 1 mega units intra-muscularly immediately and six-hourly for 5 days, was ordered.

By 7.30 p.m., the patient's general condition had improved considerably. The pulse rate was 110 per minute and the blood-pressure 100/70. Transfusion was continued. At 11 p.m., examination of the chest showed minimal signs of effusion. In all, 11 pints of blood had now been transfused and the twelfth had just been commenced.

Next day (24.8.55), progress continued. The blood-pressure was 120/60. Physiotherapy was begun. There was slight pyrexia (100° F), tachycardia (104 per minute) and leucocytosis (12,300 per cu. mm.) with a normal differential count. The haemoglobin level was now 94% (13.9 G %). The drainage-tube had become blocked but was washed out with normal saline prior to the intra-pleural instillation of 2 mega units of crystalline penicillin.

On 25.8.55, the pulse rate was 90 per minuts. Signs of consolidation and slight effusion were present at the left base (Fig. 9) with an unexplained area of consolidation in the right mid-zone. The drainage-tube was removed.

Next day, a pleural rub was audible anteriorly on the left side of the chest. Leucocytosis (14,000 per cu. mm.) continued and the differential leucocyte-count now showed an eosinophilia (16%).

On 29.8.55, the temperature and pulse-rate were normal and the patient was allowed to get up. Leucocytosis (15,700 per cu. mm.) persisted, but

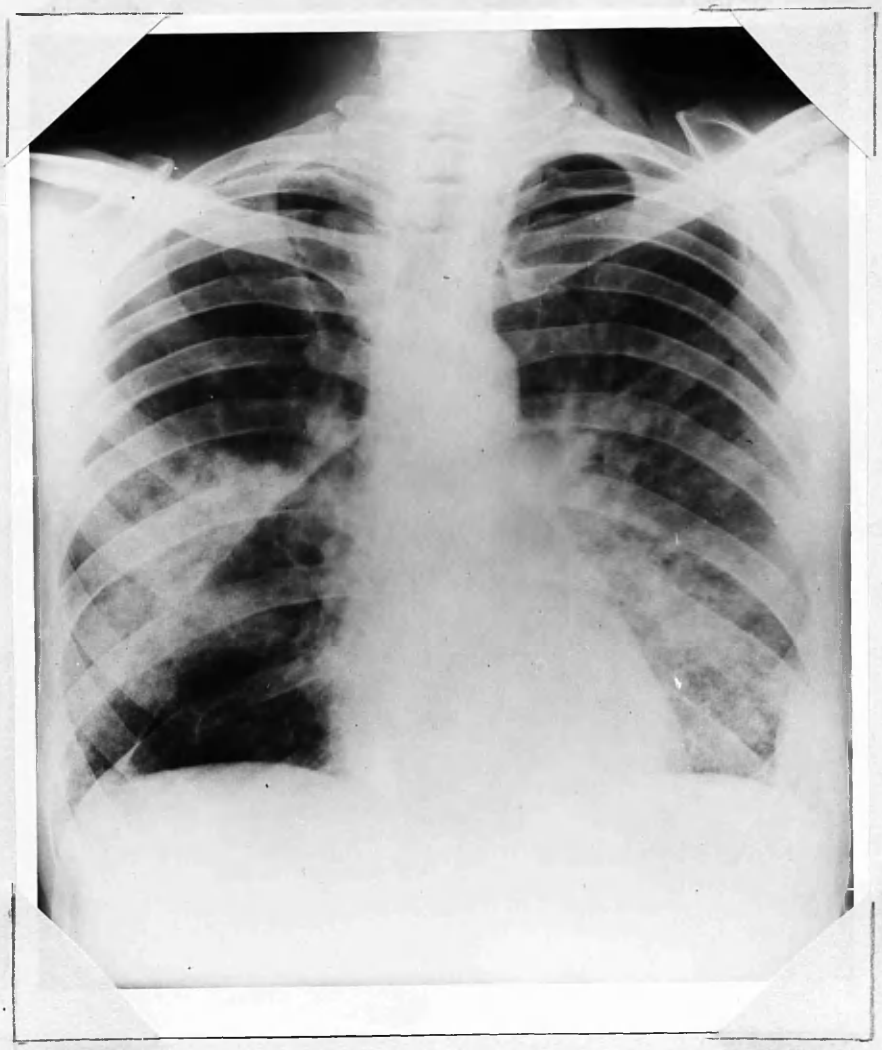

FIG. 9. Case 5. Radiological appearances on 25th August, 1955, i.e. after emergency thoracotomy. A small left basal effusion has re-accumulated post-operatively, while right mid-zonal consolidation is now apparent.

with only 2 per cent eosinophils.

On 6.9.55, the patient was discharged from hospital. The radiograph of the chest was now normal (Fig. 10) and the leucocyte count, almost normal (10,800 per cu. mm.) with a normal differential count. When last seen (19.1.56), he was perfectly fit and his radiograph of chest was normal.

CASE 6.

(Admitted to St. Andrew's Hospital, Billericay, Essex, under Dr. J. Fawcett, to whom I am indebted for permission to include this case-legend).

J.H.M., a bus-driver aged 23, was admitted to hospital as a medical emergency on November 29, 1955. During normal duty, the previous afternoon, he suddenly experienced a sharp pain, pleuritic in type and most severe in the left shoulder and back of chest. This pain radiated down the left arm and side of chest.

Duty completed, he returned home and immediately took to bed. There was no undue distress and he was treated symptomatically. However, he spent a very restless night on account of increasing breathlessness, now accompanying the pain. Next morning, his symptoms were more alarming and his urgent admission to hospital was sought, with a provisional diagnosis of tension pneumothorax. There had been no salient previous illness and there was no family history of tuberculosis.

Physical examination in hospital revealed striking dyspnoea, asthenic habitus (weight said to be 8 st. 6 lb.), apprehensive demeanour, and pallor but no cyanosis. Relevant signs were restricted to his chest. The pulse was of moderate volume, with a rate of 120 per minute. There was minimal

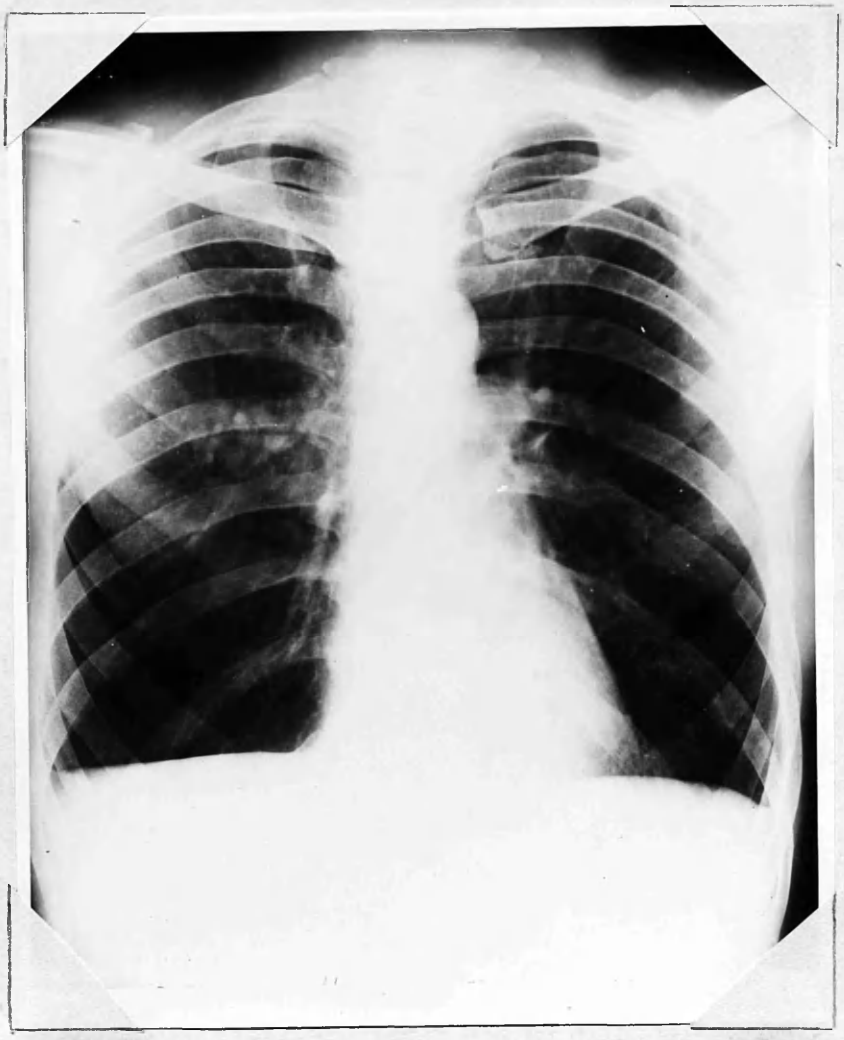

FIG. 10. Case 5. Radiograph on 6th September, 1955, i.e. a fortnight after admission, showing normal lung-fields.

jugular venous engorgement. The apex-beat was not located and the cardiac sounds were faint put pure. The blood-pressure was 115/75.

The respiratory signs included tachypnoea (32 per minute), orthopnoea, immobility of the left hemithorax, tracheal deviation to the right, and classical signs of a left-sided hydropneumothorax. There was no bulging of the inter-costal spaces to suggest a tension pneumothorax. A succussion splash was elicited. The emergency radiograph showed virtually complete collapse of the left lung, moderate mediastinal displacement to the right, and a fluid level corresponding to the posterior segment of the left 9th rib. The haemoglobin value was 84 per cent (12.4 G %) and the E.S.R., 34 mm. (Westergren) at one hour.

Immediate thoracentesis verified a suspected haemopneumothorax. Aspiration yielded 1,600 cc. of "blood," which did not clot, in vitro. The final intrapleural pressure was recorded as zero $\pm$ 2 cm. of water. Radiographically, a partial re-expansion of the left lung and correction of the mediastinal displacement were demonstrable. Blood-transfusion was now commenced and during the next two days six pints were given.

Next day, re-aspiration was only partially successful, amounting to 350 cc. of similar fluid and 700 cc. of air. The final intrapleural pressure was - 8, + 2 cm. of water.

A day later (1.11.55) there was no clinical or radiographic evidence of improvement. Indeed, the left lung was completely collapsed, while the X-ray film also showed an indistinct opacity just above the fluid-level. This opacity, much less dense than the fluid, was interpreted as fibrin. Aspiration again failed to produce any definite improvement. Only 100 cc. of sanguineous fluid and 600 cc. of air could be withdrawn. Intrapleural

fibrinolytic enzyme-therapy was given a brief trial. A combined solution of 200,000 units of streptokinase and 50,000 units of streptodornase were instilled at the end of the last-mentioned thoracentesis. A constitutional upset ensued. This included malaise, pyrexia (102.4° F) and tachycardia (120 per minute) but no rigors. A similar treatment with half-dosage was carried out next day to facilitate an anticipated decortication, in view of persistent "supernatant" opacities which were interpreted as fibrin.

The patient was transferred to a thoracic unit, where an inter-costal catheter was inserted in the 2nd interspace anteriorly. There was a definite escape of air and a considerable amount of fluid was drained. Dramatic improvement followed.

On 7.12.55, nine days after the onset of symptoms, thoracotomy was performed. Per-operatively, neither the original source of escape of air nor of blood could be identified. After the removal of the residual pleural fluid, a tedious pulmonary decortication was carried out. One pint of fibrin-clot was removed from the pleura. Two drainage tubes were inserted. Initial powerful suction was applied to these tubes and a partial re-aeration of the left lung was obtained. Next day, full re-expansion was present. Subsequent progress was uneventful.

The patient was discharged on December 16, 1955, feeling well. However, there was deficient respiratory excursion on the left side, despite a normal radiograph of the chest.

CASE 7.

J.G., aged 33, a driver's mate was admitted to hospital on December 16, 1955, with a provisional diagnosis of spontaneous pneumothorax. While lifting a crate, 2 days previously, he was momentarily transfixed by the

sudden onset of a lancinating pain in the front of the left side of the chest. This pain radiated to the back. Although the pain continued, he completed his day's work and even returned to work next morning, despite breathlessness on minimal exertion. He was unfit to resume and, instead, consulted his doctor, who referred him initially to a chest-clinic. The patient, however, was too ill to attend. His admission to hospital was expedited next morning(16.12.55).

His past history included measles and whooping-cough in infancy, and "bronchitis all his life," while four years previously he had experienced a short bout of pain in the left side of the chest, for which he was referred to a chest-clinic. There, he was informed that he had no present lung-disease, but that he had probably once had tuberculosis. There was no family history of tuberculosis.

On admission, examination revealed asthenic build, pallor and breathlessness, but he was quite comfortable while he remained in an oxygen tent. The respiratory signs included orthopnoea, tachypnoea (26 per minute), a dextro-posed trachea, negligible respiratory movement over the entire left hemithorax, which was stony dull to percussion in its lower half. Vocal fremitus, vocal resonance and breath-sounds were all absent at this site. By contrast, at the left apex the percussion-note was hyper-resonant and faint vesicular breath-sounds were audible. A succussion splash was elicited. There was no evidence of a tension pneumothorax.

The remaining relevant signs were cardiovascular. These included minimal venous congestion, an impalpable apex-beat, distant but pure cardiac sounds best heard in the right parasternal region. The pulse -rate was 90 per minute and its volume was fair. The blood-pressure was 110/60.

Morphine (gr. 1/6) was injected statim. Immediate thoracentesis produced $3\frac{3}{4}$ pints of almost pure blood and, finally, air also. The X-ray film preceding aspiration had shown a $\frac{2}{3}$ rd's collapse of the left lung and fluid up to the level of the 7th thoracic vertebra. After thoracentesis, however, the radiological appearances were a central mediastinum, a normal right lung-field, a persistent $\frac{2}{3}$ collapse of the left lung, while fluid obscured the left costophrenic angle. The haemoglobin level in the peripheral blood was 76 per cent (11.2 G %). For the remainder of the day, he was comfortable, except for slight pain requiring morphia, gr. 1/6 nocte.

Next morning, improvement was sustained and re-aspiration yielded 18 oz. of similar, but darker, fluid, followed by air. No attempt was made to fully aspirate the air, as this procedure seemed to initiate pleuritic pain. Crystalline penicillin, half a mega units, was injected into the pleural cavity. A full blood-count now showed:- haemoglobin value 76 per cent (11.2 G %); erythrocytes, 3.86 million per cu. mm.; colour index, 1; leucocytes, 17,100 per cu. mm., with a normal differential count. The radiograph, at this stage, revealed almost full re-expansion of the left lung.

The following day (20.12.55), pleuritic pain returned for several hours but physical examination showed only minimal signs of basal pleural thickening, confirmed radiologically. Movement of the left side of the chest was now unimpaired. The haemoglobin level had unexpectedly dropped to 52 per cent (7.7 G %) but the leucocyte count was normal (5,100 per cu. mm.).

Thereafter progress was uneventful. The temperature, which had fluctuated to 100° F during the first four days, was now normal. On 30.12.55,

he was allowed up and on 4.1.56 he was discharged from hospital. The haemoglobin level was now normal (96 per cent), following iron medication, without resort to blood transfusion. Three serial examinations of sputum for tubercle bacilli were all negative. The X-ray film now showed slight residual opacity in the left costophrenic sulcus, as the sole abnormality. Three weeks later, he returned to work.

CASE 8.

J.E.H., a compositor aged 22, was admitted to hospital on January 3, 1956 with a tentative diagnosis of spontaneous pneumothorax, although his doctor's first clinical impression was coronary thrombosis.

Shortly after starting work that morning, whilst in relative inactivity, the patient experienced a dramatic onset of symptoms. A tight sensation in his chest was rapidly followed by excruciating precordial pain, which radiated down the left arm. When seen by his doctor at 10.30 a.m., morphine (gr. $\frac{1}{4}$) was injected forthwith. The left arm had become transiently numb but was not paralysed. There was a feeling of faintness but no loss of consciousness. At no time was there any breathlessness but the pain continued until shortly after arrival at hospital that afternoon.

The previous history included long-standing migrainous attacks and, within the past year, six bouts of chest-discomfort, described as a mild version of his presenting symptoms. There had been no other notable illness related to his chest. The family history was negative for tuberculosis.

Examination, in hospital, revealed spare build (weight said to be 8 st. 10 lb.), striking pallor and moderate distress, despite the absence

of breathlessness and cyanosis. The cardiovascular signs were:- pulse-rate of 90 per minute and with diminished volume; apex-beat, unlocated; cardiac sounds, distant but pure; and blood-pressure, 80/60. The respiratory signs were typical of a left-sided hydropneumothorax, with a moderate-sized effusion. Hippocratic succussion was not demonstrable. The immediate radiograph displayed mediastinal dextro-displacement and fluid up to the level of the second left intercostal space anteriorly. The haemoglobin level of the peripheral blood was 84 per cent, Haldane (i.e. 12.4 G %).

Following the injection of morphine (gr. 1/6), emergency thoracentesis produced 2 pints of sanguineous fluid, with a haemoglobin level of 34 per cent (5.0 G %). Blood transfusion had been arranged but was, as yet, withheld. During the rest of that day, there were no remarkable incidents.

Early next morning, however, he appeared to be more agitated, and hypotension (105/60) persisted. Blood-transfusion was commenced at 2 a.m.. At 2.30 a.m., although there were no signs of a tension pneumothorax, it was decided to register the intrapleural pressure. The initial readings were -10, -3 cm. of water, and a fractional aspiration of 700 cc. of air was conveniently carried out through an upper anterior approach. The final readings were - 14, -9 cm. of water. The pulse-rate was now 68 per minute and the blood-pressure, unchanged (105/60). The X-ray film showed little residual effusion and almost complete re-aeration of the left lung.

Next day (5.1.56) was comparatively uneventful, following the transfusion of a total of 5 pints of blood. The pulse-rate and blood-pressure were unaltered.

On 6.1.56, he complained of a sharp pain in the left shoulder, but was

otherwise comfortable. His haemoglobin value was 82 per cent (12.1 G %).

There was an unexpected mild recrudescence of signs on 9.1.56. Clinically and radiologically, it seemed certain that there was a re-accumulation of pleural fluid and, possibly, air. The intrapleural pressure was found to be -5, -3 cm. of water. Following the extraction of 600 cc. of air, readings of -14, -10 cm. of water were obtained. A further 50 oz. of sanguineous fluid, with a haemoglobin value of 44 per cent (6.5 G %) were readily withdrawn. The peripheral blood-count showed 96 per cent haemoglobin (14.2 G %).

On successive days, 13 oz. of blood-stained fluid along with 200 cc. of air, and $6\frac{1}{2}$ oz. of rosy serous fluid were removed. Serial radiographs showed further improvement and, on discharge from hospital one month after admission, there was only a very small area of residual shadowing detectable at the left base. Examination for acid-fast bacilli was negative on three separate specimens of sputum.

In summary, he was transfused 5 pints of blood, while a total of 1,500 cc. of air and $5\frac{1}{2}$ pints of sanguineous pleural fluid had been aspirated.

Commentary on the Author's Cases.

Discussion will be reserved for the salient features.

Relation to Tuberculosis.

No patient had radiological evidence of concurrent pulmonary tuberculosis during admission for haemopneumothorax.

The remote past histories were uniformly non-contributory in terms of clinical tuberculosis. However, two years prior to her admission, Case 3 had a ganglion excised from the wrist. The histology was consistent with a tuberculous pathology. Nevertheless, subsequent serial radiographs of her chest were consistently normal. As in Case 2, she had a family history of tuberculosis. In neither patient, however, was there any direct evidence of pulmonary tuberculosis from examination of sputum, radiological study or, in Case 3, at autopsy. Likewise, serial radiographs in Case 2 have not revealed tuberculosis. Again, in Case I, a fortuitous X-ray of chest, taken 3 months before admission, was normal, while subsequent films, as in Case 4, have all appeared normal. The torn pleural adhesion, noted at autopsy in Case 3, might well have been a relic of tuberculosis, yet the histological appearances of this adhesion were non-specific, as other investigators have similarly found. This topic will be resumed in Section 3.

Inter-relation with Idiopathic Pneumothorax.

Only Cases 4, 7 and 8 gave histories, which might suggest earlier episodes of pneumothorax. Yet, in none was this proven. In Case 4, the attack of "dry pleurisy," 7 years prior to the onset of haemopneumothorax, might well have been a pneumothorax, but no radiograph was taken then. However, in Case 7, the bout of chest-pain, which occurred 4 years prior to the haemopneumothorax, suggested the possibility, in retrospect, of a

pneumothorax. Yet, this was apparently not detected on fluoroscopy at the chest-clinic. Again, in Case 8, it is considered likely that the six episodes of chest-pain, during the year preceding the haemopneumothorax, were minor pneumothoraces but radiological investigation was lacking.

Case 2 had intense pain in the chest for 24 hours, commencing ten days before admission. It is convenient, at this point, to emphasise that the onset of pain, during the week or two prior to entry to hospital, is recorded in the histories of 5 per cent of all patients with idiopathic haemopneumothorax. The only tenable explanation of this early attack of pain is that it represents the onset of the pneumothorax, while the complicating haemothorax is assumed to arise from the late rupture of an adhesion, which has been stretching by the collapsing lung.

The Problem of Haemorrhage.

This problem calls for three special decisions:-

(a) When did the haemorrhage commence?

(b) Appropriate course of action during the haemorrhage.

(c) When has haemorrhage ceased?

(a) Time of Onset of Haemorrhage.

Difficulty in settling this point only arises in those patients with a history of pain arising several days prior to admission. This is exemplified in Case 2. This patient's recurrent pain, which began 4 days before admission may signify the initiation of haemorrhage from a torn adhesion. This point remains speculative, since haemorrhage for 3 days, even assuming its origin in a small vessel, should have produced greater exsanguination. Alternative explanations exist and include the arguments that the haemorrhage

might well have ceased prior to admission or was intermittent. Since repeated immediate thoracentesis at different sites yielded little fluid, an underlying partially-clotted haemothorax was suspected. This view encouraged resort to the intrapleural instillation of fibrinolytic enzymes since, infection excluded, the favoured explanation of intrapleural clot-formation was the intrapleural retention of blood for several days. This view would harmonize with a presumed onset of haemorrhage four days before admission. It may be significant that, apart from Case 8, this was the only one of the 5 relevant patients with a higher haemoglobin level in the peripheral blood than in the pleural-fluid. This observation could denote that the haemorrhage had begun at least a few days previously. In the other 4 patients, adequate thoracentesis was conducted within 24 hours of the presumed onset of haemorrhage and in none of the 4 patients was there reason to suspect a partially-clotted haemothorax.

However, parallel observations on Case 8 clearly illustrate the occasional unexpected fallacy in drawing such firm conclusions on the exact time of onset of haemorrhage. Indeed, in Case 8, it can be safely concluded that the intrapleural haemorrhage was of several days standing, despite good evidence, from the history, that the haemorrhage had occurred within 24 hours of admission. The haemoglobin level (34 per cent) in the pleural fluid is much more consistent with the haemorrhage having commenced fully one week previously. In concluding the discussion on this controversial subject, it must be reiterated that, whilst one can usually guage the approximate time of onset of haemorrhage from the history, this decision is almost impossible or erroneous in the minority.

(b) Course of Action During Haemorrhage.

The dilemmas to be expected are well portrayed in Cases 3 and 5. Case 3

was treated on conventional medical lines, i.e. with adequate blood transfusion and thoracentesis. Four pints of sanguineous fluid were aspirated in four hours, when a syncopal attack precluded further immediate thoracentesis. Half-an-hour later the patient died with terminal signs of cerebral anoxia, as reported in other patients (McMyn, 1947: Hansen, 1949). It is thus evident that medical treatment alone, however energetic, cannot be relied upon to rescue the patient with massive and uncontrolled haemorrhage. Retrospective consideration compels the belief that increasing tachycardia, despite a normal blood-pressure, warrants resort to immediate thoracotomy in such cases.

Experience gained from events in Case 3 led to timely surgical intervention in Case 5. Although there were certain similarities in the clinical pictures of these two patients, Case 5 had additionally two clear-cut phases of tension haemopneumothorax before the operation. As in Case 3, initial therapeutic thoracentesis was associated with incipient syncope after the aspiration of only two pints of fluid. One hour after this thoracentesis, i.e. two hours after his admission, he lapsed into a state of shock, manifest by a very feeble pulse and sustained moderate hypotension. Two hours later, urgent dyspnoea appeared abruptly, suggesting a tension haemopneumothorax. This was confirmed by the manometric readings and 1,350 cc. of air were withdrawn without producing any appreciable general improvement. The dyspnoea was, however, lessened and the tachycardia was slightly reduced. It was clear that haemorrhage had not abated.

About 20 hours after admission, alarming dyspnoea recurred and the pulse-rate had now risen to 160 per minute. Under-water seal drainage was promptly instituted to control the raised intrapleural pressure until the

commencement of the operation, three hours later. It was confirmed at operation that the haemorrhage had not ceased. Following effective haemostasis and drying of the pleural cavity, recuperation was rapid. It was concluded, on the basis of these observations that, if the blood-pressure fails to revert to normal after a few hours of adequate blood transfusion, i.e. 3 pints of blood in 4 hours, an emergency thoracotomy is obligatory, regardless of the absence of a marked tachycardia.

(c) Natural Cessation of Haemorrhage.

Discussion on this important phenomenon will be prefaced by the views of earlier authors. Head (1937), from a study of intrapleural haemorrhage, made the obvious remark that the stability of the blood-pressure and respiration indicate that haemorrhage has stopped. Nevertheless, it would be distinctly advantageous if there were some simple procedure or test, which would reliably indicate the natural arrest of haemorrhage in haemothorax, irrespective of the cause. Such views were clearly entertained by Courcoux (1932) when he declared that: "when the blood which one withdraws by thoracentesis coagulates readily in the syringe there is no doubt that the haemorrhage persists. It is then dangerous to continue the aspiration. When the blood withdrawn remains incoagulable in the syringe or test-tube, the haemorrhage has been arrested for at least 4 or 5 hours."

While this pronouncement is generally true, there are apparent exceptions. In each of the cases presented here, the "blood" from initial thoracentesis failed to clot in vitro, for at least one hour. This suggested that, in each case, haemorrhage had probably stopped at this early stage of management. However, in case 3, during the two thoracenteses in the last four hours before death, a few fine coagula formed in the aspirated fluid,

after it had stood for about fifteen minutes. At her autopsy, blood-clots were observed in the affected pleural cavity. At the operation of Case 5, oozing from a pleural adhesion was observed and blood-clots occupied the pleural cavity. During the initial aspirations, in cases 3 and 5, i.e. the patients who had persistent haemorrhage, the interpretation from the precepts of Courcoux was therefore misleading.

Investigations.

Concerning haematological observations, discussion will be limited to:-

a. the haemoglobin levels in the pleural fluid as compared with the concurrent values in the peripheral blood, and

b. the presence of eosinophilia in these fluids.

Little information is obtainable on these points in the literature.

Case No.	Hb. (%) Sahli		Time after onset of Haemorrhage (days)
	Pleural Fluid	Peripheral Blood	
2	59	89	5
	22	92	11
3	62	62	1
4	76	72	1
5	68 ¥	60 ¥	1
8	34 ¥	84 ¥	? 1

¥ Haldane Scale.

Inspection of the table shows that, during the first day, at least, the haemoglobin value in the pleural fluid tends to exceed slightly the level in the peripheral blood. Similar results have already been reported (Roderiquez Pastor and Arruza, 1941; Cosgriff, 1950; Cuningham, 1950).

In Cuningham's patient, with fatal idiopathic haemothorax, haemoglobin levels of 10.6 G per cent in the pleural fluid and 9 G per cent in the peripheral blood were obtained approximately 2 days after the onset of haemorrhage. Whilst one might predict equal haemoglobin values in these two fluids shortly after the onset of haemorrhage, it appears, perhaps as a result of commencing haemodilution of the peripheral blood, that the tendency is for an insignificantly higher value to occur in the pleural fluid. Detailed study of the changes in the haemoglobin and fibrinogen concentrations of these two fluids from cases of traumatic haemothorax were reported by Sellors (1945). His results for haemoglobin differ from those of the author. He concluded that, after the first few hours from the onset of haemorrhage, the haemoglobin level of the pleural fluid might be anything from 20 to 30 per cent below that of the circulating blood. The pleural fluid is diluted by the outpouring of fluid from the pleura in response to the irritant effect of the enclosed blood. Perhaps, this discrepancy of values is due to greater trauma to the pleura in chest injuries than in natural disease.

Transient eosinophilia is an occasional feature in patients with haemothorax. Concurrent eosinophilia in the pleural fluid and peripheral blood has previously been recorded three times (Troisier, et al., 1937; Rist and Worms, 1940; Groen and Godfried, 1948) in patients with idiopathic haemopneumothorax. In Case 2, on the fourth day after the onset of haemorrhage, the leucocyte count of the peripheral blood was 7,900 per cu. mm. and, in the pleural fluid, 3,600 per cu. mm., with values of 5 and 13 per cent respectively for eosinophils. The fourth patient had slight eosinophilia (6 per cent in a leucocyte count of 6,300 per cu. mm.,

3 days after the onset of haemorrhage. In Case 5, also, eosinophilia (16 per cent) was found in the peripheral blood on the fourth day after onset of haemorrhage. Moreover, this eosinophilia was not present two days previously, nor four days later. Unfortunately, there are no corresponding values available for the pleural fluid. This topic will be discussed later. Two noteworthy radiological observations will be mentioned. Firstly, Case I was observed to have cystic changes in the left mid-zone. Other observers (Hansen, 1949; Moser, 1951; Biancanala, 1953) have reported cystic lung lesions in patients with idiopathic haemopneumothorax. Secondly, in five of the patients (Cases I, 2, 4, 5 and 6) there was a subsequent complete radiological clearing on the affected side of the chest. This is attributed to early and energetic thoracentesis, with consequent short total periods of illness which, except for Case 2, did not exceed three weeks. Case 2, however, was considered to have a partially-clotted haemothorax, which required local enzymatic treatment. His total duration of illness was extended to just over two months. In Cases 7 and 8 minimal residual pleural thickening is still (March, 1956) radiologically apparent. The total duration of illness was 3 and 4 weeks respectively. However, the delayed recovery in Case 8 is attributed to a recurrence of haemopneumothorax, five days after admission. The validity of the claim that this was a recrudescence was based on clinical and radiological evidence and was further supported by the increased haemoglobin level (44 per cent) in the pleural fluid.

Treatment.

The aim of medical treatment is to convert the haemopneumothorax to a pneumothorax by early and repeated aspiration.

Many aspects of treatment have already been discussed and only the application of fibrinolytic-enzyme therapy will now be broached. In Case 2, three weeks after the onset of haemorrhage, a refractory phase, characterised by a stationary radiological picture coupled with constitutional upset, was manifest. Whilst a complicating empyema was considered, evidence was lacking. Rather, it was realised that neither the successful aspiration nor absorption of the pleural fluid could now be expected. As the risk of re-opening either the broncho-pleural fistula or the thrombosed blood-vessel was now negligible, enzyme-therapy was undertaken in preference to the more formidable procedure of open thoracotomy to evacuate blood-clot. Although the result of this fibrinolytic treatment was considered to be satisfactory, the temporarily increased general upset was distinctly distressing. This can be appreciated from the report of McGown (1954) that this treatment produced a severe general reaction in his patient, who declined further injections. Wider experience has shown that this phase of upset can be curtailed, without apparent sacrifice of success, by reducing the interval preceding the subsequent therapeutic aspiration to about 8 hours. A further similar treatment can be carried out on this short basis, thus fractionating the procedure. In Case 6, this treatment was briefly tried, but in view of the radiographic appearances of fibrin deposits, pulmonary decortication was preferred in order to expedite convalescence and early return to work.

SECTION 2.

Statistical Survey of Idiopathic Haemopneumothorax (including comparative statistics from published data on idiopathic pneumothorax).

CASE-INCLUSION.

The accompanying compilation (Table I, Section I) embraces the world-literature up to the end of 1955. Details on 240 cases of idiopathic haemopneumothorax are analytically presented. Similar recordings for the 12 cases of idiopathic haemothorax appear elsewhere (Section 3). The descriptions of 8 published cases of idiopathic haemopneumothorax (Szenes, 1929; Braco and Braco, 1935; Brewer, 1935; Repetto et al., 1936; May, 1937; Netto and Silveira, 1939; Montoro and Alderegina, 1945; Rusby, 1947) were unobtainable in the original and do not appear in this analysis. In addition, those patients in whom pulmonary tuberculosis was demonstrable, even although probably not directly causative, are relegated to a separate group (Section 3).

Moreover, patients who were shown to have a basic haemorrhagic disease, e.g. Granadiero's fatal case (1950) with haemophilia, have been omitted. Finally, there are 37 cases on record, as citations or brief commentaries, yet lacking the necessary evidence or detail for present inclusion (Amberson, 3 cases, 1935; Minor, 1935; Falla, 1938; Kirschner, 1939; Berliner, 5 cases, one fatal, 1940; Leach, 2 cases, 1945; Leahy, 1947; Rottenberg and Golden, 3 cases, one fatal, 1949; Arst's postscript, 3 cases, 1950; Hughes et al., 1951; Moxon, 1951; Briggs et al., 3 cases, 1953; Blan and Bernard, 1953; Dubose et al., 2 cases, 1953; Rapport et al., 1953; Shefts et al., 6 cases, 1954; Crowther, 1955; Laffont and Hofman, 1955).

Epidemiology.

The universality of this entity is readily apparent (Table I), since patients from each continent are on record. The fact that most cases have been reported by American and European authors should not unduly influence the observer in his interpretation of the geographical distribution.

Age.

The significant observation concerning the age of the patients is the distinctly selective range (14 - 52 years), with a mean of 28 years. Approximately 50 per cent of the cases belong to the third decade (Table 2), and 80 per cent to the third and fourth decades combined. These features parallel those in idiopathic pneumothorax (Table 2). Indeed, Hyde and Hyde (1948) drew attention to the fact, which still applies, that all series of idiopathic pneumothorax show an approximate 50 per cent incidence in the third decade. The corresponding mean ages, in published series of idiopathic pneumothorax, i.e. 27 (Myerson, 1948), 27.5 (Shefts et al., 1954), 28 (Niehaus, 1947; Hughes et al., 1951), 30 (Rottenberg and Golden, 1949), 32 (Dubose et al., 1953) show a remarkable constancy and are in accord with the value of 28 in idiopathic haemopneumothorax. Nevertheless, idiopathic pneumothorax differs slightly from this condition (Table 2), as it can, rarely, be a paediatric or geriatric problem.

Sex.

The infrequency of idiopathic haemopneumothorax in females has been repeatedly pointed out (Hopkins, 1937; Hartzell, 1942; Fry et al., 1955; Calvert and Smith, 1955). In all, 14 cases (6 per cent) are recorded (Table I), with an even distribution over the relevant decades. The corresponding incidence has been computed as 11 per cent for idiopathic

TABLE 2.

Comparison of age distribution in decades in the idiopathic types of pneumothorax, haemopneumothorax (Idio., Hpnx.) and haemothorax (Idio. Hx.).

IDIOPATHIC PNEUMOTHORAX								IDIO. ¥ HPNX.	IDIO. HX.
Decade	Kjaer-gaard (1932)	Perry (1939)	Myer-son (1948)	Hyde and Hyde (1948)	Myers (1954)	Crow-ther (1955)	TOTAL No. of Cases	The Entire Literature	
1st	1	3	1	0	0	0	5	0	0
2nd	5	11	7	4	13	5	45	22	2
3rd	20	33	17	30	71	14	185	123	4
4th	14	16	5	13	22	21	91	69	1
5th	9	11	5	10	5	11	51	18	4
6th	0	7	1	4	3	6	21	3	1
7th	0	4	0	2	1	4	11	0	0
TOTAL	49	85	36	63	115	61	409	235	12

¥ Not recorded in 5 patients.

pneumothorax (Table 3), while the group of 12 patients with idiopathic haemothorax (Table 13) contained only one female (Crimm, 1948). This common denominator, i.e. the marked male predominance, has never been satisfactorily explained. Certainly, this is not a characteristic of pulmonary tuberculosis. This statement does not, however, necessarily imply a non-tuberculous aetiology for these conditions.

Side of Thorax Involved.

This information was available in 225 of the patients with idiopathic haemopneumothorax and reveals a predilection (58 per cent) for involvement of the left hemithorax, (Table 3, C). This left-sided preponderance is statistically significant. Indeed, assuming equal probability, a one-sided deviation of the observed extent represents $2\frac{1}{2}$ times the expected standard deviation (σ), P ($> 2.5\ \sigma = 0.006$). This observation is at variance with recent reports (Fry et al., 1955; Calvert and Smith, 1955) of an approximate equivalence for the two sides. In the group of 12 patients with idiopathic haemothorax (Table 13), both sides were affected with equal frequency.

For patients with idiopathic pneumothorax it is demonstrated (Table 3) that careful attention to the sources of data is imperative. Table 3 shows the different results, which have been obtained from two main sub-groups. In Section A, which relates to the general population, it is observed that, of 590 patients with idiopathic pneumothorax, there is an exact equality in the incidence on the two sides. By contrast, in Section B, concerned almost entirely with male patients, of the 288 cases analysed separately, there is a predilection (56 per cent) for the right hemithorax. In summary, while there is a statistically significant predilection for left-sided haemopneumothorax, there is no difference in the frequency of involvement of the sides

TABLE 3.

Sex-Distribution and Side of Thorax affected in the Idiopathic types of Pneumothorax (A and B) and Haemopneumothorax (C).

	AUTHORS	No. of Cases	SEX		SIDE		
			Male	Female	Right	Left	Bilat.*
A	GENERAL POPULATION						
	Kjaergaard (1932)	49	34	15	33	15	1
	Perry (1939)	85	78	7	35	48	2
	Ornstein & Lercher (1942)	58	56	2	25	16	17
	Hyde & Hyde (1948)	36 *	30	6	See Later		
	Myerson (1948)	36	30	6	-	-	-
	Rottenberg & Golden (1949)	97	87	10	47	44	6
	Melrose (1950)	70	65	5	31	34	5
	Myers (1954)	115	98	17	42	64	9
	Shefts et al. (1954)	114	104	10	-	-	-
	Russell (1954)	100	95	5	45	52	3
	Crowther (1955)	61	50	11	37	22	2
	TOTAL	821	727	94	295	295	45
B	MALE POPULATION (Mainly)						
	Leach (1945)	126	126	-	76	50	-
	Schneider & Reissman (1945)	100	100	-	55	44	1
	Hyde & Hyde (1948)	27	27	-	* Plus 36 cases as above		
					31	32	-
	TOTAL	253	253	-	162	126	1
C	IDIOPATHIC HAEMOPNEUMOTHORAX (entire literature)	240	226	14	** 94	** 131	

* = Recurrent or simultaneous type.

** = Not recorded in 15 patients.

in the large representative group of idiopathic pneumothorax analysed. It is noted that the sub-group, Section B, is an unrepresentative sample.

Occupation.

The occupations of 150 of the 240 patients with idiopathic haemo-pneumothorax were clearly stated in the literature. These have been classified (Table 4) according to the scheme of Kjaergaard (1932) for idiopathic pneumothorax. Although the latter's table included only 50 patients, his analysis is reproduced for comparison with the present data on idiopathic haemopneumothorax. Similar information is set out for 11 of the 12 patients with idiopathic haemothorax. The classification adopted, the only one available with occupational groups for patients with idiopathic pneumothorax, is unfortunately difficult to operate. Thus, 11 soldiers have been included in the group undertaking heavy work, although it would have been helpful to know whether any of these were army clerks, medical orderlies, batmen, etc.. Similarly, some of the patients classified under light employment, e.g. businessmen, hotel and shop-managers, might have been more correctly categorised under sedentary occupation. Again, for present purposes, drivers of vehicles (taxis, buses, and trucks) have been included under sedentary, and not light, employment.

Inspection of Table 4 permits the safe conclusion that there are three prominent types of employment, i.e. clerks, students and soldiers, in the occupational history of idiopathic haemopneumothorax. This recalls the earlier concept of "pneumothorax of conscripts" (Galliard, 1896). Furthermore, there is a general similarity in the occupational distribution in the idiopathic types of both pneumothorax and

TABLE 4.

Occupational groups of patients with idiopathic types of haemopneumothorax and haemothorax, compared with those of Kjaergaard's patients (1932) with idiopathic pneumothorax.

HEAVY	MODERATE	LIGHT	SEDENTARY	STUDENT	HOUSEWORK	TOTAL
IDIOPATHIC PNEUMOTHORAX						
13 (26%)	8 (16%)	6 (12%)	7 (14%)	6 (12%)	10 (20%)	50
IDIOPATHIC HAEMOPNEUMOTHORAX						
34 (23%) Includes:- 11 soldiers 2 sailors	26 (17%)	29(19%)	36 (24%) Includes: 23 clerks 2 book-keepers 8 drivers	18 (12%)	7 (5%)	150
IDIOPATHIC HAEMOTHORAX						
3	1	3	2	1	1	11

haemopneumothorax. Indeed, in both conditions, the social stratification is exclusively that of the working-class and middle-class.

Action at Onset.

The diverse activities, which immediately preceded or coincided with the onset of symptoms are listed in Table 5. Relevant information was lucidly available in only 138 of the 240 patients with idiopathic haemopneumothorax. For ease of interpretation, two strongly contrasting groups are shown in parallel, i.e. 67 patients engaged in minimal or ordinary activity and, secondly, 71 patients representing the "exertion" group. In each group, an arbitrary and, indeed, somewhat itemised sub-division into specific types of movement has been used.

The inherent difficulties in classification, although not obvious from the table, invite clarification. Three patients, included in the sub-group of "reaching, etc.," were undertaking such normal activities as turning in a chair, tying a shoe-lace, and taking a coat off respectively. Conversely, it might be argued that eating and shaving both involve stretching. Again, in the "exertion" group there were four patients who had clearly completed their strenuous exercise, i.e. playing a clarinet, military parade, and baseball (2 patients) respectively, when symptoms appeared.

The permissible conclusions are, however, so evident that their validity may go unchallenged. In contrast to the sub-group of 23 patients who had been undertaking fairly violent exercise, a slightly smaller sub-group of 18, not including those resting in bed, were in a state of physical rest. While 6 patients were stated to be rising from bed,

TABLE 5.

Action at, or just before, onset of idiopathic haemopneumothorax.

MINIMAL ACTIVITY	NO.	EXERTION	NO.
"No exertion"	18	Work, sport, gymnastics and weight lifting	23
In bed (often asleep)	16	Rising from bed	6
Walking	10	Walking uphill, upstairs, etc.	9
Sitting	12	Getting on or off bus, train, etc. Arising from seat.	4
Eating	5	Reaching, stretching, bending or turning	12
Standing	3	Running	1
Talking	1	Lifting	3
Shaving	2	Pulling or pushing	2
TOTAL	67	Sneezing, coughing, vomiting	3
TOTAL for both groups tabulated = 138 Patients		Washing dishes or cleaning	3
		Driving a vehicle	4
		Playing wind instrument	1
		TOTAL	71

16 were actually in bed when symptoms began. A similar comparison is made between 9 patients walking under strain and 10 patients merely "walking." Again, while 4 patients had presumably been recently sitting, 12 were described as "sitting," usually at home, in an office, cinema, bus or train. Finally, it is worthy of comment that 5 patients were actually having a meal, while another was engaged in conversation at the onset of symptoms.

When these so-called "inciting" causes are compared with those described for idiopathic pneumothorax, the analogy is striking. Among the numerous relevant conclusions from the study of patients with idiopathic pneumothorax, only a few will be briefly cited. For instance, Briggs and colleagues (1953) contended that sleep is a more frequent concomitant than is strenuous exercise. Indeed, their incidence of strenuous exercise was unusually low, i.e. 2 out of 85 patients, and of the same order as reported by Hyde and Hyde (1948), and in contrast to a 30 per cent incidence recorded by Schneider and Reissman (1945). Collective experience, however, is well summarised by Hughes and associates (1951), who stated that "in many patients the onset occurred while the patient was at rest, either sitting in a chair or a car, or during sleep."

Previous Respiratory Disease.

The paramount importance of an accurate record of previous pulmonary infections to the study of the aetio-pathogenesis of "idiopathic" haemo-pneumothorax needs no emphasis. Special entries on this point were found in only 139 of the 240 case-legends. Among these 139 cases, there was "positive" information in approximately 30 instances and it is surmised that the results of specific inquiry into childhood-illnesses, which could be

complicated by broncho-pneumonia, has not been explored.

There are, however, reasons why these data may still be incomplete.

(1) The patients are adults of recent good health so that their infantile and adolescent-illnesses may well have been forgotten or never fully known.

(2) Some primarily non-pulmonary diseases, e.g. certain exanthemata, may cause ill-recognised concurrent respiratory disease.

(3) The approximate incidence of primary tuberculous infection is difficult to assess, since the patients are of global distribution. However, mention will be made, later in this Section, of the results of standard tuberculin-tests.

The inherent difficulty and obvious inaccuracy in the retrospective determination of the frequency of earlier pulmonary infection are well illustrated in the observations of Leopold and Lieberman (1935). These investigators revealed that, at autopsy, some 50 per cent of adults over the age of twenty had pleural adhesions, despite no clear history of lung-infection.

Scrutiny of the available data reveals certain symptoms, which are possibly non-contributory, for present purposes, e.g. unexplained and isolated prolonged cough (Louria, case 2, 1938; Jennings, 1941), upper respiratory tract infection of brief duration, and repetitive attacks of coryza (Hurxthal, 1928; Milhorat, 1931). Brief attacks of chest-pain (Louria, case 4, 1938; Lorge, 1940; Raimondi and Boffi, 1943; Grimaldi, 1951; Ross, case 6, 1952; Author's cases 7 and 8), unaccompanied by any constitutional upset, provide grounds for speculation. Alternative explanations of "dry pleurisy" (Terry, 1935) or spontaneous pneumothorax (Hopkins, 1937) have been advanced. It is the author's belief that most of

such episodes were sub-clinical idiopathic pneumothoraces, a view which may perhaps be extended to some of the patients, cited in the next paragraph, with antecedent "pleurisy."

There remain 18 cases in which there was a history of "pleurisy" (Jones and Gilbert, 1936; Louria's case 1, 1938; Eidinger and Rubin's case 2, 1952; Author's cases 4 and 7), pneumonia (Holden's case 1, 1935; Frey, 1935; Rossel, 1935; Jones and Gilbert's case, again, 1936; Moser, 3 cases, 1951; Hyde and Hyde, case 8, 1951; Eidinger and Rubin's case 3, 1952; Stensrud, 1953), chronic bronchitis (Housden and Piggot, 1931; Aquilar and Ferradas, case 2, 1935; Eidinger and Rubin's case 1, 1952; Author's case 7, again), or bronchiectasis (Jacobs, 1936). The last two conditions are mentioned as they might predispose to occasional broncho-pneumonic attacks. Finally, it is apt of mention that, unexplained brief recent episodes of haemoptysis (Szenes, 1929; Hartzell's case 3, 1942; Groen and Godfried, 1948; Hyde and Hyde, case 4, 1951) have been reported.

It appears, therefore, that the incidence of antecedent pulmonary infection, excluding primary tuberculosis, in patients with idiopathic haemopneumothorax is about 10 - 20 per cent. This low incidence does not exceed that obtaining in hospital-patients of the same age and sex distribution. The fact that no correlation has been revealed between antecedent, non-tuberculous, pulmonary infection and idiopathic haemopneumothorax is, for reasons already discussed, to be interpreted with reserve. There is no comparable figure readily available from detailed studies of idiopathic pneumothorax.

Family History of Tuberculosis.

Special mention of the family-history, on this point, is available in

only 84 case-reports. Definite past or concurrent pulmonary tuberculosis in a member of the family is reported in 10 patients (12 per cent). This incidence closely corresponds to that (16 per cent) recorded by Kjaergaard (1932) for idiopathic pneumothorax. These frequencies do not appear to have any special significance.

Results of Tuberculin-Tests.

The results of Mantoux or Von Pirquet tests are recorded for 30 of the patients with idiopathic haemopneumothorax. It is noteworthy that the results were negative in 18 (60 per cent) of this small group of patients. Similar findings characterise idiopathic pneumothorax. For example, Kjaergaard (1932) reported negative reactions in 9 out of 21 patients; Leggett and colleagues (1934) in 8 of 15 cases; Myers (1954) in 71 or 112 cases, so tested. However, to be strictly accurate, in some of these instances in the idiopathic types of both haemopneumothorax and pneumothorax, the full conventional range of strengths of old tuberculin was not employed. It can be concluded that in about 50 per cent of patients with the idiopathic types of either haemopneumothorax or pneumothorax, there is weighty evidence, from these results, against a tuberculous aetiology.

Factors related to Prognosis.

1. Mortality-Rate. Table I reveals 19 deaths in the entire group of 240 patients, i.e. an 8 per cent fatality-rate, for idiopathic haemopneumothorax. This calculation does not include the only two other fatal cases (Berliner, 1941; Rottenberg and Golden, 1949) in the 45 patients cited but not analysed, either because the reports were not seen in the original or lacked detailed information. The present computed mortality-rate

is slightly less than the two recently reported rates, i.e. 12 per cent (Fry et al., 1955) and 15 per cent (Calvert and Smith, 1955). Neither of these two groups of authors discriminated between the idiopathic types of haemopneumothorax and haemothorax, for which a revised combined mortality-rate of 9 per cent is derived from the data analysed here. As shown in Table 13, there were 4 deaths in the group of 12 patients with idiopathic haemothorax.

2. Morbidity-Rate. At this juncture, the subject of morbidity will only be briefly mentioned as it will be fully discussed in Section 4, under the heading of complications.

(a) Duration of Illness.

The duration of illness was clearly indicated for 190 of the 221 patients (Table I), who recovered. The mean duration of illness was 49.7 days, i.e. 7 weeks. However, the duration was prolonged to 3 months or more in 26 patients (14 per cent); to 4 months or more in 19 cases (10.5 per cent); and to 5 months or more in 9 patients (5 per cent).

(b) Incidence of Complications.

Only three complications, i.e. fibrothorax, empyema and calcification of the pleura will be considered. Tables 6a and 6b show that 45 (20 per cent) of the 221 patients who recovered had fibrothorax, recognised radiologically, if not always clinically obvious. However, 30 of these 45 patients were subjected to pulmonary decortication, without any mortality, and with, almost invariably, subsequent improvement.

TABLES 6 a and b.

Complications of Idiopathic Haemopneumothorax Fibrothorax (including organising haemothorax and well-marked pachypleuritis).

a. Patients not subjected to Pulmonary Decortication (15 cases).

YEAR	AUTHORS	YEAR	AUTHORS
1917	Williamson	1941	Arroya
1928	Doria	1942	Tannenbaum
1934	Bellon	1943	Raimondi and Coffi
	Patino-Mayer and Patero	1946	Schneider and Reissman
1936	Wilson, Case I	1948	Orsi, Case 2
	Poli	1950	Leston and Pilheu, Case I
	Jones and Gilbert	1951	Grimaldi
1938	Egues, Case I	TOTAL	15 Patients

b. Patients Subjected to Pulmonary Decortication (30 cases).

1947	Leahy ¥	1952	Fusia and Cook, Case 2
	Harrington & Frelick		Eidinger and Rubin, Case 3
1948	Elrod and Murphy		Ross, 3 Cases
1949	Kelly		Kiekens
	Wright et al.	1953	Borrie, Case 3
	Nario		Biancanala
1950	Williams	1954	Mezzera and Aquiar
	Arst et al. - Postscript ¥		McGown
1951	Seley and Neuhof		Shefts et al., Case 6. ¥
	Carrollet al., 2 Cases	1955	Fry et al., Cases 1, 3 and 6
	Hyde and Hyde Case 8		Weiner
1952	Deiss et al., Case 2	1955	Author's Case 6
	Beatty and Frelick, Case 2	TOTAL	30 Cases

¥ Not included in full analysis.

Among the 221 patients who recovered, 8 developed empyema (Munos Monatorio, 1936; Korol, case 1, 1936; Stockler, case 4, 1947; Kastl, case 2, 1952; Garcia et al., cases 1 and 2, 1953; Mezzera and Aquiar, 1954; Agnew, 1955). Four of these patients (the last 4 mentioned) were also included in the group with fibrothorax. These 4 patients, and the patient of Kastl, all underwent pulmonary decortication, with excellent post-operative results.

Two other patients (Milhorat, 1937; McRae, 1939) developed the radiological appearances of calcification of the pleura, which were pronounced after 6 and 10 years respectively from the onset of the haemopneumothorax.

3. Surveillance concerning Tuberculosis.

Scant information is recorded on this point. However, two series (Hyde and Hyde, 1951; J.A.Myers, 1954) aid considerably by their size and the duration of the period of study. For instance, Hyde and Hyde collected 12 cases of idiopathic haemopneumothorax from a combined study, in two hospitals, extending over 12 years; while Myers reported briefly 9 cases from a 24-year survey.

Table 7 lists the 40 patients, whose surveillance was for periods of not less than one year. In 10 instances the period was only one year, while the longest was 23 years, i.e. Case I of Myers. Curiously, this patient alone, of the 40, developed pulmonary tuberculosis. Following uneventful recovery from the haemopneumothorax, moderately advanced tuberculosis of the opposite lung was discovered 5 years later. He again soon recovered. In summary, an incidence of 2.5 per cent of subsequent pulmonary tuberculosis is derived from the available limited data on idiopathic haemopneumothorax.

TABLE 7.

Clinical and Radiological Surveillance for Tuberculosis.

(40 Patients).

YEAR	AUTHORS		NO. OF YEARS
1931	Palmer and Taft, Case I		2
1932	Kjaergaard,	Case I	4
		Case 2	$4\frac{1}{2}$
1935	Catuogno, Case 2		2
1937	Hopkins, Case I		2
	Rist		2
	Milhorat C.P.		$6\frac{1}{2}$
1938	Louria, Case I		5
1942	Hartzell, Case 3		6
1945	Schneider & Reissman F.		2
1950	Cuningham, Case 2		1
	Deiss et al.,	Case 1	1
		Case 2	1
1951	Hyde & Hyde,	Case I	6
		Case 2	8
		Case 3	1
		Case 5	1
		Case 6	1
		Case 7	1
	T.P.	Case 8	2
		Case 9	8
		Case 10	3

YEAR	AUTHORS		NO. OF YEARS
1952	Beatty & Frelick, Case 3		2
1953	Garcia et al., Case I		2
	Huss		$1\frac{1}{2}$
	Biancanala		1
	Eidinger & Rubin Case I		4
1954	Mezzera & Aguiar		1
	Myers T.P.	Case I	23 ✱
		Case 2	18
		Case 3	12
	T.P.	Case 4	12
		Case 5	9
		Case 6	5
		Case 7	5
	T.P.	Case 8	4
		Case 9	2
1956	Author	Case I	$3\frac{1}{2}$
		Case 2	3
		Case 4	1

✱ Developed pulmonary tuberculosis after first 5yrs.

C.P. = calcified pleura
T.P. = thickened pleura
F. = fibrothorax

The literature on idiopathic pneumothorax, however, is replete with corresponding studies. A low incidence of 2 per cent is obtained from just over 800 unselected, collective patients, whose surveillance was for periods of 1 to 10 years (Kjaergaard, 1932; Morris, 1934; Perry, 1939; Kirschner, 1939; Ornstein and Lercher, 1942; Schneider and Reissman, 1945; Niehaus, 1947; Hyde and Hyde, 1948; Rottenberg and Golden, 1949; Melrose, 1950; Russell, 1952; Dubose et al., 1953; Myers, 1954; Crowther, 1955). This calculated incidence substantially confirms the reliability of the earlier computations of 2.4 per cent (Perry, 1939) and 3 per cent (Draper, 1948). Closer analysis of these data reveals no significant tendency to the development to early or late subsequent tuberculosis in patients with idiopathic pneumothorax. The same conclusion applies equally to idiopathic haemopneumothorax, in respect of early subsequent tuberculosis, while a low incidence of late tuberculosis would be anticipated from the present limited date.

4. Recurrence-rate as Idiopathic Pneumothorax.

Genuine recurrence, after several years, of ipsilateral idiopathic haemopneumothorax is quite unique (Rusby, 1947). Rusby (personal communication) stated that an interval of 5 years separated the episodes, in contrast to the patients of Repetti (1940) and Waring (1945) for whom the corresponding intervals were merely a few weeks. The rarity of this event is attributed to the irritant effect of intrapleural blood, which tends to promote pleural symphysis. Indeed, Rothkopf (1939) first advocated the intrapleural introduction of blood for the prophylaxis of recurrent pneumothorax.

TABLE 8.

Antecedent and Subsequent Idiopathic Pneumothoraces (Id. Pnx.) in Patients with Idiopathic Haemopneumothorax (Id. Hpnx.).

YEAR	AUTHORS		Id. Hpnx.	Id. Pnx.		
			Side	Side	Interval	
ANTECEDENT						
1937	Hees		L.	R.	1 yr.	
1942	Hartzell	Case 2	L.	R.	9 mths.	
1949	Hansen	Case 2	R.	R.	6 mths.	
1950	Deucher	Case I	R.	R.	6 mths.	
		Case 2	L.	R.(2x)	a. 5 yrs. b. 5 mths.	
1951	Hyde & Hyde	Case 12	L.	R.	2 yrs.	
1954	Shefts et al.		L.	R.	Unstated	
SUBSEQUENT						
1931	Palmer & Taft	Case I	L.	R.	2 yrs.	
1936	Rossel		L.	R.	5 days	¥
1937	Rist		R.	R.	2 yrs.	
1944	Goldman & Roth		R.	R.	6 wks.	
1951	Hyde & Hyde	Case 9	L.	R.(4x)	in 10 yrs.	
1952	Fusia & Cook	Case I	L.	R.	1 mth.	
1953	Eidinger & Rubin	Case I	L.	R.	6 mths.	
	Huss		L.	R.	6 mths.	
	Grundi		R.	L.	6 wks.	¥
1954	Myers	Case 5	R.	Un-stated	2 yrs.	
	Shefts et al.		R.	L.	Unstated	

¥ = Fatality from bilateral pneumothorax, with haemothorax.

Bilateral simultaneous or successive idiopathic haemopneumothorax has not been reported.

Table 8 indicates the recorded instances of subsequent pneumothoraces in patients with idiopathic haemopneumothorax. For the convenience of later discussion on the inter-relation of the idiopathic types of pneumothorax and haemopneumothorax, authenticated antecedent pneumothoraces in the latter condition are set out in the top-section of this table.

This limited information on recurrences as pneumothorax (Table 8) provides no indication of true incidence. The reason is that most authors have been essentially interested in reporting recently observed cases (often solitary), so that little or no information on follow-up was provided. Even in the group of 40 patients (Table 7), just discussed, whose periods of surveillance all exceeded one year, specific mention of the presence or absence of recurrences as pneumothorax was frequently omitted.

Nevertheless, in 4 patients (Palmer and Taft, Case I, 1931; Hyde and Hyde, Case 9, 1948; Eidinger and Rubin, Case I, 1953; Myers, Case 5, 1954) of this unselected group, recurrence as pneumothorax is specifically recorded. This implies a probable minimal incidence of 10 per cent.

It is tempting to make further limited comment on the information provided on Table 8. Eleven instances of recurrence as pneumothorax are recorded. Apart from 1 patient (Shefts et al., 1954) where the interval is unstated, the recurrence as pneumothorax, in the other 10 cases, was within a period of about two years of the haemopneumothorax. Indeed, in 7 instances, it was within about six months of this event. The side of recurrence, unrecorded in one case, was, with 2 exceptions, contralateral.

This observation harmonises with the theoretical prediction of improbability of ipsilateral recurrences. Moreover, it contrasts with the corresponding events in idiopathic pneumothorax.

For the latter, there is unanimity (Hyde and Hyde, 1948; Draper, 1948; Rottenberg and Golden, 1949; Melrose, 1950; Russell, 1952; Dubose et al., 1953; Myers, 1954) that, provided the follow-up period exceeds one year:

(1) recurrences are mainly ipsilateral.

(2) recurrences, as apparently also in idiopathic haemopneumothorax, are commonest within one year of the initial episode.

(3) the recurrence-rate is 15 to 25 per cent.

Any attempted comparison of the recurrence-rates in the idiopathic types of pneumothorax and haemopneumothorax must be confined strictly to contralateral episodes, in view of the local circumstances obviating a past-haemopneumothoracic pneumothorax. For idiopathic pneumothorax, the contralateral recurrence-rate is certainly less than 5 per cent, while the corresponding figure for idiopathic haemopneumothorax appears to approximate 8 per cent. It is interesting to note that no recurrences as pneumothorax have been recorded among the 8 non-fatal patients with idiopathic haemothorax.

SECTION 3.

Pathogenesis and Aetiology.

The aetio-pathogenesis of idiopathic haemopneumothorax will be discussed under several headings:-

(1) Observations
- a. at autopsy.
- b. at emergency thoracotomy.
- c. at pulmonary decortication.
- d. at thoracoscopy.
- e. from radiography.

(2) Relation to
- a. idiopathic pneumothorax.
- b. tuberculosis and the tuberculous type of haemopneumothorax.
- c. idiopathic haemothorax.
- d. haemothorax during artifical pneumothorax.

(3) Sources of intrapleural haemorrhage.

1a. Observations at Autopsy.

The full results of autopsy are available for 15 patients with idiopathic haemopneumothorax (Table 9). Apart from apical scarring which was noted in 7 of these cases, bullae alone were observed in 6 patients and adhesions alone in only 3 cases. In a further 3 patients, bullae and adhesions co-existed, with actual attachment in two patients (Pitt, 1900; Housden and Piggot, 1931). The 3 remaining patients exhibited none of these lesions.

Two mechanisms of production of the haemopneumothorax can be readily discerned. Firstly, in 3 patients, the escape of both air and blood can be attributed solely to ruptured bullae. Hansen's fatal case with underlying cystic disease of the lung, although not included in the table, might also belong to this category. Secondly, in 2 patients, ruptured bullae appeared to have produced the pneumothorax, while

TABLE 9 OBSERVATIONS AT AUTOPSY IN IDIOPATHIC HAEMOPNEUMOTHORAX.

Authors	Age of Pt.	Duration of Illness (days)	Chest X-ray	Aspiration of Blood	Blood Transfusion	Vol. of Blood in Pleural Cavity	Apical Scars	Bullae	Adhesions	Comments on Pathology
1. Pitt (1900)	18	2	0	+	0	4½ L. fluid	0	One Apical	+	Ruptured adhesion attached to apical bulla.
2. Rolleston(1900)	21	8	0	+	0	2 L. fl. Few clots	0	0	0	No bleeding point found.
3. Fisher (1922)	22	7	0	+	0	4½ L. Clotted	+	Sev.Bilat. Apical	0	Fresh blood oozed from torn bulla.
4. Kaier (1923)	30	3	0	+	0	Not stated	0	0	0	No lesion in lung or pleura.
5. Housden and Piggot (1931)	44	4	0	0	0	1 L. Clotted	+	2	+	Tough adhesions torn from chest wall.
6. Tait and Wakely (1935)	32	8	0	+	0	2½ pints fluid	0	12 near apex	0	Torn bulla filled with fresh blood.
7. Rossel (1935)	20	9	+	+	+	3½ L. fluid	0	Sev.Bilat.	0	Bilat. pneumothorax. L. haemothorax. No bleeding point found.
8. Jones and Gilbert (1936)	23	38	+	+	+	4 L. fl. free fibrin ¾ L.	Opp. Apex	1	0	Bulla intact. Bleeding point not found.
9. Louria (1938)	22	4	0	+	+	Not stated	+	1 same side. Sev.opp.side	+	Torn apical bulla. Bleeding point not found.
10. Beaumont(1938)	28	21	+	+	+	2 pints fluid	0	0	0	No lesion in lung or pleura.
11. Lorge (1940)	34	1	+	+	0	3 L. fl. Few clots	Bil.	Few	0	No bleeding point found.
12. Helwig and Schmidt(1947)	30	1	+	+	+	2 L. fl.	+	0	+	3 cord-like bands torn from parietal pleura.
13. McMyn (1947)	28	7	+	+	+	Not stated	0	Sev. apical & lateral	0	Bleeding probably from bulla
14. Arst et al. (1950)	22	4	+	+	+	¼ L. Clot. ⅓ pt. fl.	+	0	+	Bleeding from torn adhesion.
15. Calvert and Smith (1955)	40	2	+	+	+	2½ pts. A few clots	0	0	+	Torn apical adhesion had caused haemorrhage.

subsequent haemorrhage was considered to arise from stretched and torn adhesions.

In 4 further patients, one or other of these two mechanisms appeared probable but no exact opinion can be expressed. Three of these cases had intact bullae but no demonstrable adhesions and so the first mechanism would appear to be the probable explanation. In the other patient, a torn bulla was accompanied by a ruptured adhesion, and in this case the second mechanism is favoured.

There remains a third sub-group of 3 patients, with adhesions unaccompanied by bullae, in whom the haemorrhage but not the pneumothorax is explicable. In these patients, the pneumothorax might also have been due to ruptured adhesions, but the mechanism is obscure. However, the latter lesions have been considered (Rusby, 1952; Myers, 1954) as the sole cause of certain cases of spontaneous pneumothorax. A fourth sub-group consists of 3 patients with occult lesions. Finally, it is important to point out that actual oozing of blood was witnessed at autopsy in only one of the whole group of patients. The interpretation as to the source of haemorrhage has been based on the circumstantial evidence of torn adhesions or of clotted blood occupying a ruptured bulla.

As a convenient comparison, the observations of the autopsies of the 5 patients with fatal idiopathic haemothorax (Table 10) will now receive comment. Apart from apical scarring on the affected side in 3 patients, adhesions were visible on this side in 4 cases, and bullae in 3 patients. Except for 1 patient (Davidson, case 1), where lesions were restricted to the opposite side, the rest of the patients had clearly evident torn adhesions to explain the haemorrhage.

TABLE 10

OBSERVATIONS AT AUTOPSY IN IDIOPATHIC HAEMOTHORAX.

Authors	Age of Patient	Duration of Illness (Days)	Chest X-ray	Aspiration of Blood	Blood Transfusion	Vol. of Blood in Pleural Cavity	Apical Scars	Bullae	Adhesions	Comments on Pathology
Davidson (1935) Case 1	28	21	0	+	+	4 L. fluid	Opp. Side	0	Opp. apex only	No pulmonary or pleural lesion on affected side.
Case 2	26	$1\frac{1}{4}$	0	0	0	3 L. fluid	0	Sev. apical	Both apices	Many adhesions between pericardium and lung. Highly vascular.
Davidson and Simpson (1940)	* 26	$1\frac{1}{2}$	0	+	+	$2\frac{1}{2}$ L. fluid. Clots also.	+	1	+	Torn apical bulla. Torn vascular basal adhesion.
Cuningham (1950) Case 1	17	$1\frac{3}{4}$	0	+	0	$3\frac{1}{2}$ L. fluid. Clots also.	Bilat.	0	+	Torn apical adhesion as considered source of haemorrhage.
Irwin (1951)	45	9/24	0	0	0	$2\frac{1}{2}$ L. fluid	+	4 at apex	+	Torn apical adhesion (attached to a bulla) as considered source of haemorrhage

* Strictly speaking this is a case of haemopneumothorax, and included as such in the main analysis. It is included here since the authors referred to it as idiopathic haemothorax.

There is, therefore, a general similarity in the autopsy findings in the idiopathic types of haemopneumothorax and haemothorax. In both conditions, apical scarring, bullae (one of which was torn in a patient with idiopathic haemothorax), and pleural adhesions (often torn), are the standard lesions. Moreover, two patients in each group had the complete triad of basic lesions. Adhesions, constantly present in the small group with idiopathic haemothorax, were recorded in 6 of the 15 patients with idiopathic haemopneumothorax.

The occasional bilateral distribution of these lesions is also noteworthy. Bilateral apical bullae occurred in 3 patients with idiopathic haemopneumothorax; bilateral adhesions, in 1 patient with idiopathic haemothorax; and bilateral apical scarring in one patient of each group. It seems probable, therefore, that these two clinical conditions have an identical aetiology which, in view of the apical location of the scarring, bullae and adhesions, must have an affinity for lung-apices, both of which may be affected. This subject, however, must await fuller discussion.

1b. Observations at Emergency Thoracotomy.

The operative findings in the 19 patients, who underwent operation for uncontrolled haemorrhage in idiopathic haemopneumothorax, are set out in Table 11. The source of bleeding was established in all but 2 patients. In 15 patients, it was found to be torn adhesions, usually apical. However, in 2 patients, without demonstrable adhesions, the haemorrhage arose from ruptured bullae. Nine of the patients had bullae also, usually apical, one of which was ruptured in each of 5 patients.

As to interpretation in this group, the same mechanisms for the production of haemopneumothorax as described for the autopsied patients

TABLE 11 OBSERVATIONS AT EMERGENCY THORACOTOMY.

Authors (years)	Interval from onset to operation(hrs.)	Source of Haemorrhage	Additional Pathology	Estimated Total Blood-loss (cc.)	Interval from operation to discharge(dys.)
Myers et al. (1951)	60	Apical adhesion	-	4,410	11
Ross,C.A. (1952)	Unrecorded Probably 36	Apical adhesion	Bullae	"large amount"	Not noted
Holloway et al. (1952)	52	Apical adhesion not bleeding	-	6,600	60
Beatty and Frelick (1952)	72	Adhesions at apex & costophrenic sinus	Not recorded	2,700 (clots also)	10
	40	Apical adhesion	Apical bullae	1,500	10
	< 24	Apex of pleura (?adhesion)	Apical bullae	2,400 (clots also)	12
Borrie (1953)	Unrecorded Probably 30	Apical adhesion	Apical bullae. One ruptured	4,000 (clots also)	15
	7	Torn apical bulla	-	1,100 (clots also)	14
Ross et al. (1953)	96	Apical adhesions	Apical bullae. One ruptured	5,000	8
Williams et al. (1954)	120	Apical adhesion	Apical bullae	3,975	11
Fry et al. (1955)	-	Adhesion	Ruptured bulla	5,070	-
	-	Apical adhesion	Ruptured bulla	-	-
	-	Bleeding point on chest-wall	-	-	-
	-	-	-	5,750	-
	-	Torn bulla. Bleeding	-	-	-
	-	Adhesion	Ruptured bulla	3,500	-
Clyne & Hutter (1955)	24	Not found No adhesions	-	2,280 (inc. clot)	28
	144	Adhesions	-	2,825 (clots also)	13
Present Author	96	Apical adhesion	-	3,500 (inc. clots)	13

All patients survived and all had pre-operative blood transfusion and thoracentesis.

are again apparent. In 2 patients of the present group, the pathogenesis was attributed to ruptured bullae alone, while in 5 patients certainly, and 9 possibly, the combination of torn bullae and ruptured adhesions was the considered explanation. In 1 further patient, who had a torn adhesion, the mechanism remains equivocal since there is no record as to the presence of bullae. There remains a third sub-group, of 5 patients, in whom torn adhesions alone were found, and a fourth sub-group, of 2 patients, without any evident lesion.

The main difference between this group and patients examined at autopsy, is the frequency of observed adhesions. In the present group, these were noted in 15 of the 19, compared with an incidence of 6 in the 15 fatal cases. In those patients who were examined at autopsy, the source of haemorrhage was ill-defined. By contrast, in the patients who were subjected to operation for unremitting haemorrhage, the source was usually observed to be the parietal end of a severed pleural adhesion. On the other hand, from the aetiological aspect, information is lacking on the incidence of apical scarring and of bilateral lesions in this group.

1c. Observations at Pulmonary Decortication.

Pulmonary decortication, according to the literature, has now been performed on 35 patients with idiopathic haemopneumothorax. Observations during this operation have contributed little to the advancement of knowledge of the basic lesions. Indeed, mention of the presence of bullae, adhesions, or apical scarring is conspicuous by its absence. There are, however, two main reasons. Firstly, many reports are mainly concerned with technique or the results obtained. Secondly, the operation has usually been undertaken weeks or months (rarely, years)

after the onset of the haemopneumothorax. During this time the lung has become coated with a peel of fibrin and its anatomical surface cannot be inspected. Since this operation is seldom performed within three weeks of the onset of haemorrhage, signs of the source of haemorrhage are not to be expected.

There are, however, three reports (Deiss et al., 1950; Kastl, 1952; Fusia and Cook, 1952) which make special mention of the pleural and pulmonary lesions observed per-operatively. In these reports, the co-existence of bullae and adhesions is noteworthy. For instance, Deiss and colleagues report that, at the operation on their second patient, five weeks after the onset of haemorrhage, there were two apical and one lateral bullae as well as basal and lateral adhesions. Kastl's patient developed a loculated empyema, which required decortication one month after the onset of haemorrhage. At operation, several small apical blebs and many adhesions were observed. Fusia and Cook's second patient underwent pulmonary decortication three weeks after the onset of symptoms. At operation, six small apical bullae, one of which was torn, and many basal adhesions were detected. These basal adhesions, mentioned in 2 of these 3 patients, and in the fatal case of Davidson and Simpson (1940) are not commonly observed in idiopathic haemopneumothorax and should not be confused with the basal adhesions appearing later during its complication, organising haemothorax.

In 3 other patients (Borrie, 1953; Garcia et al., 1953; Agnew, 1955), pleural adhesions were discovered at operation. Moreover, in both of these patients of Borrie and Garcia, an earlier radiograph had revealed adhesions on the affected side. Garcia and associates concluded

that the torn adhesions, seen at the operation of their patient, were those which had been radiologically apparent. However, in Borrie's patient the adhesions which were seen per-operatively were fibrinous, not fibrous. In all the other patients mentioned here, however, it seems fairly certain that the adhesions seen at operation were not newly formed as a sequel to the haemopneumothorax. Any attempt to reconstruct the mode of production of the haemopneumothoraces in this group would be too speculative to be valuable. Nevertheless, the basic lesions described above are similar to those described in the two previous groups of patients.

1d. Observations at Thoracoscopy.

Thoracoscopy has not been widely practiced in idiopathic haemopneumothorax. Its limitations will be discussed later. However, this procedure has occasionally provided useful information in this condition (Cardenas et al., 1937; Birch, 1938; Hansen, case 3, 1949; Sattler, 1954). Thus, Cardenas and colleagues demonstrated, during deep respiratory movements of their patient, the point of perforation in the lung. They visualised a batch of fine bubbles, associated with oozing of blood. No adhesions were demonstrable. This direct evidence of haemorrhage from the lung surface corroborated the view of Castex and Mazzei (1936), that haemorrhage in haemopneumothorax is not always due to ruptured vascular adhesions. Birch succeeded in inspecting several adhesions, one of which was covered with blood-clot, while Hansen observed unexpectedly widespread cystic disease of the lung. Sattler identified and cauterised a bleeding adhesion. His observations will be described in greater detail later.

1e. Observations for Radiology.

A few reports (Arroyo, 1941; Hartzell, case 2, 1942; Van den Meer, 1946) mention the detection of apical pleural thickening, which had presumably preceded the attack of haemopneumothorax. Again, initial radiographs may show only a pneumothorax (Rist and Worms, 1940; Staffieri, case 1, 1943; Harrington and Frelick, 1947; Thomas and Beerens, 1948; Arst et al., case 1, 1950; Borrie, case 1, 1953; Garcia et al., case 1, 1953; Clyne and Hutter, 1955; Agnew, 1955) but the appearances of effusion are seldom delayed beyond 12, or at most 24 hours. The longest recorded delay, in this respect, was 10 days, in the patient of Agnew (1955). The probable explanation of this well-demarcated interval is that the haemorrhage arose from the delayed rupture of a pleural adhesion. This mechanism was suggested in case 1 of Garcia and associates (1953). The first radiograph revealed a pneumothorax with cord-like adhesions holding up the apex of the lung. Next day, there was radiologically a moderate effusion, while the adhesions had vanished and, at subsequent operation, were found to be torn. The alternative interpretation that a ruptured bulla had initiated both the pneumothorax and slow oozing of blood cannot be discarded since about 400 ml. of intrapleural blood are required to cast a radiologically-definite shadow in the costo-phrenic angle.

However, there is no such alternative explanation for the somewhat similar events in Borrie's first patient. He presented with a small pneumothorax and an associated apical "string" adhesion. In view of the "trivial" lesion he was admitted only after re-consideration. Within 24 hours there was a dramatic deterioration with almost complete exsanguination. At thoracotomy, blood was seen to ooze from the apical

parietal pleura, the presumed site of the radiologically-demonstrated adhesion, but no adhesions could actually be seen. At the lung-apex, there was a group of bullae, one of which was ruptured but, as stated, this bulla was not the source of haemorrhage.

Discussion, Summary and Conclusions from these Observations.

It can be safely concluded from the available objective evidence that two modes of production of idiopathic haemopneumothorax have been established. A third mechanism appears probable, but not readily explicable. There remain a few cases for which no explanation can be advanced on the basis of these observations. The causations, without prejudice to aetiology, may be enumerated as follows:

(1) A ruptured vascular bulla alone producing the escape of both air and blood into the pleural cavity.

(2) A ruptured bulla causing a selective pneumothorax, which in turn produces at least partial collapse of the lung, thereby stretching and eventually rupturing a vascular pleural adhesion.

(3) Less certainly, a ruptured vascular adhesion alone.

The last-mentioned lesion appears to be the cause of some of the idiopathic haemopneumothoraces and of nearly all truly idiopathic haemothoraces. This latter qualification is a safeguard until more is known about such well-nigh unique causes as aberrant lung-tissue (Hart and Jones, 1947) and other congenital anomalies situated contiguously to the pleura.

It may well be questioned whether a ruptured adhesion per se could produce a haemopneumothorax. Any valid explanation must include a mode of perforation of the lung parenchyma. The factor which determines whether

a ruptured adhesion produced a haemothorax or a haemopneumothorax must be the actual site of rupture on the adhesion, i.e. whether in its stem or its end adjacent to the visceral pleura.

Finally, concerning the aetiology of idiopathic haemopneumothorax, the apical, and occasionally bilateral distribution of the basic lesions, including scarring, would be consistent with an old and healed tuberculous process. This might account for some of the cases of the idiopathic types of haemopneumothorax and haemothorax. Furthermore, the puckered apical scarring, recorded in a few of the cases coming to autopsy (Fischer, 1922; Housden and Piggot, 1931; Davidson and Simpson, 1940) has been described as characteristic of healed tuberculosis. It must, however, be fairly stated that there is no direct evidence to substantiate this suggestion. It is also possible that in some of the cases in which no apical scarring was seen macroscopically, this might be present in microscopic degree.

(2) a. Relation to Idiopathic Pneumothorax.

The precise aetiology of idiopathic haemopneumothorax, although admittedly obscure, is identical in part with that of idiopathic pneumothorax. Indeed, intrapleural haemorrhage complicates some 5 per cent of cases of idiopathic pneumothorax (Table12). Nevertheless, any such explanation of the pathogenesis of idiopathic haemopneumothorax must account adequately for the associated intrapleural haemorrhage. This merits separate consideration laterin this section.

The fundamental study of idiopathic haemopneumothorax demands full knowledge of the nature and background of the primary condition, idiopathic pneumothorax. This will now be examined. In view of the assumed importance of a tuberculous aetiology, a temporary reversion in nomenclature to the wider term, spontaneous pneumothorax, is essential.

The earlier emphasis on active tuberculosis, as the main cause of spontaneous pneumothorax in hospital practice, has now been clearly rescinded in favour of an idiopathic aetiology. The word "active" is used expressly, in contradistinction to such inert vestiges as pulmonary cicatrization and pleural adhesions. Formerly, it was freely accepted (Biach, 1880; West, 1887; Palmer and Taft, 1931; Fishberg, 1932) that between 80 and 90 per cent of spontaneous pneumothoraces were due directly to pulmonary tuberculosis. This accentuation on the sanatorium-type of patient is well depicted in the dictum of Samuel West (Bradshaw Lecture, 1887): "of the causes to which spontaneous pneumothorax is due, phthisis is the chief."

It is hardly surprising, therefore, that the full recognition and characterisation of the idiopathic type of pneumothorax was delayed. However, there are added reasons. For example, the first report on the use of

TABLE 12

Incidence, in idiopathic pneumothorax, (Idio. pnx.), of associated intrapleural haemorrhage, i.e. of haemopneumothorax (Hpnx.), as reported in the literature. A 5 per cent incidence is revealed.

AUTHORS	YEAR	NO. OF CASES	
		Idio. Pnx.	Idio. Hpnx.
Kjaergaard	1932	51	2 ¥
Kirschner	1939	18	1
Leach	1945	126	2
Schneider & Reissman	1945	100	1
Rottenberg & Golden	1949	97	3 ¥
Hughes et al.	1951	40	1 ¥
Hyde and Hyde	1951	112	5 ¥
Moxon	1951	26	1
Dubose et al.	1953	75	2
Briggs et al.	1953	76	3
Myers et al.	1954	115	9 ¥
Shefts et al.	1954	114	6
Fry et al.	1955	108	13 ¥
Crowther	1955	61	1
TOTAL		1,119	50

¥ = tabulated in the analysis (Section I) of idiopathic haemopneumothorax.

X-rays in spontaneous pneumothorax was in 1899 (Williams). Again, sparse autopsy data existed on patients with the idiopathic type since they almost invariably recovered. By contrast, opportunity for post-mortem study in the tuberculous group was immense. This is reflected in the report of Morse (1900). He concluded, from an analysis of 51 cases of spontaneous pneumothorax, that: "the cases which recovered usually died from pulmonary tuberculosis; pneumothorax was the direct cause of death in 60 per cent; and, 80 per cent of all cases died within a year." Indeed, most of these patients exemplified the terminal and rapidly fatal onset of spontaneous pneumothorax in tuberculous subjects (Parkes Weber, 1905).

Having stressed the time-honoured role of tuberculosis, a re-orientated perspective will now be presented by a discussion on the non-tuberculous type.

Pathogenesis.

Kjaergaard's study (1932) clearly established that "spontaneous pneumothorax in the apparently healthy subject" is almost never due to active tuberculosis. Moreover, these patients are not especially prone to tuberculosis. Kjaergaard's evidence was based on serial radiological observations. The usual mechanism underlying spontaneous pneumothorax in healthy subjects appeared to be the rupture of one or other of two recognised types of subpleural "valve vesicles." Earlier histological studies (Hayashi, 1915; Fischer, 1922) revealed the distinctive morphological features of these structures, as cited and confirmed by Kjaergaard (1932), who in the following year added as a third and rare variety, the so-called "congenital valve-vesicle."

The commonest and frequently multiple type, the "scar-tissue vesicle," usually arises in the lung-apex near the scar of a healed inflammatory process. The latter, of course, includes an old healed tuberculous focus.

Kjaergaard's illustrated scar-tissue vesicles exhibit a valve-mechanism, attributed to shrinkage of scar-tissue, between a communicating bronchiole and the actual vesicle.

The second type, the "emphysematous valve-vesicle," frequently solitary, is due to purely localized emphysema, without scar-tissue. The valve is formed by a tongue or flap of distorted or compressed emphysematous lung-parenchyma between the vesicle and the communicating bronchiole.

The third type, the congenital valve-vesicle is probably more of a pathological curio. Kjaergaard (1933) could find only five cases in the entire literature in which autopsy demonstrated congenital pulmonary cysts as the cause of spontaneous pneumothorax, a subject to which Ross and Fullerton (1939) later contributed. As a variant, Kirschner (1938) suggested a congenital pleural defect, i.e. that pleural blebs might result from a primarily weakened pleura. This view is mainly unsupported. Without entering into the controversy on the validity of the term congenital, it suffices to state that the onus rests on the advocate to disprove an acquired basis.

The valve-vesicle, regardless of type, will distend and rupture to produce a localized interstitial emphysema, represented by the classical sub-pleural bleb such as has been visualized frequently by radiography and thoracoscopy, and also at operation or autopsy.

Apart from the main mechanisms of idiopathic pneumothorax just outlined, two later contributions (Macklin and Macklin, 1945; Brock, 1948) deserve brief mention. The Macklins produced pneumothorax and haemopneumothorax in experimental animals by forcible overinflation of the lungs. The first stage was the induction of pulmonary interstitial emphysema. They postulated similar events in man, following the intra-pulmonary

rupture of alveoli, which had been weakened from any cause. Air is then free to dissect along the sheaths of the pulmonary vessels to the mediastinum, where it ruptures through the thin mediastinal pleura to the pleural cavity. The clinical implications of this sequence have been discussed by other authors (Hamman, 1939; Draper, 1948) in relation to the production of spontaneous pneumothorax and to its concomitance with mediastinal emphysema. This complicated explanation of the Macklins should mainly be reserved for the pathogenesis of spontaneous pneumomediastinum. It is untenable for the vast majority of idiopathic pneumothoraces and haemopneumothoraces, in which there is now ample evidence and general agreement that subpleural bullae are the usual precursors of the pneumothorax.

Brock (1948) described certain bizarre, and previously unreported, observations appertaining to his thoracoscopic investigations in patients with chronic and recurrent spontaneous pneumothorax. In six patients he detected air exuding from the pleura as fine froth resembling "cuckoo-spit." This indicates alveolar and pleural leakage. Its full significance awaits elucidation.

As a final possible cause of idiopathic pneumothorax, the rupture of pleural adhesions must be entertained. The only plausible explanation is that the adhesion tears at its attachment to the visceral pleura and traumatises subjacent air vesicles (Weber, 1903; Perry, 1939), although this has not been subject to confirmation. In view of the marked frequency of pleural adhesions in pulmonary tuberculosis (Hyde and Hyde, 1949), one is left surmising why there is little mention of these lesions as a cause of pneumothorax, or indeed haemopneumothorax in these subjects, if they are important precursors. Nevertheless, if adhesions play a minor or doubtful rôle in the production of idiopathic pneumothorax, their importance as a

source of massive intrapleural haemorrhage has been put beyond doubt.

Aetiology.

As previously stated, few patients with idiopathic pneumothorax reach autopsy. Kjaergaard (1932) was only able to ascertain four such recorded cases. All four had emphysematous bullae, which in two patients (Ranking, 1860; Brunner, 1921) had ruptured. The remaining two patients (Hayashi, 1915) also had scar-tissue vesicles. Concerning the origin of these scar-tissue vesicles, neither Hayashi (1915) nor Fischer (1922) was able to offer any definite conclusions, as two of the autopsied cases showed no signs of tuberculosis either grossly or microscopically. Nevertheless, these authors favoured a previous, limited healed tuberculous lesion as the explanation.

The aetiological source of these scars remains unproven. Healing apical tuberculosis, admittedly, favours cicatrization of the lung and overlying pleura and, by producing localized compensatory emphysema, provides the physical basis for valve-vesicles. Thus, the puckered or stellate cicatrix of an old, healed, apical tuberculosis may explain the development of some spontaneous pneumothoraces in apparently healthy subjects. This view has the merit of explaining the frequent formation of adhesions, which like scar-tissue vesicles and apical scarring, may signify long-inactive, non-demonstrable tuberculosis (McKie, 1905; Cummer, 1915; Weber, 1931). In discussing the aetiology of bullae, Sycamore (1936) pointed out with subtle discrimination that bullae, in the academic sense, may be of tuberculous origin but from the practical aspect they are non-tuberculous. By the same token, Rothstein and Moberly (1952), in discussing emphysematous bullae in relation to pulmonary tuberculosis, contended that when these

structures have been examined after excision, the descriptions often refer to fibrosis and some non-specific inflammatory process but no pathological reports of tuberculosis as the underlying cause have been recorded in the literature.

Whilst, tuberculosis may well play an indirect part in some cases of spontaneous pneumothorax, other explanations are also necessary. For, as shown in Section B, a significant proportion of patients with idiopathic pneumothorax do not react to the tests with tuberculo-proteins. For this group, other lung-infections (Coope, 1948) provide the only alternative explanation.

Identity of Idiopathic Types of Pneumothorax and Haemopneumothorax.

The conclusion that the idiopathic types of pneumothorax and haemo-pneumothorax are intimately related rests on the following evidence:-

(1) The fact that intrapleural haemorrhage occasionally complicates idiopathic pneumothorax. In odd cases, the initial radiograph has shown only a spontaneous pneumothorax.

(2) The close similarity of case-particulars, i.e. age-groups, sex-distribution, occupation, frequency of side of chest involved, etc. (Section B).

(3) The frequent successive occurrence of pneumothorax and haemo-pneumothorax in the same patient (Section B).

(4) The frequent demonstration in both groups of sub-pleural bullae by radiology, thoracoscopy, at operation and at autopsy.

(5) The absence in both groups of historical, clinical, radiological and bacteriological evidence of pulmonary tuberculosis during supervision.

Similarly, the relative infrequency in both conditions of the development of tuberculosis during surveillance. Also, microscopically similar valve-vesicles (Hayashi, 1915; Fischer, 1922) have been demonstrated in the idiopathic types of pneumothorax and haemopneumothorax.

(2) b. Relation to Tuberculosis and the Tuberculous Type of Haemopneumothorax.

The incidence of spontaneous pneumothorax as a complication of pulmonary tuberculosis had been variously estimated (3.5 to 10 per cent, Parkes Weber, 1905; 2 to 4 per cent, Watson and Robertson, 1928; 1.4 per cent, Hyde and Hyde, 1949). This diminishing frequency may be explained by a corresponding lowered incidence of advanced tuberculosis, i.e. the stage in which spontaneous pneumothorax is most prevalent. This association is reflected in the casual remark of Parkes Weber, 1905 when discussing spontaneous pneumothorax in tuberculous subjects: "it will not be necessary to give examples of the well-known rapidly fatal terminal cases." Indeed, Williams and Williams (1887) discovered that spontaneous pneumothorax was present in 10 per cent of the autopsies performed on consumptives.

Conversely, the present incidence of concurrent pulmonary tuberculosis in patients with spontaneous pneumothorax is generally computed as about 5 - 15 per cent (Kirschner, 1938; Perry, 1939; Briggs et al., 1953; Rapport et al., 1953), albeit with notable exceptions. For instance, Myerson (1948) found that, of one hundred patients with spontaneous pneumothorax, seen at the Boston City Hospital between 1934 and 1943, 38 had pulmonary tuberculosis.

There are several explanations for the occurrence of pneumothorax in tuberculous patients. Excluding therapeutic and accidental forms, the two usual causes are (1) the rupture of a co-incidental emphysematous bulla, and (2) a genuine tuberculous pneumothorax, resulting from sub-pleural caseation or cavitation eroding the visceral pleura. The site of ulceration is usually in the upper third of the pleura and on its anterior and lateral aspect (Hyde and Hyde, 1949). In this true tuberculous type, a pleural

inflammatory response, characterized by hydro- or pyopneumothorax, but not haemopneumothorax, usually supervenes, with increased respiratory embarrassment, toxaemia, fever and tachycardia (Hyde and Hyde, 1949).

Although pulmonary tuberculosis, when complicated by spontaneous pneumothorax, is generally well-advanced (Korol, 1936; Perry, 1939; Hartzell 1942; Hyde and Hyde, 1949), there are occasional instances of co-existent minimal or single tuberculous lesions (Parkes Weber, 1905; Kjaergaard, 1932). Few observers would gainsay Hartzell's dogmatism (1942) that "if tuberculosis was not easily discovered at the time of the haemorrhagic episode, the case could confidently be considered non-tuberculous," in terms of true tuberculous haemopneumothorax.

The true relative frequencies of ulceration of subpleural tuberculous lesions and of ruptured bullae in initiating pneumothorax and haemopneumothorax in tuberculous subjects is unrecorded. Unquestionably, ruptured bulla account for some, an as yet undetermined proportion, of these pneumothoraces. Perry (1939) recorded that in three fatal cases of spontaneous pneumothorax in tuberculous subjects, the pneumothorax originated from a ruptured bulla, and not from tuberculous pleural ulceration. As Kjaergaard (1932) suggested, when a dry spontaneous pneumothorax remits in a consumptive patient, the natural assumption is that the incident was due to a ruptured bulla. Moreover, even in ingravescent pulmonary tuberculosis, a complicating pneumothorax may exceptionally be of bullous origin, (Wilson, 1937). Waring (1945) stated that, of approximately one thousand patients treated during a seven-year period at the National Jewish Hospital (Denver), there were no instances of tuberculous haemopneumothorax, 6 of spontaneous pneumothorax due to a ruptured bulla during well-controlled tuberculosis, and only one due to

caseous ulceration through the pleura in patients not undergoing artificial pneumothorax therapy.

Spontaneous pneumothorax in healthy subjects is not due to active tuberculosis. Kjaergaard (1932) italicized this fact: "in all the literature, as far as I have been able to find out, there is not one single instance in which the rupture of a tiny tuberculous focus has been the cause of spontaneous pneumothorax in an apparently healthy person, taking the course that is typical of this lesion and unaccompanied by fever and pleural effusion." Perry (1939) endorsed this view: "It would appear that the idea of a minimal subpleural tubercle as a cause of this type of pneumothorax is entirely theoretical and completely devoid of any backing either clinical or pathological." Parkes Weber (1931) contended that the only difference between tuberculous and idiopathic pneumothorax or haemo-pneumothorax is that in the former the tuberculous process is active while in the latter it is inactive. This is undoubtedly an over-simplification, for it seems certain that tuberculosis is by no means the sole causa causans of the idiopathic varieties of these conditions, as judged by the frequency of negative reactions to tuberculin tests (Section B).

Although relevant statistics are unavailable, true tuberculous haemopneumothorax appears to be much rarer than either true tuberculous pneumothorax or the haemothorax which occasionally follows artificial pneumothorax refills (Korol, 1936; Raimondi and Lerner, 1941; Hyde and Hyde, 1951). There appear to be two explanations for the rarity of true tuberculous haemopneumothorax (Hurst and Epstein, 1937).

Firstly, and more important, as just described, a tuberculous focus or cavity may rupture through the visceral pleura. However, this mechanism rarely leads to intrapleural haemorrhage, since in slowly progressive

tuberculosis the blood-vessels in the affected area usually become thrombosed. In the case of cavitation, these vessels appear as mere traversing cords. However, in rapidly progressive disease with cavitation at the lung periphery, a still patent vessel might be eroded and lead to haemopneumothorax when the visceral pleura is ruptured.

The second cause or factor in the tuberculous haemopneumothorax is the rupture of pleural adhesions. While torn adhesions alone constitute a doubtful cause of haemopneumothorax their potential role in producing intra-pleural haemorrhage, after the development of spontaneous pneumothorax by the first mechanism, is indisputable. In this respect, it is perhaps noteworthy that Hyde and Hyde (1949) reported that 92 per cent, of a series of 40 patients with tuberculous pneumothorax, exhibited radiological evidence of pleural adhesions, which were not demonstrable at all in any of their series of 85 patients with idiopathic pneumothorax.

A third cause of haemopneumothorax in tuberculous subjects is the rupture of an emphysematous bulla, just as in non-tuberculous patients. This explanation, aided in certain cases by torn adhesions as a possible source of haemorrhage, would account for the haemopneumothoraces in the tuberculous patients of Allen (1925), Dorendorf (1932), Birch (1936), Beatty (1939), Crimm (1948) and Myers (1953), now to be discussed.

The fourth cause of haemopneumothorax in tuberculous patients, i.e. haemothorax related to artificial pneumothorax will be separately considered later.

The occurrence of haemopneumothorax in patients with simultaneous or subsequent pulmonary tuberculosis will now be carefully and critically reviewed by reference to the main illustrative cases in the literature. It is to be noted that in many patients the haemopneumothorax and the

tuberculosis were located in different sides of the chest.

Allan (1925) narrated two cases of spontaneous haemopneumothorax which would have been acceptable examples of the idiopathic type except that after recovery from the haemopneumothorax there was radiological evidence of minimal tuberculosis. Both patients were then entirely symptom-free. No firm conclusion on the aetiology of these haemopneumothoraces is warranted, but a pure tuberculous aetiology seems unlikely.

Dorendorf (1932) reported the fatality from spontaneous haemopneumothorax of a previously healthy 33-year old male shop-assistant. While jumping off a moving tram-car he was seized with pain in the right side of his chest. Three hours later he was admitted to hospital with signs of a right-sided tension pneumothorax with effusion. Deflation-therapy afforded relief but shock ensued. He died twelve hours after the onset of the pain. At autopsy, both air and over two litres of blood occupied the right pleural cavity. The right lung-apex showed local signs of haemorrhage from a bulla. Adhesions were not mentioned. Bilateral apical scarring, presumably tuberculous, and a pea-sized area of caseation in the left (uninvolved) lung were also noted. There was no direct proof that the haemopneumothorax was of tuberculous aetiology. The probable explanation was the onset of both the pneumothorax and the haemorrhage from the ruptured bulla.

Since the literature on spontaneous haemopneumothorax in tuberculous patients is mainly confined to single cases, the four illustrative cases of Korol (1936) will be analyzed in greater detail. He reviewed the subject of tuberculous haemorrhagic pleurisy after 1900 and concluded that this was not an inflammatory condition. Instead, it was due to haemorrhage associated with a spontaneous pneumothorax. He discredited the view that

the haemorrhage arose from intra-pleural granulations. He attributed the pneumothorax to a ruptured emphysematous bulla and the associated haemorrhage to the rupture of a pleural adhesion, i.e. the common mechanism in the production of idiopathic haemopneumothorax. He reported six cases of spontaneous haemopneumothorax, four of which had pulmonary tuberculosis. These four cases will now be described and discussed.

The first patient (Case 2), a man aged 38 years, felt a sudden stabbing pain in the left side of his chest while walking. He managed to walk home and took to bed. Next morning, he felt faint and his doctor diagnosed a left-sided hydro-pneumothorax. On admission to hospital, three weeks later, thoracentesis revealed "pure blood," which gave a negative result on guinea-pig inoculation. Two months later he was discharged from hospital with virtual re-expansion of the left lung. Six months later, he was re-admitted for two months, with radiological evidence of right apical infiltration as a new feature. His sputum was negative for acid-fast bacilli. Three years later, when last seen, he appeared well nourished but complained of lassitude. No concurrent radiological report is given. In summary, the only evidence of tuberculosis was the radiological appearance of right apical infiltration, six months after a left-sided haemopneumothorax. There is, therefore, no certainty that the latter was of tuberculous origin.

The second patient (Case 3), a man aged 40 years, had tubercle bacilli in his sputum. He had been treated for four years for progressive tuberculosis of the right lung, associated with two episodes of pleural effusion, when the latter recurred. Aspiration now revealed air under high pressure and a yellow fluid. After two aspirations, however, the pleural fluid was haemorrhagic but did not infect the inoculated guinea-pig.

Eighteen months later, the need for aspiration became less frequent, the fluid appeared less red, and tubercle bacilli disappeared from the sputum. X-ray now showed few remaining cavity-shadows in the right lung. Briefly, therefore, this may have been a true tuberculous haemopneumothorax, although the delayed intra-pleural haemorrhage is unusual, and raises the possibility of traumatic haemorrhage from thoracentesis.

The third patient (Case 4), a male of unstated age, had felt ill for 2 years, with morning cough, breathlessness and lassitude. A diagnosis of pulmonary tuberculosis with a left-sided effusion was made. Thoracentesis, however, revealed a fluid resembling blood, which gave a negative result on guinea-pig inoculation. Haemorrhagic effusion persisted for the next six years while serial X-ray films showed a persistent left-sided hydro-pneumothorax but no abnormality of the right lung. He was considered to have inactive tuberculosis. The sputum was consistently negative for acid-fast bacilli. Repeated aspiration failed to re-expand the left lung, presumably because of a persistent broncho-pleural fistula. In summary, while the evidence of pulmonary tuberculosis was purely clinical, this may well have been a case of tuberculous haemopneumothorax, in view of the chronicity of the effusion.

The fourth patient (Case 5), a male of unstated age, had been treated for tuberculosis of the left lung for $4\frac{1}{2}$ years, when a left-sided haemopneumothorax was confirmed . In the earlier phase of his illness, treatment with artificial pneumothorax and, later, section of pleural adhesions effected the closure of his tuberculous cavity and his sputum became negative for acid-fast bacilli. For the next 2 years he had a gradually accumulating left-sided serous effusion, and so pneumothorax refills were discontinued.

Thoracentesis revealed a haemorrhagic fluid under tension. This fluid contained tubercle bacilli. The effusion persisted for at least $2\frac{1}{2}$ years and frequent aspirations became necessary to relieve dyspnoea. Korol concluded that the combination of haemorrhage, intra-pleural tension and demonstration of tubercle bacilli denoted a simultaneous erosion of a blood-vessel and perforation through the visceral pleura.

Dufourt and colleagues (1938) described the insidious onset of haemopneumothorax in a tuberculous patient, a woman aged 45 years, 4 years after her initial admission to a sanatorium. She was re-admitted one month after the discovery of a left-sided pleural effusion. Exploratory thoracentesis disclosed a fluid resembling blood. The sputum contained acid-fast bacilli. For the next month her signs remained stationary and it was concluded that the haemopneumothorax had progressed to a chronic phase of pachypleuritis haemorrhagica. This might have been a genuine case of tuberculous haemopneumothorax.

Birch (1936) recorded a fatality from spontaneous haemopneumothorax in a clerk aged 35 years. Symptoms began 48 hours before admission to hospital, where he died within a few hours of arrival. Autopsy revealed several pints of blood in the right pleural cavity. In the right antero-apical region was a roughened area on the pleura, the presumed site of haemorrhage and escape of air. No emphysematous bullae and no pleural adhesions were demonstrable. At the right lung-apex there was a minute tuberculous cavity with intact walls. In a later report (1938), Birch expressed the view that this haemopneumothorax was not directly attributable to the tuberculous process, i.e. the closed tuberculous cavity.

Beatty (1939) reported a case with co-existent haemopneumothorax and infiltration at the opposite lung apex, radiologically. The patient a 20-year old

messenger, presented with a left-sided tension haemopneumothorax. A total of 2,400 cc. of air was fractionally withdrawn with great relief. He was discharged eleven days after the onset. Four months later, on routine re-attendance, his sputum contained acid-fast bacilli while on X-ray examination the right lung-apex (uninvolved side) showed infiltration and cavitation. The left lung, however, was clear. Beatty concluded that the haemopneumothorax was probably of tuberculous aetiology.

At this juncture, it is convenient to refer collectively to several cases of haemopneumothorax in which clinical recovery seemed to be complete and yet there was clear bacteriological evidence of tuberculosis. These cases are not described in detail here as they differed in no clinical respect from typical cases of idiopathic haemopneumothorax. Wilson (1936) referred to a railway-clerk aged 30 years with tubercle bacilli in his sanguineous pleural fluid. In case 3 of Bernstein and associates (1946) a man aged 25 years with spontaneous haemothorax, the tubercle bacillus was obtained on culture of the pleural fluid while in both the patient briefly mentioned by Berliner (1941) and case 1 of Crimm (1948), a bank-clerk aged 24 years, the guinea-pig responses to inoculation with pleural fluid were positive for tuberculosis.

Beatty and Frelick (1952) recorded a rather unusual event (Case 7). A left-sided haemopneumothorax developed eighteen months after a right-sided therapeutic pneumothorax for right apical tuberculosis. The patient's previous records disclosed no evidence of tuberculosis in the left lung. Suddenly, while at home, he developed a very severe pain in the left lower chest. On admission to hospital the X-ray film showed bilateral pneumothorax with a left-sided effusion. After one week's stay in hospital, where six blood transfusions were given and $2\frac{1}{2}$ litres of "blood" were

removed by thoracentesis, he was discharged home with virtually complete re-expansion of the left lung.

Myers (1954) narrates the case of a man aged 24 years who had a left-sided spontaneous haemopneumothorax, unassociated with clinical evidence of pulmonary tuberculosis. Five years later he exhibited moderately advanced tuberculosis in the right lung, which was promptly treated successfully.

Conclusions.

Any attempt to draw conclusions on the spontaneous varieties of pneumothorax and haemopneumothorax in tuberculous patients is at once rebuffed by the limited information available. However, certain broad conclusions appear to be permissible.

(1) Incidence.

Tuberculous patients now appear to comprise about 5 - 15 per cent of all cases of spontaneous pneumothorax, while the latter condition supervenes in perhaps about 2 - 3 per cent of all patients with pulmonary tuberculosis. These present incidences represent a marked decline in those obtaining early in this century.

The corresponding figures for spontaneous haemopneumothorax are unknown but are believed to be very much lower, since spontaneous pneumothorax contrasts strongly with spontaneous haemopneumothorax because its infrequency as a reported complication of pulmonary tuberculosis.

(2) Pathogenesis.

The true tuberculous types of pneumothorax and haemopneumothorax are due to the erosion of the visceral pleura by a tuberculous subpleural lesion and are mainly confined to patients with advanced tuberculosis. The source of haemorrhage in tuberculous haemopneumothorax is uncertain. When not

arising from an eroded subpleural vessel, this haemorrhage may be due to ruptured pleural adhesions. Indeed, pleural adhesions frequently accompany tuberculous pneumothorax.

In other instances of pneumothorax and of haemopneumothorax in tuberculous subjects, the pathogenesis may be similar to that obtaining in the idiopathic types of pneumothorax and haemopneumothorax, i.e. the rupture of subpleural bullae and, perhaps, of pleural adhesions.

The relative frequencies of these two modes of production of pneumothorax and haemopneumothorax in tuberculous subjects is unknown.

(3) Prognosis.

The onset of spontaneous pneumothorax or haemopneumothorax in patients with pulmonary tuberculosis is usually a grave event. This view was well illustrated by Fishberg (1932). He averred that more nocturnal fatalities in sanatoria-patients were due to a swift visitation of pneumothorax than of internal pulmonary haemorrhage.

In those tuberculous patients, in whom the causes of idiopathic haemopneumothorax are operative, the prognosis should be relatively enhanced over the preceding group, but less sanguine than in patients with idiopathic haemopneumothorax. While all these varieties of haemopneumothorax are potentially fatal, the actual range of clinical severity of individual cases, can be extreme, notably in the idiopathic type.

(2) c. Relation to Idiopathic Haemothorax.

The diagnosis of idiopathic haemothorax rests on the elimination of the accepted causes of haemothorax (Berliner, 1941; Crawford and Shafar, 1946; Cuningham, 1950). Briefly, excluding trauma, there are four main groups of causes to be considered:-

(1) Neoplasm of the lung or pleura.

(2) Pulmonary infections, especially tuberculosis.

(3) Haemorrhagic diseases.

(4) Vascular catastrophes, e.g. pulmonary embolus or rupture of an aortic aneurysm.

Berliner (1941) presented a comprehensive account of haemorrhagic pleural effusions and haemothorax, with an analysis of the case-records of 120 patients, who had been supervised in a large general hospital within a period of eleven years. He reported that 78 (65 per cent) of the haemorrhagic pleural effusions were due to neoplasm and, in all but one instance, there was associated metastatic pleural involvement. Surprisingly, pulmonary infarct and tuberculosis emerged as less important causes, i.e. comprising only 10 and 8, respectively, of the 120 cases.

In the same period, 6 cases of spontaneous haemopneumothorax had been admitted. Of these 6 patients, 5 were considered to be idiopathic, while the remaining patient's pleural fluid gave a positive guinea-pig response. This tuberculous patient recovered. One of the idiopathic group died and had, at autopsy, several subpleural blebs, while another patient had no demonstrable air in the pleural cavity and at first appeared to be a genuine example of idiopathic haemothorax, but the

supervention of rheumatic heart-disease and bilateral pleural effusions confused the issue.

The literature on idiopathic haemothorax reveals the need for careful consideration prior to a definitive diagnosis. Here the margin of error exceeds that for the diagnosis of idiopathic haemopneumothorax. Indeed, the only requisite for the production of a clinical picture simulating idiopathic haemothorax is a haemorrhagic lesion involving, invading or irrupting into the pleural cavity.

The entire literature on idiopathic haemothorax totals twelve acceptable cases (Table 13). Admittedly, therefore, one hesitates to draw conclusions. Yet, certain general comments are warranted. For instance, the resemblance in main features to the group of patients with idiopathic haemopneumothorax is striking. Indeed, the age-range (17 to 50 years) with a mean age of 32 (excluding the school-boy of unstated age), the sex-ratio of 11 : 1 male-predominance, the equal frequency of involvement of each hemithorax, and, perhaps, the mortality-ratio (4 : 12), are in general harmony. Similarly, the personal and family histories, the immediate history as regards action at onset, as well as the duration of illness are also in accord.

Added convincing evidence that these two groups are closely allied is derived from the information at autopsy (Table 10). To recapitulate, the patient of Davidson and Simpson (1940) had the full basic pathology of a haemopneumothorax. Moreover, apart from Case I of Davidson (1935) with no apparent significant lesion on the affected side, yet contralateral adhesions and apical scarring, the other three fatal patients all

TABLE 13.

Main Features in Idiopathic Haemothorax (12 patients).

AUTHOR (year)	AGE & SEX	SIDE	NATIONALITY and OCCUPATION	PREVIOUS LUNG INFECTION	FAMILY T.B.	ACTION at ONSET	LATENT PERIOD (days)	TOTAL PERIOD OF ILLNESS	OUTCOME	HB. (%)
Davidson (1935) Case I	26 M.	R.	British Tailor	-	-	-	1	1 day	Died	-
Davidson (1935) Case 2	28 M.	R.	British Clerk	Nil	No	In bed	21	3 wks.	Died	-
McRae (1939) Case I	50 M.	L.	Canadian Doctor	Nil	Yes	Cranking car	1	6 wks.	Recovered	65
McRae (1939) Case 2	34 M.	L.	Canadian Farmer	Nil	-	Stretching	Few hours	6 wks.	Recovered	-
Snively et al. (1942)	Boy	R.	American Schoolboy	-	Yes	-	1	8 wks.	Recovered	65
Almeyda (1943)	41 M.	R.	Brit.Printer's roller maker	Pneumonia Bronchitis	-	Bout of Coughing	10	3 mths.	Recovered	-
Crawford and Shafar (1946)	44 M.	L.	British Civil serv.	-	-	In tram	8	8 wks.	Recovered	-
Deodhar (1947)	20 M.	L.	Indian Farmer	Nil	No	-	28	3 mths.	Recovered	-
Crimm Case 2 (1948)	48 F.	L.	American Housewife	-	-	-	2	6 wks.	Recovered	-
Walton Case I (1950)	29 M.	L.	British Engineer	Nil	No	Sitting	1	10 wks.	Recovered	95
Cuningham Case I (1950)	17 M.	R.	New Zealand -	-	-	-	1	2 days	Died	60
Irwin (1951)	45 M.	R.	American Major	-	-	-	9/24	9/24	Died	-

exhibited relevant lesions. The affected side showed:

(a) emphysematous bullae.

(b) pleural adhesions, which were torn in all but one patient (Davidson, Case 2).

(c) apical scarring, in all but one instance (Davidson, Case 2).

From this limited, yet surprisingly uniform evidence, in the study of idiopathic haemothorax, it can be tentatively concluded that:-

(1) Haemorrhage usually arises from a ruptured pleural adhesion.

(2) Emphysematous bullae may be a frequent feature, since they are usually evident at autopsy.

(3) A unity of aetiology with idiopathic haemopneumothorax seems certain. Indeed, some of the reported cases of idiopathic haemothorax may in reality be idiopathic haemopneumothoraces.

2 (d). Relation to Haemothorax during Artificial Pneumothorax (A.P.)

This topic merits only briefest mention. Since the first clinical manifestation in the development of idiopathic haemopneumothorax is often a pneumothorax, the analogy with haemothorax during A.P. is readily apparent. This analogy, however, must not be pushed too far since the patients with idiopathic haemopneumothorax are of recent previous good health. Yet, it seemed of interest, for comparative purposes, to ascertain the recognised sources of haemorrhage in tuberculous patients, subjected to A.P..

The limitations of this approach are that:-

(1) Haemothorax rarely complicates A.P.. The reported incidence of clinically recognisable cases varies from 0.003 (Harold, 1951) to 0.03 (Mitchell, 1952) per cent.

(2) There are numerous sources of haemorrhage (Harold, 1951), and their relative frequency is undertermined.

(3) Most patients recover from this complication of A.P. and, needless to say, the precise source of bleeding is rarely established.

(4) Haemothorax may occur either during A.P. or as a recognised late complication, i.e. weeks or months after the cessation of A.P. (Hurst and Epstein, 1937; Diamond, 1952).

The general subject has been reviewed and discussed (Korol, 1936; Hurst and Epstein, 1937; Harold, 1951; Mashiter, 1952; Mitchell, 1952; Diamont, 1952).

Haemorrhage, coincident with A.P. treatment, is at times traumatic. The lateral branch of the internal mammary artery (Cleland, 1948), an intercostal or intrapulmonary vessel, or a vessel within an adhesion, may be traumatised.

In the non-traumatic group, the cause of haemorrhage is often indeterminate. Indeed, few direct observations are recorded. In the fatal case of Krause and Heise (1920), no source of haemorrhage was identified but it was surmised that an adhesion had been pulled away from the thoracic wall by the instilled air. Again, Rocha and Mendes (1945) narrate a case, in which thoracotomy revealed haemorrhage from the parietal pleura, presumably from a ruptured adhesion.

Paradoxically, therefore, as judged by the literature, the history of A.P. reveals an unexpected low incidence of haemorrhage from torn adhesions. This apparent rarity is hard to reconcile with the acknowledged prevalence of pleural adhesions in tuberculous patients. It can, however, be concluded that stretched and torn adhesions constitute a recognised source of haemorrhage during A.P., but the true frequency of this complication may well be concealed by an incomplete and therefore unrepresentative documentation.

Finally, for completeness, haemothorax following the discontinuation of A.P. deserves mention. Here the source of bleeding is speculative and is attributed to the rupture of delicate newly-formed pleural vessels or adhesions. Korol (1936) contended that while the lung is collapsed, pleural adhesions, which are usually present, are gradually stretched by the elastic recoil of the lung; Diamond (1952) maintained that the important factor was the pronounced negative intrapleural pressure, resulting from incomplete re-expansion of the lung after cessation of A.P.. These arguments are not very cogent and the problem justifies extended study.

(3) Sources of Intrapleural Haemorrhage.

The two sources of haemorrhage in idiopathic haemopneumothorax are, in order of importance and frequency, torn adhesions and torn bullae, while in idiopathic haemothorax, torn adhesions are virtually the sole cause of bleeding. These conclusions were derived more from information at operation than at autopsy. Separate consideration will now be given to haemorrhage from adhesions and from bullae.

Haemorrhage from Adhesions.

Pleural adhesions are believed to originate from pleural disease, usually associated with pulmonary infection from one of numerous causes. It is hard to reconcile this concept with the observations of Leopold and Lieberman (1935). These workers identified pleural adhesions, at autopsy, in 50 per cent of all patients above the age of 20, i.e. the age-group under present consideration, after they had eliminated those patients with a history of acute or chronic pulmonary disease.

However, the same discrepancy occurs in the correlation of previous damage to peripheral lung-tissue with idiopathic pneumothorax (Coope, 1948) and with idiopathic haemopneumothorax (Author, Section B). Thus, the explanation for "scarring of the lung periphery" the precursor of scar-tissue vesicles and adhesions in idiopathic haemopneumothorax, is not readily elicited. Nevertheless, a partial explanation is that pulmonary infections of childhood, including the broncho-pneumonias of measles and whooping-cough are not always known or readily recalled in adulthood.

There is evidence from histological study in fatal cases of the

idiopathic types of haemopneumothorax and haemothorax that pleural adhesions can be highly vascular (Davidson and Simpson, 1940; Helwig and Schmidt, 1947; Irwin, 1951). Similarly, the bleeding following the section of adhesions (pneumonolysis) can be profuse (Sellors, 1945, Tucker, 1945). In the entity, under present consideration, direct observation at emergency thoracotomy has shown that the intrapleural haemorrhage usually arises from the parietal tag of a ruptured adhesion. This parietal segment is supplied by anastomotic collaterals from the intercostal vessels (Matson, 1942), whose pressure is that of the systemic circulation, i.e. six times that of the lesser or pulmonary circulation. The visceral pleura is supplied by the pulmonary arteries and ramifications of the bronchial arteries (Gray, 1954). Only the latter, of these vessels, participates in the systemic circulation.

Thus, haemorrhage from a parietal tag tends to be more dangerous, especially also since it is not subject to the haemostatic influence of the collapsing lung, as in the case of pulmonary haemorrhage. It is pointless therefore to rely on pulmonary compression by the maintenance of the pneumothorax for the control of intrapleural haemorrhage in the idiopathic types of haemopneumothorax and haemothorax.

Haemorrhage from Bullae.

It might at first seem surprising that significant bleeding could originate from a ruptured bulla since some bullae arise from scar tissue. Moreover, the collapsing lung should reduce the circulation to that area. However, fatal intrapleural haemorrhage has occasionally been attributed,

on good evidence, to torn bullae (Fischer, 1922; Tait and Wakeley, 1935; McMyn, 1947) while oozing from this site has been witnessed at operation (Borrie, 1953; Fry et al., 1955). Thus, the early observation of Mazzei and Pardal (1934), that subpleural bullae may be richly vascularised has been confirmed.

Finally, oozing of blood rather than frank haemorrhage may occasionally be witnessed per-operatively. In these instances, it must be assumed that the bleeding is occurring simultaneously from numerous small vessels rather than from one of larger calibre.

SECTION 4.

Clinical Features and Diagnosis of Idiopathic Haemopneumothorax.

Symptoms.

The onset is dramatic in about 95 per cent of cases and it is not the onset but the evolution, which differs from that in idiopathic pneumothorax. The patient, while in good health and frequently during relative inactivity, experiences pain, of a sharp or stabbing, rarely tearing or transfixing type. Two patients (Korol, Case 2, 1936; Wilson, 1937) felt "as if something had snapped" in the chest. Two patients (Jennings, 1940; Lea, 1942) had merely a dull ache, while in a further six (Bushby, 1913; Louria, Case 3, 1938; Egues, Case I, 1938; Schneider and Reissman, 1945; Fusia and Cook, Case I, 1952; Garcia et al., Case I, 1953) pain was apparently absent.

The pain is usually felt anteriorly, rather than posteriorly. Although often felt near the mid-line, the site of pain reliably indicates the side of involvement. The radiation of pain can be diverse and confusing, as will be discussed. The initial phase generally lasts for several hours, even up to a day or more. After a variable respite within the first 48 hours, pain is liable to recur. Nevertheless, the three clear-cut alternating phases, i.e. pain, relief and pain, regarded as classical (Korol, 1936), are not mentioned in more than half of the case-descriptions. These two episodes of pain were considered by Korol to represent respectively the onset of the pneumothorax and the rupture of an adhesion as the source of haemorrhage.

Radiation of the pain to the shoulder or back of the chest is occasionally described, while radiation to the arm and hand (Davidson, Case 2, 1935; Poli, Case I, 1936; Centeno et al., 1936; Rist and Worms, 1940; McMyn, Case 2, 1947; Dardet, 1948; Deucher, Case I, 1949; Leston and Piheu, 1949; Author's Cases 6 and 8) deserves special mention. The pain may rarely be entirely located in the abdomen or in the posterior aspect of the thorax, especially in the scapular and inter-scapular zones. In just under 10 per cent of cases, upper abdominal pain co-exists with chest-pain, especially when the latter is on the right side.

Right upper abdominal pain is well recognised and may even be associated with abdominal tenderness and rigidity, (Waring, 1945). This pain may arise from the irritation of the diaphragmatic and costal parietal pleura with a reflex pathway via the sympathetic supply to the abdominal viscera. Since the pain in idiopathic haemopneumothorax can be as severe as in the rupture of an abdominal viscus (Holden, 1935), an occasional erroneous laparotomy is understandable (Fischer, 1922; Morlock, 1933; Stockler, 1947; Fusia and Cook, 1952; Ross, Case 4, 1952). Nausea and vomiting, noted in about 10 per cent of all cases, may further divert attention to the abdomen.

Breathlessness, although rarely absent, is seldom the major complaint, except in cases of tension haemopneumothorax (Schneider and Reissman, 1945; Hansen, Case 2, 1949; Borroni, 1951; Beatty and Frelick, Case 6, 1952; Agnew, 1955) or in atypical cases with deferred pain (Landron and Irwin, 1937; Louria, Case 3, 1938). The onset of tension pneumothorax can also be deferred (Rist and Worms, 1940; Beatty and Frelick, 1951; Author's Case 5).

Symptoms of syncope have frequently been mentioned either pre-admission or while in hospital and, in particular, in relation to aspirations. Disquietude and lassitude are fairly constant while frank exhaustion may even arise.

On the basis of these symptoms alone, six rather arbitrary varieties of idiopathic haemopneumothorax can be distinguished by the site and character of the pain, the degree of shock and of the breathlessness.

These are:- (1) the anterior pleuritic type (classical type).

(2) the "acute abdominal" type.

(3) the pseudo-coronary type.

(4) the syncopal type.

(5) the suffocating type.

(6) the scapular or posterior thoracic type.

The anterior pleuritic type has an approximate incidence of 75 per cent, while the remaining types each occur in about 5 per cent of the whole group. The various abdominal conditions which have been simulated include perforation of, or haemorrhage from, a peptic ulcer; acute cholecystitis; and acute appendicitis. The thoracic conditions, which have been entertained, include acute pericarditis (Hees, 1937; Egues, Case 2, 1938). The scapular type is liable to superficial confusion with fibrositis.

Signs.

The signs of idiopathic haemopneumothorax do not differ significantly from those of any other type of acute haemopneumothorax. They are the summation of a pneumothorax, pleural effusion and of haemorrhage. The anxious and pallid appearance is rarely accompanied by venous

congestion. Cyanosis is specifically recorded in only about 10 per cent of all cases and it should immediately suggest the possibility of a tension pneumothorax. Tachypnoea and shallow respiratory excursion of the affected hemithorax are rarely absent. A short, dry, irritating cough, often transient, is present in at least 25 per cent of the cases. Presumably the reflex arises from the collapsing lung or indirectly from the pleura. The various classical signs, as for a hydropneumothorax, need no repetition; nor, indeed those of shock. Signs of shock are present in about 85 per cent of all cases at some time within the first two days and may re-appear in, or be delayed for, several days. This occasional chequered course, therefore, permits no relaxation in vigilant management.

Occasionally, the patient may be immobilised with pain (Catuogno, Case 2, 1935; Holden, Case I, 1936; Rist and Worms, 1940; Hartzell, Case 3, 1942; Arst et al., Case I, 1950; Author's Case 7) from spasm of the intercostal muscles. Abdominal signs, consisting of tenderness, spasm or, rarely, rigidity, especially in the right upper quadrant, are exhibited in about 20 per cent of all cases.

Without resort to a "confirmatory" radiograph or diagnostic thoracentesis, there are two virtually pathognomonic features:-

(1) the sudden appearance, in a previously healthy patient, of both a pneumothorax and signs of a pleural effusion, especially with well-marked signs of shock. Indeed, pleural accumulations other than blood invariably have a slower onset.

(2) A succussion splash, under similar circumstances. There are even recorded cases (Landron and Irwin, 1937; Troisier et al., 1937; Maxwell, 1938; Deiss et al., Case 2, 1950; Borrie,

Case 3, 1952), where the patient volunteered a sensation of splashing in his chest. The patient of Troisier and colleagues was aware of this sensation during each step, while walking home.

Finally, there are three diagnostic emergencies, i.e. tension haemopneumothorax, bilateral pneumothorax with intrapleural haemorrhage, and exsanguinating haemothorax. The immediate release of air under tension from the pleural cavity by under-water seal drainage may be life-saving in the first condition, while urgent blood transfusion, preceded if necessary by plasma infusion, is imperative in the third. For the patient with bilateral simultaneous pneumothorax, extraction of air from the more involved side would be indicated. This condition, even without an accompanying haemothorax, carries a 50 per cent mortality (Perry, 1939). In this respect, there are two rather similar doubly-complicated cases recorded (Rossel, 1935; Grundi, 1953) where an idiopathic haemopneumothorax was complicated several days later by a contralateral idiopathic pneumothorax. Both patients died.

Complications (see also Section 2).

Idiopathic haemopneumothorax may be complicated, apart from fatality and tension haemopneumothorax, by:- fibrothorax, including organising haemothorax and pachypleuritis; empyema; calcification of the pleura; and, possibly, pulmonary hypertension. These complications are readily recalled when it is appreciated that they are almost entirely due to the haemothorax. This realisation encouraged a more "aggressive" approach to therapeutic aspiration (Barrett, 1945; Sellors, 1945; Miller and Rinkel, 1947; Ogilvie, 1948) to reduce the foreign body reaction and pleural exudation.

Fry and colleagues (1955) wisely pointed out that overall morbidity with loss of occupational-time, which is apparent from the literature. Whilst Calvert and Smith (1955) remarked that re-expansion of the affected lung usually occurred within two months after the onset of illness, present data show that 26 cases (14 per cent) of the 240 patients had an illness of 3 months or more.

This extreme delay in recovery, not to mention permanent impairment of pulmonary function, is explained by a close study of the sequential adverse effects of intrapleural fibrin and blood-clots. Intrapleural blood-clots may occasionally occur despite apparently early and adequate attempts at aspiration (Beatty and Frelick, 1952; Author's Case 5) and clots have frequently been observed at autopsy and at operation. Presumably, in these patients, massive haemorrhage and more complete associated pulmonary collapse have impeded efficient agitation and defibrination.

The natural history of haemothorax has been reviewed (Sellors, 1945; Harold, 1951) and will only be briefly referred to here. The natural exudative response of the pleural membranes to the irritant effects of blood and its products is manifest (Sellors, 1945) by a large serous effusion, rich in fibrinogen. Secondary clots may now appear.

From the pleural surfaces, coated with fibrin and clots, angioblastic and fibroblastic proliferation later extend into the clot. This process of organising haemothorax commences after one week and by one month (Ross, 1952) the layer contains well-formed adult fibrous tissue. The visceral pleura itself remains as a thin, translucent membrane (Moore, 1949). This fibrothorax, sometimes accompanied by loculation, is disabling (Smithy, 1943) and is characterised by impaired lung-expansion from a flattened hemithorax of approximated ribs, with secondary scoliosis, described as the "frozen" or fixed chest (Sellors, 1951).

Studies on the physio-pathology of respiratory function in relation to the operation, pulmonary decortication (Gordon et al., 1945; Wright et al., 1949; Carroll et al., 1952) have re-affirmed the factors concerned in the production of so-called "inexpansible lung" (Langston et al., 1947). Pulmonary collapse is maintained by:- (1) the pleural contents (air, blood, blood clot, fibrin and, rarely, pus).

(2) the "peel" or involucre of fibrin on the pleural surfaces.

(3) pleural adhesions between the lung, chest-wall, diaphragm, mediastinum and pericardium. The adhesions develop simultaneously with the "peel." In short, "the lung is compressed by the pleural contents, imprisoned by the peel, and restrained by pleural

adhesions" (Williams, 1950).

From the literature on idiopathic haemopneumothorax, the frequency of fibrothorax, taken in the broader sense to include organising haemothorax and well-marked pachypleuritis, appears to be at least 20 per cent (Section 2). However, with pulmonary decortication, to which two-thirds of these patients were subjected, this incapacitating complication need no longer be permanent. Indeed, with more energetic earlier treatment, to be discussed later, this complication, and hence the need for this operation, should seldom arise. Other complications are rare. For instance, only 8 cases of empyema (Munos-Monatorio, 1936; Korol, Case I, 1936; Stockler, Case 4, 1947; Kastl, Case 2, 1952; Garcia et al., Cases 1 and 2, 1953; Mezzera and Aguiar, 1954; Agnew, 1955) are on record. This infrequency is even more surprising since blood is an excellent culture medium and many patients have had multiple thoracenteses. A third complication, calcification of the pleura has twice been reported (Milhorat, 1937; McRae, Case I, 1939), at intervals of 6 and 10 years respectively from the initial haemothorax.

Investigations.

Laboratory Investigations.

Bacteriological.

Routine investigation was usually undertaken to exclude tuberculosis, as far as possible, by examination of the sputum and pleural fluid. In about a quarter of the cases, the fluid was also injected into guinea-pigs. Sputum was frequently unobtainable and resort to gastric lavage was sometimes adopted.

Haematological.

These investigations revealed several interesting features; notably, the rarity of severe anaemia, the frequence of leucocytosis, and the occasional occurrence of eosinophilia in the blood and/or pleural fluid. Haemoglobin values for peripheral blood were available for 94 of the 240 patients and, for present purposes, only the lowest recorded value in each patient is considered. Yet, in only 11 cases (9 per cent) were values of less than 50 per cent haemoglobin obtained. The lowest level was 28 per cent (Salaris, 1938). The patient of McGown (1954) is of special interest, since the value was only 29 per cent, following the transfusion of 7 pints of blood. The overall low incidence of severe anaemia contrasts with the extreme frequence (85 per cent) of associated signs of shock. Indeed, broadly speaking, these signs vied in frequency and severity with those encountered in patients who are hospitalised for haematemesis from peptic ulcer.

Nevertheless, there are certain obvious explanations for this apparent discrepancy:- (1) the haemoglobin values under review represent mainly

those for the non-fatal group. Indeed, there are only available haemoglobin levels for 5 (Rossel, 1935; Louria, 1938; Arst et al., 1950; Grundi, 1953; Calvert and Smith, 1955) of the 19 fatal cases. However, in only one patient (Louria, 1938) of these 5 was the value less than 50 per cent (i.e. 40 per cent).

(2) in many of the more recently reported cases, the haemoglobin value coincided with the pre-haemodilution phase and, through early blood-transfusion, the lowest haemoglobin level remains unknown.

(3) haemopneumothorax, in contradistinction to haematemesis, produces an early impairment of cardio-respiratory function from the triad of intense pain, associated restricted respiratory excursion and the mechanical effects of intrapleural air and blood. Thus shock, not entirely due to haemorrhage is manifest as an early event.

Leucocyte counts on the peripheral blood were recorded in 84 of the patients with idiopathic haemopneumothorax. There was leucocytosis, polymorphonuclear in type, in 56 of these patients, 28 of whom had values between 15,000 and 35,000 per cu. mm.. Normal values were found in 25 patients, while the remaining 3 patients had unexplained leucopenia. Since empyema rarely complicates idiopathic haemopneumothorax and since a normal leucocyte count is the rule in patients with pneumothorax, these striking leucocytic responses must be attributed to the haemothorax. This view is supported by the fact that two of the three recorded leucocyte counts in idiopathic haemothorax (Table13) exceeded 15,000 per cu. mm..

Frank eosinophilia, i.e. where the percentage of eosinophiles in the differential count was 4 or more, was recorded in 8 of the patients with

leucocytosis (Rist and Worms, 1940; Franklin, 1945; Groen and Godfried, 1948; Solovay, 1949; Walton, Case 2, 1949; Deiss et al., Case I, 1950; Hyde and Hyde, Case 6, 1951; Fusia and Cook, Case I, 1952; Case 5 of the Author's series) and in 6 patients with a normal total leucocyte count (Troisier et al., 1937; Salaris, Cases I and 2, 1938; Hyde and Hyde, Case 3, 1951; Mezzera and Aguiar, 1954; Author's Cases 2 and 4).

Since the pleural fluid is seldom examined for evidence of a concomitant eosinophilia it is not known if an eosinophilia in the peripheral blood is always associated with a similar finding in the pleural fluid. The converse is certainly not treu (Meo and Tallone, 1954). Marked eosinophilia has been observed in the pleural fluid (74%, Troisier et al., 1937; 33%, Staffieri, 1943; 43%, Franklin, 1945; 44%, Groen and Godfried, 1948) associated with lower values (5%, Troisier et al.; 12%, Groen and Godfried), in the peripheral blood. For the latter, the highest recorded value is 18 per cent (Deiss et al., 1950).

The fact that higher values obtain in the pleural fluid suggests a local cause, perhaps some degradation product of the sangineous effusion. Partial absorption to the circulation of this factor might then evoke an eosinophilic response in the peripheral blood. The explanation of this transient eosinophilia is obscure since, in other conditions which are often associated with transient eosinophilia, there is usually an allergic basis. However, this explanation, if applied to idiopathic haemopneumothorax, would invoke the participation of an autogenous allergen, which is hardly conceivable. If it is argued that the observation is the result of an artefact, this argument could not be extended to the eosinophilia in the peripheral blood. Such theorisation on the cause of the local

eosinophilia even ante-dates Grabfield's description (1921) of "eosinophilic pleurisy" and remains sub judice (Chapman, 1955).

Erythrocyte sedimentation-rates were rarely recorded (31 cases) during the height of the illness. The values were raised over a wide range but were normal in one third of these patients.

Radiological and Thorascopic Investigations.

Radiological.

The routine radiograph of the chest in idiopathic haemopneumothorax reveals, apart from varying degrees of mediastinal displacement and pulmonary collapse, a hydropneumothorax with the fluid level situated usually between the fourth and eleventh ribs posteriorly. Whilst the relative proportions of air and fluid vary considerably, both components are almost invariably recognisable in those patients who require admission to hospital. It has been pointed out (Greenberg, 1934; Wilson, 1937) that recognition of the air might be overlooked in minimal cases of spontaneous pneumothorax, unless a film were taken in expiration. For the reason stated, this diagnostic difficulty is inapplicable to the patients under present consideration.

Pleural adhesions, usually apical, have occasionally been seen radiologically on the same side as the haemopneumothorax (Hansen, Case 3, 1949; Hyde and Hyde, Case II, 1951; Fusia and Cook, Case I, 1952; Borrie, Cases I and 3, 1953; Garcia et al., Case I, 1953) or on the opposite side (Patino-Mayer and Pataro, 1934). Similarly, adhesions have not been frequently observed (Ornstein and Lercher, 1942; Taschman, 1944) in patients with idiopathic pneumothorax and, if present, should lead to a careful search for evidence of tuberculosis (Cohen and Kinsman, 1946). In this respect, Rusby (1952) maintains that adhesions are associated more frequently with pulmonary tuberculosis than with idiopathic pneumothorax.

After re-expansion of the lung in idiopathic haemopneumothorax, apical bullae or blebs have sometimes been reported (Castex and Mazzei, 1935; Troisier et al., 1937; Franklin, 1945; Bernstein et al., Case I, 1946;

Cuningham, Case I, 1950) on the affected side. The sole and, indeed, plausible interpretation is that bullae are often multiple. Those observed radiologically, after an incident, represent intact "survivors." This explanation has the merit of providing a rational basis, applicable also to idiopathic pneumothorax, for previous or subsequent pneumothoracic episodes. As to the frequency of similar radiological observations for idiopathic pneumothorax, opinions differ. Kjaergaard (1932), who was obviously keen to determine the incidence to link up with collateral evidence in his comprehensive study, observed these lesions on X-ray study in only one of his series of 51 patients. His case (No. 42) merits recall as there were visible bullae in relation to all of three attacks of pneumothorax. By sharp contrast, however, Gordon (1936) reported that he had detected bullae radiologically during the recovery of each of five patients. He boldly concluded that all cases of idiopathic pneumothorax were due to rupture of these bullae or blebs. Verification of this view must await further intense study. Nevertheless, it has partial support. For instance, Brock (1948) determined that 50 of his series of 71 patients with recurrent or chronic idiopathic pneumothorax had radiographic or thorascopic evidence of generalised or localised bullous emphysema. Similarly, Dubose and associates (1953) reported that a careful radiological review of their series of 75 patients with idiopathic pneumothorax revealed that 57 (78 per cent) had evidence of localised or diffuse bullous emphysema, usually involving the upper lobes.

Cystic appearance of the lung has rarely been reported (Moser et al., Case 4, 1951; Calvert and Smith, Case I, 1955) during X-ray study of

patients with idiopathic haemopneumothorax. Hansen (1949), however, first reported this association, which he observed per-operatively in two (Cases 2 and 3) of his three patients, while Biancanala (1953) added a similar observation. It is of interest to note that spontaneous pneumothorax can complicate "congenital" cystic disease of the lung (Rusby, 1952).

Finally, as previously mentioned, residual pleural thickening, of varying degree and persistence, is a frequent sequel to haemopneumothorax. Extreme degrees, constituting fibrothorax are well illustrated by Williamson (1917), Arroyo (1941), Schneider and Reissman (1945) and Grimaldi (1951). For example, Arroya (1941) reported that later radiographs of his patient showed "diffuse pachypleuritis with discrete retraction of the thorax," while Schneider and Reissman's case exhibited "pleural thickening with marked retraction of mediastinal structures."

Thoracoscopy.

Thoracoscopy has facilitated the identification of pleuro-pulmonary lesions in patients with pneumothorax (Castex and Mazzei, 1936 and 1937; Brock, 1948; Deucher, 1950; Hughes et al., 1951; Morrengoni et al., 1955). While the actual site of irruption of air from the lung has rarely been visualised, subpleural blebs, often multiple and intact, have frequently been detected. For example, Morrengoni and colleagues (1955) observed these in 13 out of 20 of their series of 80 patients with idiopathic pneumothorax, so examined. However, it is otherwise with idiopathic haemopneumothorax. Here, thoracoscopy has rarely aided in the detection of the source of bleeding, (Deucher, 1950; Harold, 1951;

Fry et al., 1955). For this reason, when it is essential to identify the bleeding point to effect haemostasis, open thoracotomy is the preferred procedure. The evacuation of blood and blood-clot can then also be conveniently and simultaneously carried out.

These remarks require slight qualification in view of the thoracoscopic observations of Sattler (1954). His patient, a man aged 42 with a left-sided spontaneous haemopneumothorax, is not included in the analysis of patients with idiopathic haemopneumothorax on the grounds of insufficient data. The patient's chances of recovery, as judged by the description, appeared to depend on an emergency thoracotomy. However, Sattler commendably completed investigation and treatment by thoracoscopy under local anaesthesia. His observations are instructive, if not unique. He noticed that the lung, tethered by adhesions, protruded above the level of the fluid. There was a large quantity of "blood," of which $1\frac{3}{4}$ litres were aspirated, leaving visible blood-clot and a fibrin deposit on the diaphragmatic pleura. Closer inspection revealed that the partially collapsed lung was anchored to the thoracic wall by two membranous adhesions, of which the upper one was bleeding and showed tears while the other was intact. He electro-coagulated the bleeding point and severed the adhesions by diathermy. A catheter, connected to a motor pump, was left in situ. Next day, the X-ray film revealed complete re-expansion of the lung. It was noted with surprise that the total duration of illness approximated two months, but no explanation was given.

Treatment.

Medical treatment is based on the assumption that the haemorrhage will cease within the first day or two. This is the sequel in about 85 per cent of cases. When confronted with profuse or protracted haemorrhage, medical treatment alone is unavailing. Such cases have recently been "rescued" by the combination of adequate blood transfusion, and emergency thoracotomy to secure haemastasis. Bearing in mind that treatment must be individualized, the general therapeutic aims may be summarised as follows:-

(1) The adoption of general measures, such as, strict bed-rest; administration of morphine; inhalation of oxygen; use of anitbiotics intrapleurally, and possibly systemically, where repeated thoracenteses are undertaken; frequent recording of pulse-rate and blood-pressure, during phases of danger, to aid the assessment of the need for surgical intervention.

(2) The prevention of exsanguination and syncope by early and adequate, yet judicious, blood transfusion. In any event, this constitutes the preliminary treatment to operation for the arrest of haemorrhage.

(3) The promotion of rapid re-expansion of the lung by prompt and energetic aspiration of blood and air to prevent the harmful consequences of blood retained in situ. The minimal aim is to convert the haemopneumothorax to a pneumothorax. For a tension haemopneumothorax, under-water seal drainage or equivalent is obligatory. A pneumothorax refill apparatus can conveniently be used in reverse. It has the advantages of providing measurements of the volume of air extracted and of recording the intrapleural pressures.

(3) Exceptionally, the facilitation of complete aspiration of fluid by

The intrapleural instillation of fibrinolytic enzymes. This is especially applicable to patients with suspected intrapleural blood-clots. This procedure is only justifiable when haemorrhage has ceased and when one is prepared to carry out further aspiration about 6 to 8 hours later, to reduce the unpleasant constitutional effects. More detailed consideration of these therapeutic principles will follow.

At this juncture, it is profitable to review concisely the therapeutic history of idiopathic haemopneumothorax. The earlier arguments advanced to support a policy of minimal interference were motivated by the risk of infection from multiple aspirations and an implicit reliance on a high intra-thoracic pressure and a low blood-pressure to stop the haemorrhage. Kjaergaard (1932) cautioned against the use of blood transfusions and Hopkins (1937) against frequent aspirations. Louria (1938) regarded thoracentesis as "a double-edged sword." He contended that it should be reserved for the correction of mediastinal displacement, since complete evacuation might result in further bleeding and open up a previously sealed broncho-pleural fistulous track. Hartzell (1942) shared this view on thoracentesis but considered that occasional blood transfusions, in small amounts, might be beneficial. The sanguine view of Payn and Lief (1945) is surprising. They wrote, "the disease usually runs a favourable course, resulting in absorption of blood and air; treatment should therefore be conservative." These authors, however, discerningly added" too little is known about the late results of patients who recovered." As late as 1949, Dorset and Terry recommended "the initial diagnostic aspiration of a small quantity of fluid and that only after several days of watching should small amounts of blood be removed every few days with partial replacement by air."

By contrast, however, certain earlier authors (Wilson, 1937; Maxwell, 1938) appreciated the wisdom of the thorough aspiration of the pleural contents. They differed slightly on the question of the earliest safe time to proceed with therapeutic aspiration. While Maxwell suggested the very safe interval of one week, Wilson inferred that a delay of only two to three days would be opportune. He averred that the failure of blood withdrawn by thoracentesis to clot in vitro indicated that the bleeding had ceased at least several hours previously, so that it was then safe and desirable to evacuate all the blood. Coope (1948) pointed out that, since the haemorrhage usually arises from the parietal end of a torn adhesion, re-expansion of the lung per se will not cause further haemorrhage, but could re-open a sealed perforated bulla. He, therefore, advocated leaving the blood in the pleural cavity for two days before engaging in therapeutic aspiration. He, however, recommended air-replacement when large volumes of fluid were aspirated.

In 1945, several authors (Barrett; Sellors; Simpson) were unanimous, from their war-experience in the management of chest injuries, that the method of choice for the early treatment of haemothorax was adequate aspiration of blood and air, unaccompanied by air-replacement. Indeed, the latter measure is to be deprecated (Sellors, 1945; Moore, 1946). It is irrational and unnecessary (Nalls and Matthews, 1949; Deiss et al., 1950; Beatty and Frelick, 1952; Fusia and Cook, 1952; Kastl, 1952), moreover, it delays the desired rapid re-expansion of the lung. There is no evidence (Barrett, 1945; Sellors, 1945) that decreased intrapleural pressure favours renewed haemorrhage.

From similar war-time studies, Lush and associates (1944) drew

attention to the late morbidity from clotted haemothorax. They urged that blood-clots must be evacuated and that pulmonary decortication is the appropriate measure to restore adequate respiratory movement. Reports of the successful adaptation of this operation to such complications of spontaneous haemopneumothorax as pleural deposits of fibrin, clotted haemothorax and organizing haemothorax began in 1948 (Elrod and Murphy).

From 1951 onwards two further dissociated therapeutic advances, each for selected cases, became firmly established procedures, i.e. the operation of emergency thoracotomy for haemostasis and secondly, the intra-pleural injection of fibrinolytic enzymes to aid in medical debridement of the affected pleural cavity.

The institution of emergency thoracotomy was epic in the history of idiopathic haemopneumothorax. Whereas, in the preceding era there were 17 deaths (12 per cent mortality-rate) in the 140 reported cases, only 2 of the 100 subsequently reported cases died. Moreover, neither of these 2 fatal cases reached operation. This surgical success is even more spectacular, since all of the 19 cases (Table11), so treated, survived. Indeed, their average duration of illness was less than that of contemporary patients, who were not subjected to operation. Moreover, the former, as a group, had precarious pre-operative clinical states comparable to those reported in the earlier fatalities.

The use of fibrinolytic enzymes for pleural debridement and the liquefaction of blood-clots has proved efficacious in selected cases of haemothorax (Sherry et al., 1950; Read and Berry, 1950; Carr and Robbins, 1951; Miller et al., 1951; Ross, 1952; Jones and Bigham, 1953; Calvert and Smith, 1955). Combinations of streptokinase (S.K.) and streptodornase (S.D.) have usually been employed but more recently other workers

(McCroskey and Hardin, 1953; Delannoy and Ribet, 1953) have used intra-pleural injections of trypsin with apparent success.

The general principles of treatment will now be amplified.

(1) General measures:

Those applicable to the average case have been described. For those with moderate anaemia, haematinics will hasten recuperation. Physiotherapy, mainly the use of breathing exercises and of "blow bottles" may aid complete lung expansion post-operatively.

(2) Prevention of Exsanguination and indications for emergency thoracotomy.

Despite earlier remonstrations against the use of blood transfusions, the view that properly administered blood does not incite further haemorrhage is well supported. Moreover, in patients with massive intra-pleural haemorrhage, blood transfusion enables them to withstand such procedures as thoracentesis to correct mediastinal displacement and emergency thoracotomy. Indeed, Williams and colleagues (1954) refer to a patient who received 4 litres of blood and one pint of plasma before and during this operation, while case 5 in the present series was transfused 6 litres of blood under similar circumstances. In William's patient, the volume of blood administered no more than equalled the intra-pleural blood-loss. There are occasions when fairly rapid administration is indicated. Certain authors (Knight and Oelrich, 1949; Nalls and Matthews, case I, 1949) have in emergency even auto-transfused patients with the "blood" obtained from thoracentesis. Borrie (1953) described the concurrent use of blood transfusion at two different sites in the same patient. In case 3 of the present series, when syncopal attacks appeared

prior to her demise, we were about to commence intra-arterial blood transfusion.

Patients with minimal evidence of shock, irrespective of the size of the effusion, usually make a rapid recovery with or without resort to blood transfusion, provided they are brought under early close observation and have adequate thoracentesis. Moser and colleagues (1951) described four cases responding to these measures, while Hyde and Hyde (1951) had similar success in 11 our of 12 patients. His remaining case required pulmonary decortication. In the author's first patient with syncopal attacks and only slight shock on admission, complete aspiration, without blood transfusion, sufficed. The remaining patients also had blood transfusion.

The indications for emergency thoracotomy await more precise definition. The broad and brief criteria of Williams and colleagues (1954) are tentatively useful. They suggested:-

(1) An initial erythrocyte count of 2,500,000 per cu. mm. or less.

(2) Haemorrhage or shock requiring the transfusion of more than 500 cc. of blood eight-hourly.

However, the author recommends the following more precise and practical indices:-

(1) An initial haemoglobin value of 50 per cent or less, in the peripheral blood, provided there are still signs of shock.

(2) An initial period of four or, at most, six hours for close observation of the response to blood transfusion and therapeutic thoracentesis. If, following the transfusion of 3 pints of blood in this period, there are no clear signs of improvement, including the blood pressure, immediate thoracotomy is indicated. Even although the blood

pressure has reverted to normal, if a tachycardia exceeding 110 per minute persists, this operation is similarly imperative.

(3) The appearance of signs of tension haemopneumothorax, coupled with inconclusive signs that the haemorrhage has ceased. Under water seal drainage should be instantly set up while awaiting thoracotomy. Operation provides the opportunity to inspect and treat the cause of each of these potentially fatal hazards simultaneously. It is pertinent to mention that Clyne and Hutter (1955) take a more extreme view. They recommend "emergency thoracotomy, after resuscitation, as the treatment of choice for all cases" and add "the real risk in these cases lies not in operating but in not operating." This is, however, a counsel of unnecessary perfection and of impracticability.

(3) Rapid Pulmonary Re-expansion.

In idiopathic pneumothorax the perforation in the lung is sealed after 2 to 48 hours (Fry et al., 1955), so that re-inflation should then commence automatically. In the idiopathic types of haemo-pneumothorax, however, rapid re-inflation is retarded by the other pleural contents, i.e. blood and its products. Thoracentesis should be commenced immediately and, if necessary, should be continued daily so that a fairly empty pleural cavity is obtained within the first 3 to 4 days. The risk of thoracentesis producing a continuation or recurrence of haemorrhage is slight. No haemostatic temponade-like effect from the pleural contents is to be expected by a more conservative policy since the haemorrhage is almost always from the parietal end of a pleural adhesion fixed to the chest wall. In contrast to pulmonary haemorrhage, factors tending to promote or maintain pulmonary collapse are, for this reason,

ineffective haemostatics.

(4) Use of Fibrinolytic Agents.

S.K. liquefied blood-clots and fibrinous deposits, thus exerting mainly a fibrinolytic action (Christensen and MacLeod, 1945; Tillet, 1952), while S.D. acts only on the nucleo-proteins of the degenerate cells in the exudate (Tillet et al., 1948). Neither enzyme is harmful to healthy cells, while both enhance phargocytosis (Johnson, 1950), thus acting synergistically with antibiotics. An important part of the procedure is the aspiration of the lysed fluid and residual enzyme solution several hours later. Further delay in re-aspiration causes unnecessary discomfort to the patient.

Three reactions may accompany the use of S.D. and S.K. instillations:-

(1) A local effusion with living polymorphonuclear leucocytes, i.e. the beneficial reaction.

(2) A pyrogenic response, with the initial rise of temperature commencing in about 6 hours and becoming maximal (105° F) in 24 hours, if the enzymes and lysed material are not evacuated sooner. The constitutional symptoms, i.e. headache, nausea, malaise, orthalgia and, rarely, rigors are often relieved by aspirin. These pyrexial reactions may occasionally confusingly suggest that further haemorrhage is occurring.

(3) Allergic reactions, especially involving the peri-orbital tissue may rarely appear from the antigenic action of S.D. or S.K.

Speaking practically, a freshly prepared solution of both these enzymes is injected into the pleural cavity towards the end of an

aspiration. This procedure may need repetition for maximal therapeutic effect. Caution is required during fibrinolytic enzyme therapy to avoid an injection directly into a blood-vessel. Moreover, its use should be delayed till the beginning of the second week of illness to minimise the risk of re-opening the vessel, which had caused the haemorrhage, or the sealed-off broncho-pleural fistula.

There are two clear-cut indications for this treatment, i.e. clotted haemothorax and dense intra-pleural fibrin deposits. Its use may prevent subsequent decortication (Read and Berry, 1950; Beatty and Frelick „ 1952) although the former is no substitute for the latter (Carr and Robbins, 1951).

The efficacy of this treatment in idiopathic haemopneumothorax has not been fully assessed. Reported observations (Read and Berry, 1950; Grundi, 1953; Jones and Bigham, 1953; Mezzera and Aguiar, 1954; McGown, 1954; Calvert and Smith, case 2, 1955; Weiner, 1955) are each based on one case. Three of these reports (Read and Berry; Jones and Bigham; Calvert and Smith) acclaimed it valuable while in three cases (Mezzera and Aguiar; Weiner; McGown) it did not obviate decortication. In the remaining case (Grundi) other fatal and unrelated complications supervened and precluded any assessment.

Finally, recourse to late decortication should become increasingly unnecessary with a wider appreciation of its prevention by early and adequate thoracentesis, combined, where necessary, with fibrinolytic enzyme therapy. Pulmonary decortication is of proven value. Rarely other complications, such as further haemorrhage, air embolus or broncho-pleural fistula (Deucher, 1950) may attend its use.

Summary and Conclusions.

Summary.

The entire literature on idiopathic haemopneumothorax, comprising 240 well-documented case-reports, has been surveyed, analysed and discussed. The author's eight illustrative cases, which are representative of the varied problems, have been separately presented and discussed. For comparative purposes, twelve acceptable published cases of idiopathic haemothorax have been critically considered.

With the intent of characterising the entity, idiopathic haemopneumothorax, a section has been entirely devoted to statistical analysis, in which the corresponding information on idiopathic pneumothorax has been collaterally scrutinised. These data, which are too extensive for reproduction here, can be conveniently obtained from consultation of Section 2.

Available information on the aetio-pathogenesis, clinical features, simulations and complications, laboratory and radiological observations, and treatment has been fully outlined. Certain recent therapeutic applications for selected cases, i.e. emergency thoracotomy to secure haemostasis; pulmonary decortication for clotted haemothorax and fibrothorax; and firbinolytic enzyme-therapy, have been reviewed.

Conclusions.

(1) Idiopathic haemopneumothorax is an uncommon complication of the not uncommon condition, idiopathic pneumothorax. Indeed, about 5 per cent of idiopathic pneumothoraces are complicated by haemothorax.

(2) The hypothesis of an identical aetiology for the idiopathic types of haemopneumothorax, haemothorax and pneumothorax is strongly supported by

the evidence presented. Similarly, the pathogeneses are allied. Some cases of idiopathic haemothorax may, in reality, have been instances of haemopneumothorax.

(3) Unquestionably, there are two aetiologies, a distant tuberculous and a non-tuberculous type. An over-ready identification with past pulmonary tuberculosis is rebuffed by the fact that about 50 per cent of patients with idiopathic types of haemopneumothorax and pneumothorax exhibit negative responses to standard tuberculin-tests. Nor do non-tuberculous pulmonary infections, as judged by available information, constitute an adequate uniform explanation.

It appears irrefutable that the basic lesions, i.e. subpleural bullae, apical scarring and pleural adhesions, are relics of pulmonary infection and it can be safely concluded that both distant tuberculous and non-tuberculous infections are progenitors of an, as yet, undetermined relative proportion of the idiopathic types of haemopneumothorax, haemothorax and pneumothorax.

(4) Two main mechanisms in the production of haemopneumothorax have been established.

(a) an initial pneumothorax from a ruptured subpleural bleb, with subsequent haemorrhage from stretched and torn pleural adhesions. This explanation, alone, has the merit of satisfactorily accounting for undoubted cases of delayed intrapleural haemorrhage. Here, as in the cases of idiopathic haemothorax, haemorrhage arises mainly, if not entirely, from the parietal tag of the ruptured adhesion.

(b) the simultaneous irruption of air and extravasation of blood from a torn emphysematous bulla, without the invocation of torn adhesions.

(5) The indications for emergency thoracotomy have been tentatively defined:-

(a) an initial haemoglobin value of 50 per cent or less, in the peripheral blood, provided there are still signs of shock.

(b) an initial period of four or, at most, six hours for close observation and therapeutic thoracentesis. If, following the transfusion of three pints of blood, in this period, there are no clear signs of improvement, including the blood pressure, immediate thoracotomy is obligatory. Even although the blood pressure has reverted to normal, if a tachycardia exceeding 110 per minute persists, this operation is similarly imperative.

(c) the appearance of signs of tension haemopneumothorax, coupled with inconclusive signs that the haemorrhage has ceased.

Early and adequate aspiration of intrapleural "blood" constitutes the most important prophylaxis of the disabling complication, fibrothorax, and minimises the total duration of illness. The minimal aim is the conversion of the haemopneumothorax to a pneumothorax.

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AN ANALYTICAL REVIEW OF SPONTANEOUS HAEMOPNEUMOTHORAX

BY

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AN ANALYTICAL REVIEW OF SPONTANEOUS HAEMOPNEUMOTHORAX

BY

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In 1900 Newton Pitt wrote, "There is no reference to spontaneous haemopneumothorax in the Index Catalogue of the U.S. Library, nor is it discussed in any of the standard treatises on medicine, in either English, French, or German." It has remained little discussed even in specialized textbooks although about 150 cases, including the present three, have been recorded. Both spontaneous haemopneumothorax and spontaneous haemothorax are complications of spontaneous pneumothorax, of which careful follow-up (Kjaergaard, 1932; Perry, 1939) has failed to reveal any correlation with tuberculosis. It is also probable that spontaneous haemothorax may at times have no coexistent or immediately antecedent spontaneous pneumothorax.

Both Pitt (1900) and Rolleston (1900) realized that spontaneous haemopneumothorax was potentially serious. Each reported a fatal case. Noteworthy contributions to this subject include those of Hyde and Hyde (1951) with 12 case reports, Ross (1952) with six, Louria (1938) with five, Moser (1951) with four, and Hopkins (1937), Hartzell (1942), Hansen (1949), Harrell (1949), Nalls and Matthews (1949), Rottenberg and Golden (1949), Eidinger and Rubin (1952), and Borrie (1953) each recording three cases. This subject has been briefly reviewed (Jones and Gilbert, 1936; Hopkins, 1937; Hartzell, 1942; Helwig and Schmidt, 1947; Nario, Bermúdez, and Espasandin, 1949; Deucher, 1950; Fusia and Cook, 1952; and Eidinger and Rubin, 1952). Some authors (Hartzell, 1952; Helwig and Schmidt, 1947; Cuningham, 1950) have also analysed available necropsy data. The literature indicates an overall mortality rate of 15%, although a figure of 25% has been determined previously (Myers, Johnston, and Bradshaw, 1951; Ross, 1952). All these fatality rates are undoubtedly artificially high as the more serious or fatal cases are specially reported, while the milder cases are frequently unrecognized or unrecorded. Most of the fatalities have been relatively immediate, occurring in the first two days.

Only 10 cases (Hopkins, 1937; Hartzell, 1942; Crimm, 1948; Dorset and Terry, 1949; Deucher, 1949; Hansen, 1949; Harrell, 1949; Knight and Oelrich, 1949; Freund and Hicks, 1953; Towson, 1954) have been reported in women. Both sides of the chest have been equally involved. Recurrence is quite exceptional, but has been authenticated (Repetti, 1940; Rusby, 1947), while an earlier or later spontaneous pneumothorax has occasionally been described (Rist and Worms, 1940; Hartzell, 1942; Goldman and Roth, 1944; Deucher, 1950).

Our three illustrative cases, treated in the past two years, refute its rarity. Chapman (1950) claimed that, in spontaneous pneumothorax, free intrapleural haemorrhage occurs at the onset of pulmonary collapse in about one in eight patients. This frequency is not evident in the larger series of spontaneous pneumothorax. Rottenberg and Golden (1949), for instance, reported that three of 97 consecutive cases of simple pneumothorax had massive haemothorax from which one died, while Hyde and Hyde (1951), in a similar series of 112 cases, found that five of these patients had spontaneous haemopneumothorax.

This condition ranks with other forms of internal haemorrhage as a strict emergency. Indeed, our third patient died while too ill for exploratory thoracotomy.

CASE REPORTS

CASE 1.—J. H., a previously healthy clerk aged 26, was admitted on May 28, 1952, with moderate breathlessness and a sharp inspiratory pain, radiating from the left lower chest behind the left shoulder. This pain had appeared abruptly on the previous evening while he was walking home from work. Despite distress, he reached home unaided and immediately went to bed. He experienced discomfort on attempting to lie on either side. Next morning he had several syncopal attacks, during one of which he was unconscious for five minutes. As movement increased his pain, he remained stock-still. His doctor diagnosed a left-sided pneumothorax, but considered that there was associated acute intercostal fibrositis

as the intercostal muscles splinted the left hemithorax. The past history disclosed no respiratory disease. Indeed, a routine chest radiograph carried out three months previously was normal. There was no family history of tuberculosis.

Examination revealed a pale, anxious patient of fairly good physique with minimal venous congestion. The respirations were shallow and their rate was 30 per minute. The salient signs were confined to the respiratory and cardiovascular systems. The trachea was displaced to the right. The remaining respiratory signs were on the left side, where there was fullness, greatly restricted movement, and a very dull percussion note in the base, mid-zone, and axilla. In these sites tactile vocal fremitus, breath-sounds, and vocal resonance were absent, but aegophony was heard both in the scapular region and anteriorly. The vascular signs included tachycardia (120/minute), an easily compressible but regular pulse, and hypotension (115/70 mm. Hg). The apex beat could not be located, while the cardiac sounds were pure but faint.

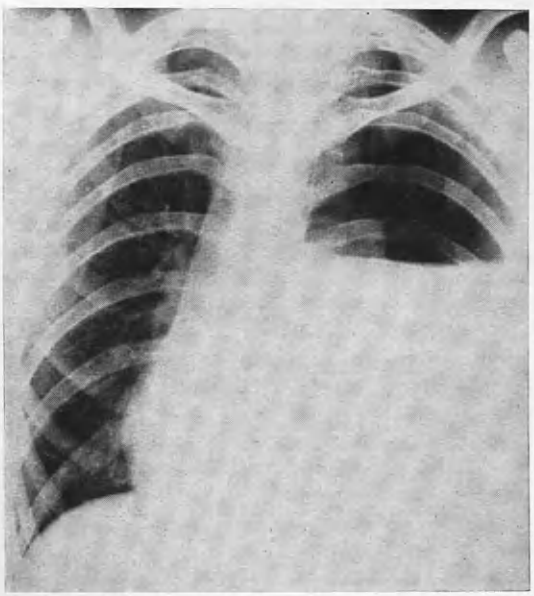

FIG. 1.—Case 1. Radiograph on May 28, 1952, on admission to hospital. Large left-sided pleural effusion before thoracentesis.

Immediate investigation showed a haemoglobin level of 56% with a normal total and differential white cell count, and a chest radiograph (Fig. 1) revealing a left-sided pneumothorax with effusion and ipsilateral apical pleural thickening. The electrocardiogram was normal apart from ST segment depression in the limb leads, suggesting a relative coronary insufficiency. An hourly pulse chart was begun and blood was cross-matched for emergency. Morphine sulphate ($\frac{1}{4}$ gr.) was injected before thoracentesis, which yielded $5\frac{1}{4}$ pints of sanguineous fluid with a haemoglobin value of 62%. Almost complete re-expansion of the lung promptly followed. The next day the patient was

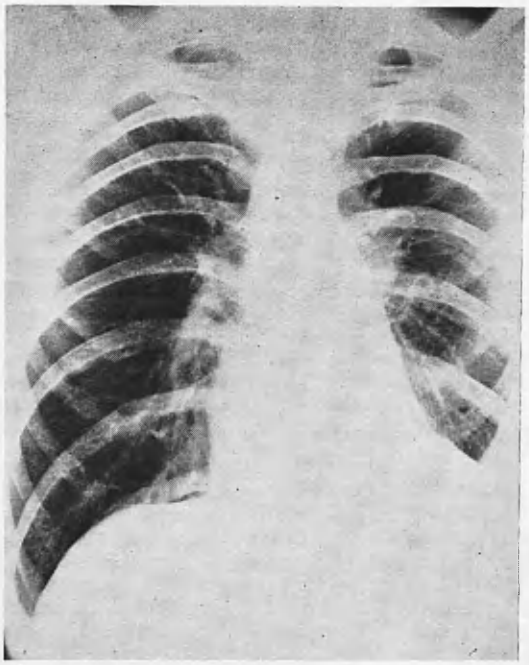

FIG. 2.—Case 1. Radiograph on June 16, 1952. Almost three weeks after the single aspiration of $5\frac{1}{4}$ pints of haemorrhagic effusion. The left dome of the diaphragm is raised and a small effusion persists.

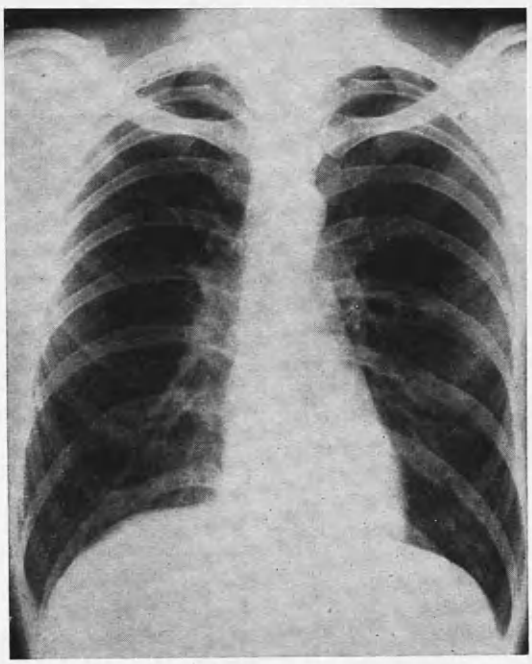

FIG. 3.—Case 1. Radiograph on January 8, 1953, six months after leaving hospital. Lung fields now normal apart from cystic disease in the left mid-zone.

very comfortable with a pulse rate of 90 per minute, blood pressure of 115/75 mm. Hg, and the trachea central. Progress was now uneventful and the chest radiograph of June 16, 1952 (Fig. 2), was almost normal. Five weeks after admission he was discharged home feeling fit, having gained 8 lb. in weight, and with a haemoglobin value of 84% following treatment with ferrous sulphate (0.2 g.), ascorbic acid (50 mg.), and " casilan " (15 g.) each thrice daily. No blood transfusion was given throughout.

Two months later he returned to work, having gained a further 7 lb. in weight. He has since been followed up as an out-patient at three-monthly intervals. The E.S.R.s and chest radiographs, apart from cystic disease in the left mid-zone (Fig. 3), have remained normal. There has been no evidence of tuberculosis, including examination of the sanguineous fluid directly and on culture.

CASE 2.—J. G., a robust foundry-worker, aged 29, was admitted here on August 8, 1952, with a history that 10 days previously he experienced a sudden severe pain in the left side of the chest, especially posteriorly, but also felt in the left upper abdomen. He remained in bed under his doctor's supervision. The pain subsided after one day, but four days before admission it recurred with its previous severity. It was aggravated by deep breathing and coughing. He now felt listless and breathless and had an irritating non-productive cough. His doctor stated that there had been no pyrexia.

The past history included no illness of note, and, in particular, no chest illness or symptoms, but the family history revealed that five years ago a sister had spent a year in a sanatorium.

On physical examination he was pallid, but showed no venous congestion or distress. The respiratory system showed the classical signs of a moderate left-sided pleural effusion, including displacement of the trachea to the right. The pulse was regular at a rate of 82 per minute and of moderate volume, while the rest of the cardiovascular system was also normal, apart from a rather low diastolic pressure (130/60 mm. Hg). The other systems were normal.

Initial investigations revealed a haemoglobin level of 88%, a normal total and differential white blood count, and an E.S.R. of 42 mm. (Westergren) in one hour. The radiograph of the chest showed a left-sided pneumothorax with effusion and associated partial collapse of that lung. Immediate diagnostic thoracentesis produced 8 oz. of fluid resembling venous blood. This showed 59% haemoglobin, an erythrocyte count of 2,760,000 per c.mm., and a leucocyte count of 3,600 per c.mm. (polymorphonuclears, 17% ; lymphocytes, 70% ; eosinophils, 13%) compared with the concurrent peripheral blood count of 89% haemoglobin, an erythrocyte count of 4,200,000 per c.mm., and a leucocyte count of 7,900 per c.mm. (polymorphonuclears, 60% ; lymphocytes, 33% ; eosinophils, 5% ; monocytes, 2%). The radiographic appearances of the chest remained unchanged. Next day (August 9), despite frequent attempts at aspiration, only 2 oz. of fluid was removed, but four days later 1 pint of haemorrhagic fluid was withdrawn and the radiograph now showed that the left lung had almost completely re-expanded, although a small effusion persisted. The aspirated fluid contained 22% haemoglobin and 830,000 per c.mm. of red cells.

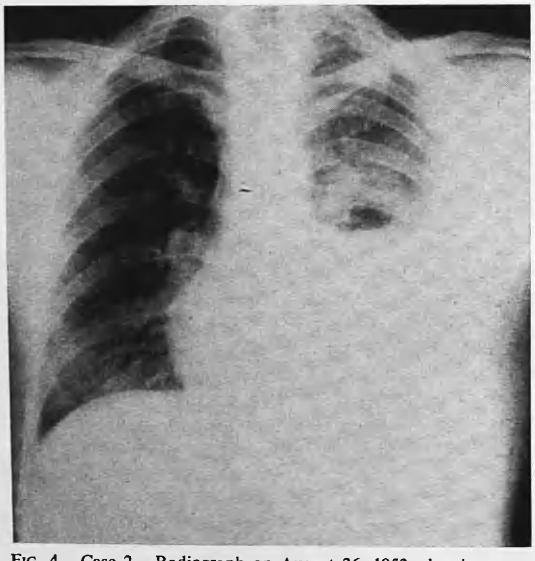

FIG. 4.—Case 2. Radiograph on August 26, 1952, showing recurrence of left-sided effusion preceding the aspiration of a further 1½ pints of haemorrhagic fluid and the instillation of " varidase."

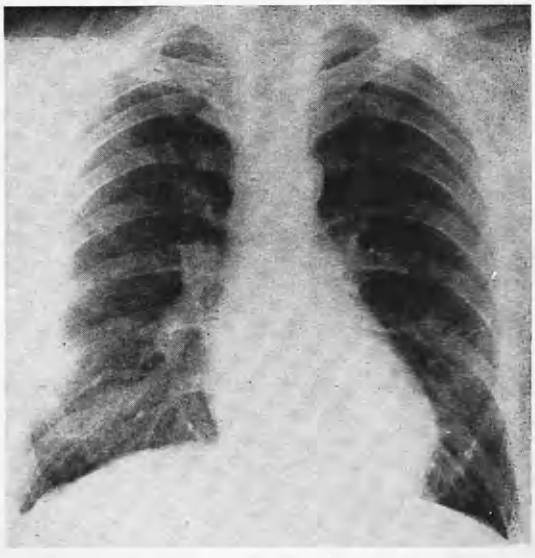

FIG. 5.—Case 2. Radiograph on October 15, 1952, a fortnight after discharge from hospital. No residual effusion.

Up to August 26 there was evidence of recurrence of the effusion (Fig. 4). This refractory phase, coupled with evening pyrexia to about 100° F., prompted further aspiration, when 30 oz. of xanthochromic, relatively acellular fluid was withdrawn. This fluid had a protein concentration of 4.9 g.% and a bilirubin level of 1.7 mg.%. This aspiration was immediately followed by the instillation of "varidase" (200,000 units of streptokinase and 50,000 units of streptodornase, dissolved in 10 ml. of physiological saline) into the pleural cavity. During the next 48 hours the patient felt further distress. There was a recurrence of chest pain, breathlessness, perspiration, pyrexia (100–102° F.), a tachycardia (90–110 per minute), a leucocytosis of 13,100 per c.mm. (polymorphs 90%, lymphocytes 10%), and increased effusion. On August 27 a pint of clear, relatively cell-free fluid was aspirated. Its bilirubin level was 0.15 mg.%. Uneventful progress ensued. Further thoracenteses were unproductive. By September 17 the chest radiograph revealed only partial collapse of the left lung, associated with a small effusion.

On October 3 he was discharged home, feeling fit and regaining weight. Follow-up has shown virtually a complete recovery, for the serial chest radiographs have appeared normal since the film of October 15 (Fig. 5). He resumed work one month after discharge. He had then gained a further 7 lb. in weight. No evidence of pulmonary tuberculosis was obtained from the radiological investigations or sputum (four separate specimens were examined). The Mantoux test was positive to a 1 in 1,000 dilution.

CASE 3.—F. C., a housewife, aged 40, was admitted here on June 22, 1953, for the treatment of a recurrent compound ganglion on the dorsum of the wrist with a course of combined streptomycin and P.A.S. An identically located ganglion had been excised two years previously and it revealed typical melon-seed bodies with the histological appearance of tuberculosis.

Apart from three attacks of rheumatic fever in childhood, producing no subsequent evidence of cardiac disease, she had no previous illnesses of note. Although there was a strong family history of tuberculosis, her frequent serial chest radiographs and erythrocyte sedimentation rates had always been normal. In particular, the lung apices remained clear.

Physical examination showed no striking signs apart from facial pallor, koilonychia, and the ganglion. Her blood pressure was 140/80 mm. Hg. A blood count revealed haemoglobin 73%, erythrocyte count of 3,600,000 per c.mm., mean cell diameter 7.2 μ, and a leucocyte count of 14,000 per c.mm. with a normal differential count. The mean E.S.R. was 9 mm. in one hour. On August 10, 1953, the ganglion was excised and again it contained typical melon-seed bodies.

On August 13 her temperature rose to 99° F., while she experienced left-sided pleuritic pain, unaccompanied by a pleural rub or an effusion. The following afternoon she suddenly became shocked and dyspnoeic with a moderate left-sided pleural effusion. The mediastinum was not displaced (Fig. 6). The relatively sudden onset of a large pleural effusion and pallor immediately suggested a diagnosis of haemothorax. This was promptly confirmed by the aspiration of apparently pure blood with a haemoglobin value of 62%. Moderate shock was shown by free perspiration, a pulse rate of 120 per minute, and hypotension (80/40 mm. Hg). A half-hourly pulse

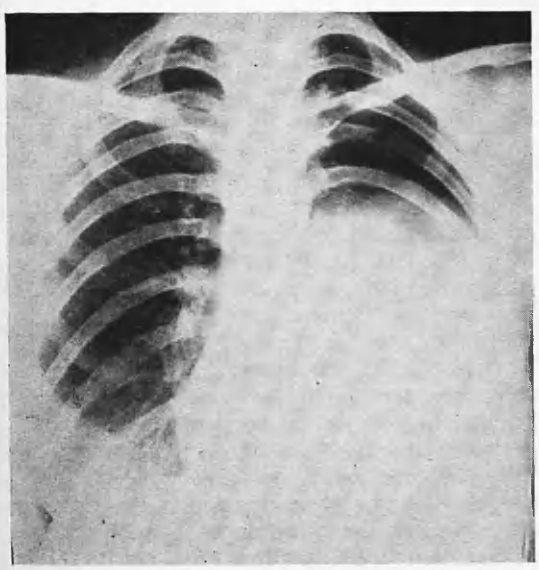

FIG. 6.—Case 3. Radiograph on August 14, 1953. a large initial left-sided pleural effusion before thoracentesis was begun, but no mediastinal displacement.

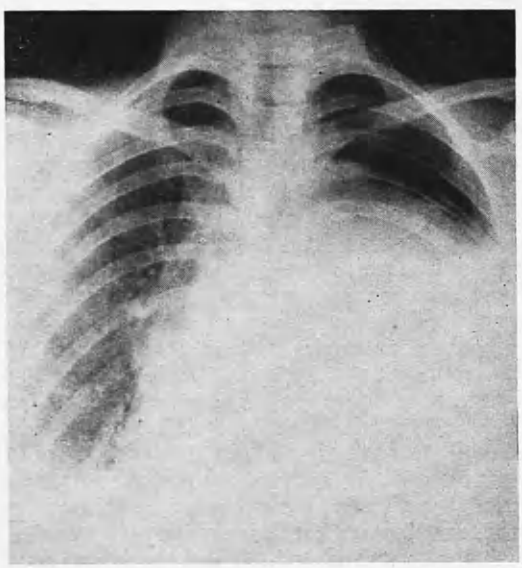

FIG. 7.—Case 3. Radiograph on August 15, 1953, three hours before death. The radiograph resembles Fig. 6, but there is now mediastinal shift.

and blood pressure chart was now begun and a blood transfusion set up. By 10 a.m. next day (August 14) her haemoglobin level was only 64%, despite the administration of 4 pints of blood and 1 pint of plasma. The blood pressure was now 120/80 mm. Hg, but the pulse rate had increased to 140 per minute. Therapeutic thoracentesis was now begun, but after the aspiration of 2 pints of sanguineous fluid the patient demanded a respite. Meanwhile blood transfusion was continued; a further 5 pints of blood were at hand.

By 2.30 p.m. the blood pressure was 140/85 mm. Hg, with the pulse rate still 140 per minute, but rose to 150 per minute by 4 p.m. The blood pressure was unchanged. The chest radiographs (Fig. 7) now showed mediastinal shift to the right with a persistent large pleural effusion. Respiratory distress continued without signs of cerebral anoxia. Just before further aspiration at 4.30 p.m. there was a sudden general deterioration. The blood pressure dropped abruptly to 110/60 mm. Hg, and the pulse was thready and its rate was uncountable. A further 2 pints of the haemorrhagic fluid were promptly aspirated, but a syncopal attack precluded further immediate thoracentesis. At 5 p.m. she lapsed into extreme respiratory distress, accompanied by restlessness, disorientation, and delirium. The pupils were now large and reacted only sluggishly to light. As an arterial transfusion was being set up the patient died at 5.30 p.m.

Necropsy revealed 2½ pints of partly clotted blood in the left pleural sac, while the underlying lung was completely collapsed. A stretched and torn apical pleural adhesion with a bleeding point was located between the necks of the first and second ribs. The lungs showed no evidence of tuberculosis. It was concluded that the haemopneumothorax had resulted from the spontaneous tear of an old tuberculous pleural adhesion.

Discussion

The main aspects of spontaneous haemopneumothorax will now be reviewed.

Terminology, Aetiology, and Pathology.—Hansen (1949) preferred the term "pneumohaemothorax," as the primary event is the intrapleural extravasation of air, followed by haemorrhage. The terms "spontaneous" or "idiopathic" may be erroneous (Cummer, 1915) as this condition could be a sequel to previous tuberculosis, no longer demonstrable. Indeed, Parkes Weber (1931) averred that "tuberculous" and "idiopathic" pneumothorax or haemopneumothorax differed only in that in the "idiopathic" type the tuberculous lesion was healed. Most cases of the "spontaneous" variety probably have occult congenital anomalies or relics of subpleural pulmonary disease, but it is quite exceptional for pulmonary tuberculosis to develop later (Louria, 1938; Hyde and Hyde, 1951).

Pleural adhesions are often observed in routine necropsies even where there is no previous history or concurrent evidence of lung infection (Leopold and Lieberman, 1935). The contributory role of such supposed exciting causes as straining, coughing, sneezing, and yawning is undetermined. They may be absent (Louria, 1938). A spontaneous pneumothorax first develops and its various mechanisms have been critically discussed (Kjaergaard, 1932; Perry, 1939). Air escapes intrapleurally following the rupture of a valve-vesicle or an emphysematous bulla. An alternative view is that of Macklin and Macklin (1944), who produced pneumothorax and haemopneumothorax in experimental animals by the initial induction of pulmonary interstitial emphysema. They postulated that this might occur in man, following the intrapulmonary rupture of alveoli with air, then dissecting subpleurally along the interstitial tissues to leak through the visceral or mediastinal pleura into the pleural cavity. This subject has been discussed in detail (Solovay, 1949).

The accepted explanations incriminate rupture of both subpleural bullae and adhesions, the former allowing the escape of air and the latter blood into the pleural cavity. The exact source of bleeding has rarely been demonstrated (Hartzell, 1942; Helwig and Schmidt, 1947; Cuningham, 1950). Pleural adhesions are torn by the traction of the expanding pneumothorax, and in the case of massive haemothorax, at least, the haemorrhage is believed to come from the parietal end of a severed pleural adhesion (Hartzell, 1942), which is supplied by the intercostal vessels and is independent of the haemostatic influence of the accompanying pulmonary collapse.

Both subpleural bullae (Pardel and Mazzei, 1934) and pleural adhesions (Helwig and Schmidt, 1947) can be highly vascularized. Since Pitt (1900) first reported a torn emphysematous bulla attached to a fibrous adhesion, other authors (Hopkins, 1937; Hartzell, 1942) have either produced evidence of or have directly observed (Hansen, 1949) haemorrhage from a torn bulla. Yet others (Davidson and Simpson, 1940; Arst, Lahey, and Kunkel, 1950; Irwin, 1951) have shown that vessels in torn adhesions can be the source of haemorrhage or have witnessed this at thoracotomy (Beatty and Frelick, 1952; Myers *et al.*, 1951; Ross, Dugan, and Farber, 1953), where the appearances closely resemble the occasional haemorrhagic complication in the operation of thoracotomy with division of pleural adhesions (Sellors, 1951).

Although other relevant observations on haemothorax can only be briefly cited, it is worth recalling that Hart and Jones (1947) record this as a sequel to aberrant lung tissue, while Crawford and Shafar (1946) considered that haemothorax might follow the spontaneous rupture of a pleural adhesion during sudden muscular movement. Again, two of Hansen's three cases (1949) of spontaneous haemopneumothorax had cystic disease of the lungs, an association which had not then been reported, but is also apparent on the affected side in our first case (Fig. 3).

Intrapleural blood clots rapidly and this explains the frequent absence of coagulation *in vitro* of such pleural aspirates (Cosgriff, 1950). The natural history of haemothorax has been reviewed recently (Harold, 1951). This effusion remains fluid because of the defibrinating action of the cardiorespiratory movements, while fibrin is deposited on the pleural surfaces. Melick and Spooner (1945) showed in experimental animals that intrapleural clotting recurs if further fibrinogen is added. Blood in the pleural space acts as an irritant, provoking a large serous effusion (Sellors, 1945) which dilutes the sanguineous fluid present. Secondary clots may occur several days later due to an increased concentration of fibrinogen. Large gelatinous masses of bloody fibrin may laminate the parietal pleura and be followed quickly by angioblastic and fibroblastic proliferation, extending into the clot from the pleura, which itself remains as a thin, translucent membrane (Moore, 1949). This fibrothorax, sometimes accompanied by loculation, is disabling (Smithy, 1943) and characterized by impaired lung expansion from a flattened hemithorax of approximated ribs with secondary scoliosis, described as the "frozen" or "fixed" chest (Sellors, 1951).

CLINICAL SYNDROME AND PROGNOSIS.—Our three cases typify the remarkably clear clinical picture, which resembles that of spontaneous pneumothorax but with varying additional evidence of haemorrhage and pleural effusion. The patient, with previous good health, although sometimes underweight (Hyde and Hyde, 1951), is usually a male of 15 to 45, frequently in the third decade. During mild activity, or sometimes while at rest, he is seized with a sudden, sharp, stabbing unilateral pain in the anterior, or occasionally posterior, chest. This pain is increased by movement and may radiate to the shoulder or abdomen and often subsides within 24 hours, only to return. The pain may be confined to the abdomen (Fusia and Cook, 1952). Irritation of the diaphragmatic pleura may produce signs simulating an acute abdominal emergency, especially if there are well-marked signs of shock, while nausea, vomiting, and even abdominal rigidity may further mislead (Rolleston, 1900 ; Hurxthal, 1928 ; Milhorat, 1931 ; Waring, 1945 ; Crimm, 1948 ; Hansen, 1949 ; Deiss, Gale, and Brown, 1950), even to the extent of leading to laparotomy (Fischer, 1922 ; Fusia and Cook, 1952 ; and Ross, 1952). It may simulate appendicitis (Milhorat, 1931 ; Jones and Gilbert, 1936), gall-bladder disease (Hurxthal, 1928 ; Frey, 1935), bleeding peptic ulcer (Irwin, 1951), or perforated peptic ulcer (Rolleston, 1900 ; Fischer, 1922). Again, the pain may suggest coronary thrombosis (Castex and Mazzei, 1936 ; Rist and Worms, 1940 ; Sloer, 1939 ; Hansen, 1949), and if there is electrocardiographic evidence of myocardial ischaemia, as in Case 1, further confusion in diagnosis might arise. Thus, in the older patient, a dissecting aneurysm of the aorta with subsequent haemothorax might well be wrongly suspected. Coexistent haemoptysis has occasionally been described (Szenes, 1929 ; Groen and Godfried, 1948). As in Case 2, an eosinophilia of the blood in both the circulation and the effusion has occasionally been described (Troisier, Bariéty, and Dugas, 1936 ; Groen and Godfried, 1948 ; Deiss *et al.*, 1950 ; Hyde and Hyde, 1951). Complete recovery from spontaneous haemopneumothorax is usual within two months, but death from relentless haemorrhage is always a danger within the first three days. A late fatality may result from large fibrinous deposits (fibrothorax) as described by Jones and Gilbert (1936). Slight pyrexia, leucocytosis, and a raised erythrocyte sedimentation rate are common accompaniments of spontaneous haemopneumothorax.

TREATMENT.—The intrapleural haemorrhage frequently stops within the first three days. Such cases respond to bed rest, sedation, oxygen inhalation, antibiotic therapy, adequate blood transfusion, and early repeated and complete evacuation of blood and air from the pleural cavity. Experience in the management of chest wounds during and since World War 2 has led to a radical alteration in the treatment of haemothorax (Sellors, 1945 ; Tuttle, Langston, and Crowley, 1947). It was previously considered that thoracentesis should be delayed or, if adopted, that it should be accompanied by air replacement. This latter procedure is both useless (Fusia and Cook, 1952) and even a hindrance to early recovery (Sellors, 1951). It has been pointed out (Sellors, 1945) that immediate and repeated aspiration, although decreasing the intrapleural pressure, does not restart bleeding and will prevent a later fibrothorax. There is no evidence

(Fusia and Cook, 1952) that properly administered blood transfusion incites further haemorrhage. Moser and his colleagues (1951) described four cases responding to these measures, while Hyde and Hyde (1951) had similar success in 11 out of 12 cases, with the remaining patient requiring late decortication. In Case 1, which had syncopal attacks on admission, the adoption of conservative treatment, not including blood transfusion, sufficed to produce a speedy recovery.

Melick and Spooner (1945) have shown that haemothorax may be followed within five days of its onset by fibroblastic proliferation, which in turn leads to organization and the formation of a "peel" on the contracted lung surface. This sequence can now be prevented by enzymatic debridement following the intrapleural injection of fibrinolytic enzymes (Sherry, Tillett, and Read, 1950; Read and Berry, 1950; Carr and Robbins, 1951; Miller, Ginsberg, Lipin, and Long, 1951; Ross, 1952; and Jones and Bigham, 1953) in those patients in whom aspiration is indicated, but has proved unsuccessful and haemorrhage has stopped.

Combinations of streptokinase (S.K.) and streptodornase (S.D.) have usually been employed, but more recently other workers (McCroskey and Hardin, 1953; Delannoy and Ribet, 1953) have used trypsin with equal success. Ross (1952) advocates that aspiration of the blood should be followed by instillation of these enzymes, allowing them to remain *in situ* for only six to eight hours. Then complete aspiration of the residual enzyme solution should be carried out, using irrigation with physiological saline, if necessary. Repeated injections, employing several sites, may be required. This treatment is applicable to those patients who are liable to prolonged convalescence because of dense fibrin deposits or clotted haemothorax. Indeed, this fibrinolytic therapy may prevent subsequent decortication (Read and Berry, 1950; Beatty and Frelick, 1952), although the former is no substitute for the latter (Carr and Robbins, 1951).

Streptokinase liquefies fibrinous exudates and blood-clots, thus exerting mainly a fibrinolytic action (Christensen and MacLeod, 1945; Tillett, 1952), while streptodornase acts only on the nucleoproteins of the degenerate cells in the exudate (Tillett, Sherry, and Christensen, 1948). Neither enzyme is harmful to healthy cells, while both enhance phagocytosis (Johnson, 1950), thus acting synergistically with antibiotics. Aspiration of the lysed fluid is an important part of the procedure, while trypsin cannot be usefully combined with the other enzymes as it destroys streptokinase. This enzymatic treatment proved valuable in Case 2 which had, however, an associated febrile response with pleuritic pain, as Carr and Robbins (1951) had described in six of their 10 cases of clotted haemothorax so treated.

Three reactions may accompany the use of S.D. and S.K. instillations. First, a local effusion with living polymorphonuclear leucocytes. Secondly, a pyrogenic reaction, with the initial rise of temperature occurring about six hours later and becoming maximal in 24 hours, provided that the enzymes and lysed material are not evacuated sooner. The febrile reaction is usually accompanied by headache, nausea, malaise, and arthralgia, but these symptoms usually respond to aspirin administration. Thirdly, and rarely, since both S.D. and S.K. are antigenic, allergic reactions involving the periorbital tissues may result. In the case of trypsin, slight histaminic reactions may occur, manifest by pyrexia, hypotension, and tachycardia, but neutralized by antihistaminics. Caution is required during fibrinolytic enzyme therapy to avoid an injection directly into a blood-vessel, while the pyrexial reactions may wrongly suggest that further haemorrhage is occurring.

Massive, uncontrolled intrapleural haemorrhage is a surgical, rather than a medical, emergency. Early thoracotomy is often indicated to identify and either ligate or electro-coagulate the bleeding point as a life-saving procedure. Hansen (1949) first undertook this measure, but he was unfortunate enough to observe multiple torn emphysematous bullae which were too extensive for suture. Other workers (Deucher, 1949; Myers *et al.*, 1951; Beatty and Frelick, 1952; Holloway, Speir, and Sadler, 1952; Ross, 1952; Ross *et al.*, 1953; Borrie, 1953) demonstrated its value. Indeed, in the patient described by Myers and his colleagues (1951) the intrapleural bleeding was estimated to exceed 5 litres. This operation permits the simultaneous removal of the blood-clot, and the recovery phase may thus be reduced from months to weeks. The bleeding point may not, however, be identified (Harold, 1951). This is hardly surprising as identification of the source of haemorrhage has frequently not been possible in cases submitted to necropsy. We considered resort to thoracotomy in our third case, but its deferment beyond the first few hours from the onset left us virtually with a moribund patient, in whom simple thoracentesis produced syncope. Our reflections on this case led us to conclude that more emphasis should be placed on the pulse rate than on the blood pressure as an index for immediate surgery. Thus, when

the pulse rate rose above 120 per minute and mediastinal displacement was manifest we had over-delayed resort to remedial thoracotomy, despite a normal blood pressure. The abrupt deterioration in this patient was especially surprising as she was the only patient presenting without mediastinal shift.

Finally, for those patients who fail to benefit from enzymatic instillation, thoracotomy with pulmonary decortication (pleurectomy) is now the established procedure (Elrod and Murphy, 1948; Hansen, 1949; Deiss *et al.*, 1950; Seley and Neuhof, 1951; Carroll, McClement, Himmelstein, and Cournand, 1951; Hyde and Hyde, 1951; Kiekens, 1952; Eidinger and Rubin, 1952; Fusia and Cook, 1952) to prevent extensive fibrinous deposits on the pleural surface, a calcified pleura (Milhorat, 1937), cor pulmonale, and spinal deformities. Pulmonary decortication, however, may lead to other complications such as further haemorrhage, air embolus, or bronchopleural fistula (Deucher, 1949). Carroll and his colleagues (1951) used this technique in nine cases of haemothorax, two of which were of the spontaneous variety. Of these two patients, one derived great benefit, while the other showed only slight improvement in lung function. He concluded that operative success may occur even without much associated clinical relief. Kiekens (1952), on the other hand, claimed that this operation was of great value.

Summary and Conclusions

The literature on spontaneous haemopneumothorax, comprising some 150 cases, is surveyed. The aetiology, differential diagnosis, and recent therapeutic trends are outlined. This complication of spontaneous pneumothorax merits wider recognition as a potentially grave emergency. The fatality rate is 15%.

Three recently observed cases are described. All had frequent and early thoracenteses. The first patient responded to conservative measures without resort to blood transfusion; the second required blood transfusion and, later, enzymatic pleural debridement; the third had continued intrapleural haemorrhage requiring repeated blood transfusions, but towards the end of the first 24 hours even simple thoracentesis induced syncope. She died before arterial transfusion was begun while unfit for remedial thoracotomy. Retrospective consideration compels the belief that increasing tachycardia despite normal blood pressure warrants immediate thoracotomy in such cases.

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POSTSCRIPT

A fourth case of spontaneous haemopneumothorax was treated recently by one of us (R. J. C.).

J. W., a motor mechanic aged 27, was admitted to the Royal Victoria Hospital, Boscombe, on October 2, 1954. At work that morning he suddenly felt an acute, left-sided sub-mammary pain, worse on movement and deep breathing, and radiating to the left clavicular and scapular regions. On admission to hospital a few hours later he had signs of a moderate left-sided pleural effusion with mediastinal displacement to the right. He was pallid, but the pulse rate and blood pressure were normal.

The clinical impression of haemopneumothorax was supported by a radiograph of the chest and was confirmed by thoracentesis. A single slow aspiration of 3 pints of sanguineous fluid was carried out. No further treatment was adopted. The haemoglobin concentration of the effusion was 76%, compared with 72% in the peripheral blood. A week later the radiograph of the chest was normal. Subsequent progress was uneventful. He was discharged from hospital on October 30 and began work again one month later.