

## RECURRENT AND CHRONIC SPONTANEOUS PNEUMOTHORAX\*

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The general problem of spontaneous pneumothorax in the apparently healthy, or "pneumothorax simplex" as it has been termed by Kjaergaard (1932), has been dealt with fairly extensively by a number of authors. A certain amount has been written about recurrent attacks and somewhat less about chronic spontaneous pneumothorax, but nothing really authoritative has been published. This paper deals with these two manifestations of spontaneous pneumothorax; it is based on a study of seventy-one personal cases.

From a study of the literature the incidence of single attacks of spontaneous pneumothorax would not appear to be high. One of the most comprehensive papers is by Kjaergaard (1932), whose fifty-one cases were gathered from hospitals in Copenhagen and from about fifty others in Denmark, and covered a period of twenty years. Perry (1939) based his interesting and useful study of the condition on eighty-five cases which were admitted to the London Hospital between 1924 and 1937, a period of fourteen years. Ornstein and Lercher (1941-2) discuss fifty-eight cases occurring in the Seaview Hospital. In practice "pneumothorax simplex" is found to be commoner than this, especially when the condition is being looked for and when patients are radiographed more frequently. It is, for instance, diagnosed in students and nurses often enough to suggest that many more cases are overlooked in the population at large. Perry found many extra cases in the out-patient department when he was deliberately on the lookout for the condition.

The majority of cases described by authors are of simple, single attacks; in the literature the incidence of recurrence and chronicity is extremely small. Thus Kjaergaard's fifty-one cases included seven which had recurrent attacks and four chronic cases. Perry records only four recurrent and two

chronic cases amongst his eighty-five; in his survey of the literature he found thirty-three single cases of recurrent pneumothorax. Ornstein and Lercher's series of fifty-eight cases included seventeen with recurrent attacks. Nikolski (1912) had nine recurrent cases in his series of ninety. Wood (1931) states that, in seventy-one patients with spontaneous pneumothorax at the Mayo Clinic, 21 per cent gave a history of multiple attacks and in 11 per cent both sides had been affected at different times; in three cases both sides had been affected simultaneously. There are a certain number of reports in the literature of chronic cases, mostly single cases, occasionally two or three and never more than a handful; a number of these have been found accidentally, and from the radiographs produced it is certain that some are not examples of chronic pneumothorax but of giant tension bullae or cysts.

I have been unable to discover any comprehensive account of a large series of recurrent and chronic cases of spontaneous pneumothorax, and it is fair to state that the series of seventy-one cases that forms the basis of this study is unique. I would stress that these are all of the chronic or the recurrent type and include none with simple isolated or short attacks; all have been in my own practice, seen and treated by me, and they cover the years 1936-47. While it is true that a number of friends and colleagues, knowing my interest in this condition, have kindly referred their cases to me, it is nevertheless worthy of comment that such a large number has been seen by one surgeon in only eleven years. It suggests that recurrent and chronic cases are far commoner than has hitherto been supposed. It is certain that they constitute a real and important problem and one that it is wrong to dismiss lightly as is so often done in discussion of the condition. From my experience of the disability caused, I find it difficult to accept the complacent comments often made that there is no need to worry about treatment; that, given time,

\*Based on a paper read before the Thoracic Society in February, 1948.

the lung will always expand; that the patient should be rested in bed or, in recurrent cases, merely advised to avoid doing anything strenuous. Such remarks betray complete failure to understand the problem as it really exists.

# DEFINITION

Although the lung in "pneumothorax simplex" usually expands in two or three weeks, it may take two or even three months to do so. If it is still collapsed after three months it is fair to classify it as a chronic case. Chronicity occurred in forty-six of my own series, and the *average* duration in these was fifteen months; the longest was nine and a half years; in one the duration was three and a half years, in two it was two and a half years, and in a fifth two years. Amongst these forty-five chronic cases the condition was entirely chronic in twenty-nine; in seventeen, chronicity supervened upon recurrent attacks (Table I).

TABLE I

CASES OF CHRONIC PNEUMOTHORAX IN A SERIES OF SEVENTY-ONE CASES OF SPONTANEOUS PNEUMOTHORAX

Age	Chronic	Chronic supervening or recurrent	Total chronic
1-10	2	0	2
11-20	0	2	2
21-30	3	7	10
31-40	6	6	12
41-50	13	2	15
51-60	5	0	5
61-70	0	0	0
Total	29 { 21 men 8 women	17 { 10 men 7 women	46 { 31 men 15 women
Duration	Average 15 months; longest 9½, 3½, 2½, 2½, and 2 years		

Twenty-five in my series presented as purely recurrent cases; in a further seventeen recurrences ended in chronicity, making a total of forty-two in which relapses occurred (Table II). The recurrences may be all on one side, or they may be alternating bilateral (fifteen in my series) as first reported by Goodhart (1896), or both sides may be involved simultaneously (Plate XXIa) (8 examples). In others a chronic pneumothorax has been present on one side and recurrent attacks have occurred on the opposite side. The average number of attacks was four; the greatest number in one patient was fifteen; another patient had eleven attacks, and a third had ten. Locke (1929) mentions a recurrent case in which eighteen attacks

occurred over a period of seven years; both sides were affected at different times.

TABLE II

CASES OF RECURRENT PNEUMOTHORAX (IN SEVENTY-ONE CASES OF SPONTANEOUS PNEUMOTHORAX)

Age	Recurrent	Recurrent ending in chronic	Total
1-10	0	0	0
11-20	4	2	6
21-30	10	7	17
31-40	6	6	12
41-50	2	2	4
51-60	2	0	2
61-70	1	0	1
Total	25 { 21 men 4 women	17	42
Bilateral:			16
	Alternating { 10 men 5 women		15
	Simultaneous		8

Average number of attacks 4: largest numbers 15, 11, 10

# SIGNIFICANCE

These figures emphasize the seriousness of the condition, and this is amply borne out by first-hand knowledge of the cases themselves. I have already referred to misguided statements that chronicity does not seem to matter and that the lung will usually expand in time; or that relapses can be treated by further rest in bed or by prohibiting exercise. The first statement is simply not true; most patients with a chronic pneumothorax suffer considerable disability, and many are chronic invalids. This is quite apart from the very real danger of a further pneumothorax on the opposite side, and also the danger of an acute respiratory infection on the good side. One of the few cases in the literature studied post-mortem (Brunner, 1921) was of a patient who died from an attack of influenzal pneumonia in the opposite lung. Many of these patients with a chronic pneumothorax have wasted months or even years resting in bed or in sanatoria. If tuberculosis is not the cause of the condition, it seems illogical, unkind, and wasteful to condemn a patient to spend from six to twelve months resting in bed for what is usually a simple mechanical event which can be quickly relieved by active measures.

The same unnecessary restriction is often applied to the relapsing cases, and it is particularly misguided to enjoin these patients to live a life of restricted activity. Many of them are otherwise

healthy, and such advice moreover reveals ignorance of the fact that many of the attacks occur while the patient is at rest; it is by no means uncommon for them to happen when the patient is lying in bed, or when he first gets out of bed in the morning. One of my patients, a man who had had fifteen attacks on both sides and who had been told by a doctor to avoid strenuous exercise, said, "My last attack occurred when I lifted up my baby daughter; I am not to be allowed to do even that?" It is, of course, true that violent exercise is more likely to cause an attack, and cycling is a common precipitating cause.

Although spontaneous pneumothorax is often designated as something that occurs in the apparently healthy, my experience with and analysis of these patients with recurrences or chronicity reveals that the condition is often an indication of the presence of notable disease in the lungs for which treatment may be needed. In fact it is my impression that in chronic and recurrent pneumothorax we have not so much a clinical entity as a manifestation in one aspect of the natural history of many lung diseases. This receives support from the fact that five of my patients have since died of their primary disease.

#### ANALYSIS OF MATERIAL

Statistics are usually tedious and often disappointing in what they convey, but it is still necessary to present them, albeit in as brief and simple a way as possible. Table III shows the relative

TABLE III

COMPARISON OF THE PRESENT SERIES WITH 358 CASES FROM THE LITERATURE

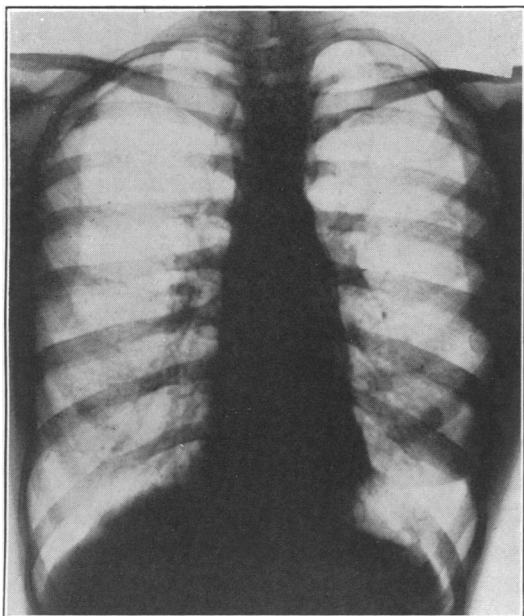
Age	Present series (youngest patients $1\frac{1}{2}$ years, $4\frac{1}{4}$ years; oldest patient 64 years)	358 cases collected by Perry (1939)
1-10	2	14
11-20	6	51
21-30	20	120
31-40	18	57
41-50	17	39
51-60	7	16
Over 60	1	5
Total	71 { 52 men 19 women	302 in which age recorded

incidence in men and women (52 men, 19 women), which compares closely with the figures collected from the literature by Perry (301 men, 57 women). The age incidence is of interest, for it will be seen

that all decades are represented, the two youngest patients being  $1\frac{1}{2}$  and  $4\frac{1}{4}$  years, and the oldest 64. The age distribution is, however, significantly different from that of pneumothorax simplex, in which the greatest incidence is between 20 and 30. The higher incidence of chronic and recurrent cases in the third and fourth decades reflects the differing aetiology in these cases.

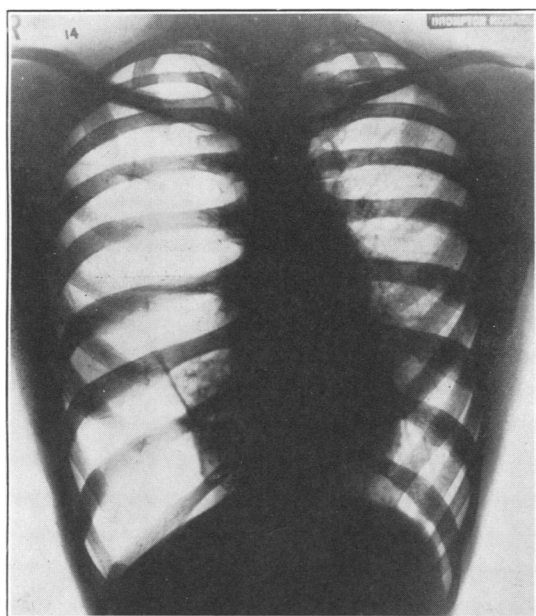
*Clinical features.*—The clinical features of pneumothorax simplex have been amply and frequently presented and there is no need to elaborate on the well-known facts. Certain points deserve mention in the chronic and recurrent cases. The greater tendency for the older age groups to be affected has just been mentioned, and this is associated with a greater incidence of the degenerative changes of chronic bronchitis and emphysema. In very young patients congenital cystic disease may be a possible cause, but the most frequent is staphylococcal pneumonitis with lung abscess. It is now generally accepted that tuberculosis, although a common cause of spontaneous pneumothorax, is but rarely the cause in the group known as "pneumothorax simplex" or "pneumothorax occurring in the apparently healthy." Ornstein states that three of his fifty-eight patients developed tuberculosis about two years later; only one of Kjaergaard's fifty-one patients developed the disease, and that was several years later and after definite exposure to infection. One of my seventy-one patients has developed symptoms (five and a half years after) suggesting pulmonary tuberculosis, and so far even this is not proven; another patient, a boy of 12, with bilateral chronic pneumothorax from severe congenital polycystic disease, died of acute pulmonary tuberculosis two years after the onset of his pneumothoraces and eighteen months after they had been cured. Pneumothorax in tuberculosis is a late, often subterminal, complication rather than an early manifestation. This is borne out in my series of chronic and recurrent cases in which pleural tuberculosis was found to be the cause in only one instance. Although this patient presented as a case of chronic pneumothorax of two and a half years' standing, and the pleura was dry when examined, there was a history of several bouts of fever and of fluid formation in the air-space; this indicated the probability of the tuberculous basis which was easily confirmed at thoracoscopy. Fluid formation is rare in the non-tuberculous cases, and when it does occur it is little more than a trace.

It is not uncommon for the presence of a pneumothorax to be noticed accidentally. For instance a doctor was found to have a pneumothorax when he was being examined for insurance; the only

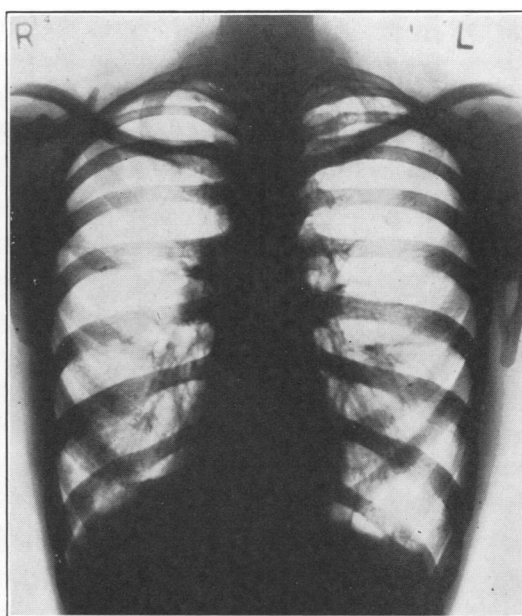


(a)

PLATE XXI.—(a) To show bilateral spontaneous pneumothorax in a young woman who suffered from asthma, chronic bronchitis, and emphysema. (b) Right chronic pneumothorax of seven months' duration in a woman aged 26 who was an asthmatic and bronchitic. (c) Radiograph of the same patient as in (b) after right pleurodesis with silver nitrate; the lung is now fully expanded.



(b)



(c)

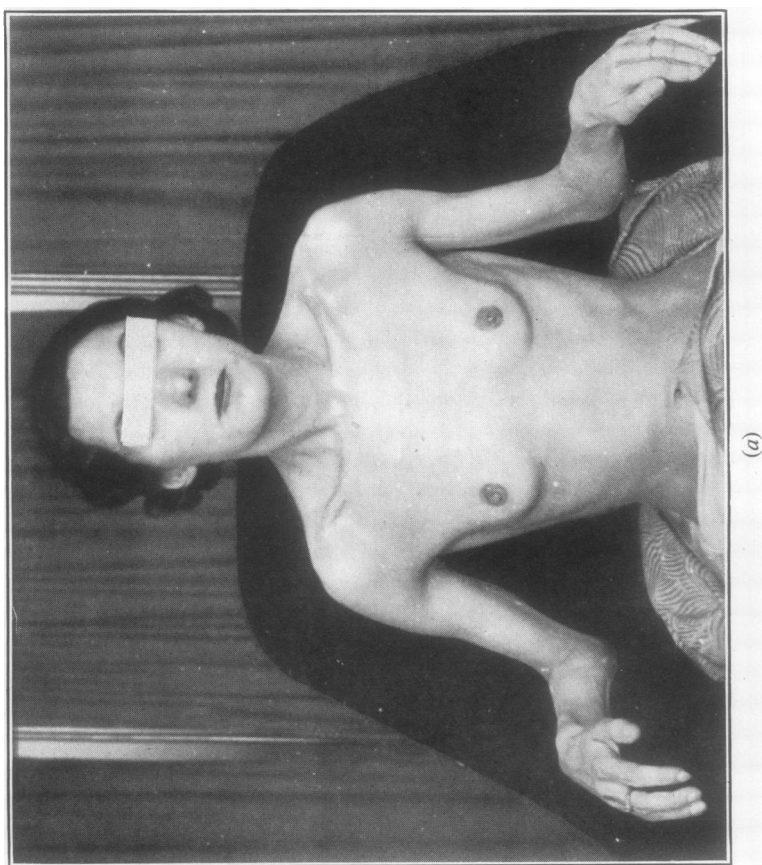
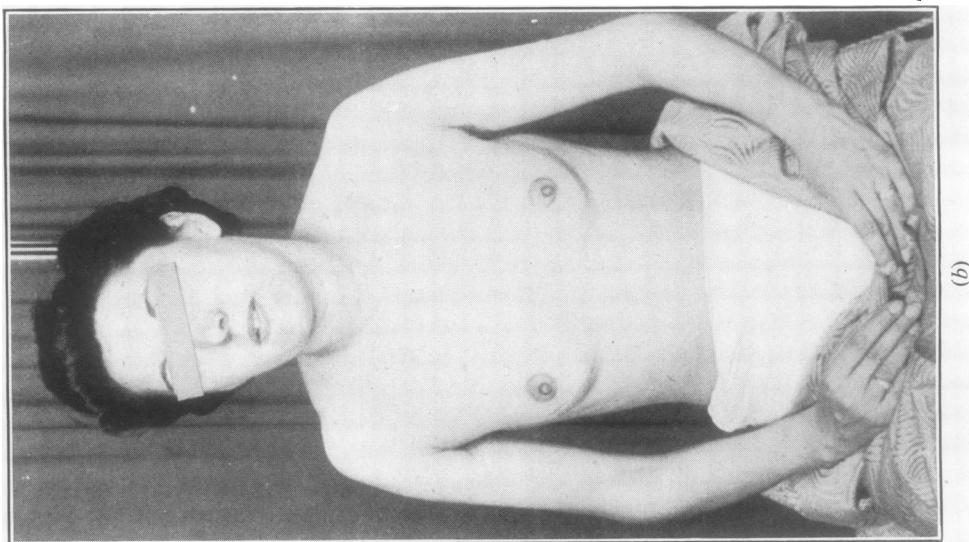
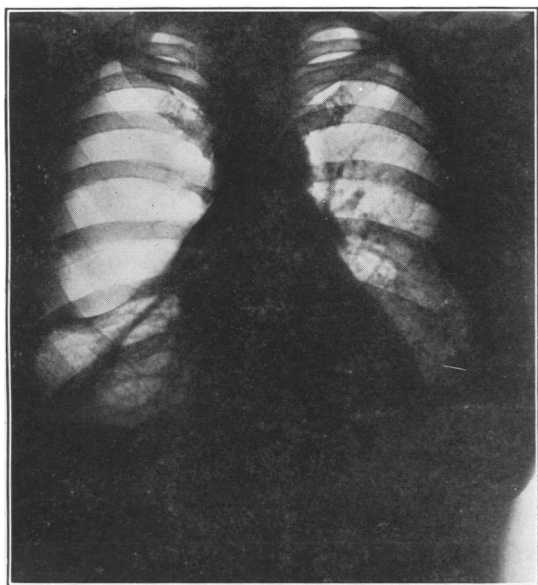
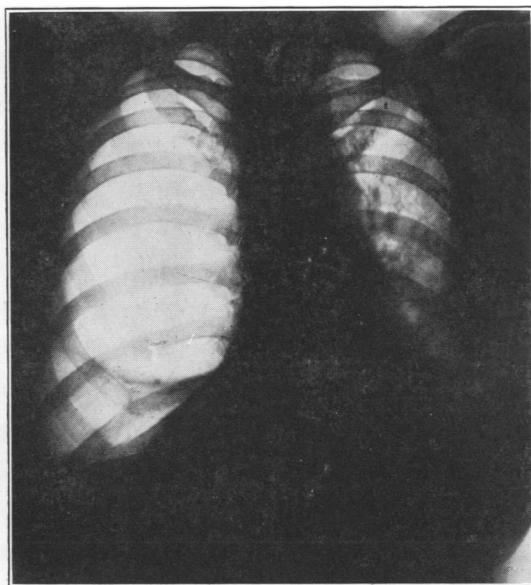


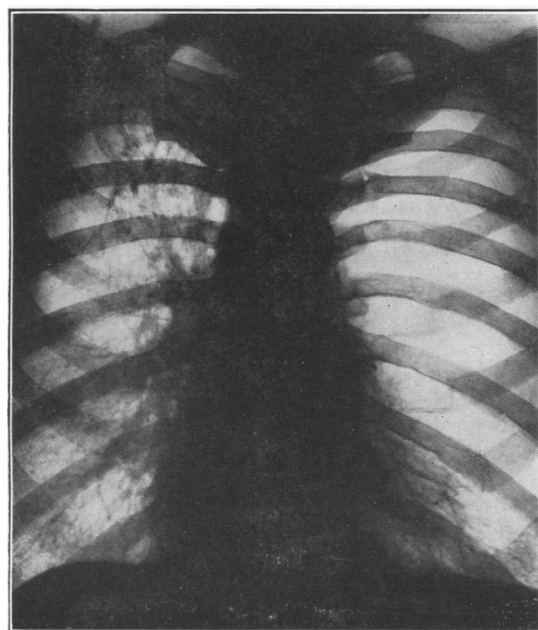
PLATE XXII.—(a) Photograph of patient at time of chronic pneumothorax shown in Plate XXIb. She had lost 33 lb. in weight. (b) Photograph of the same patient after pleurodesis.



(a)

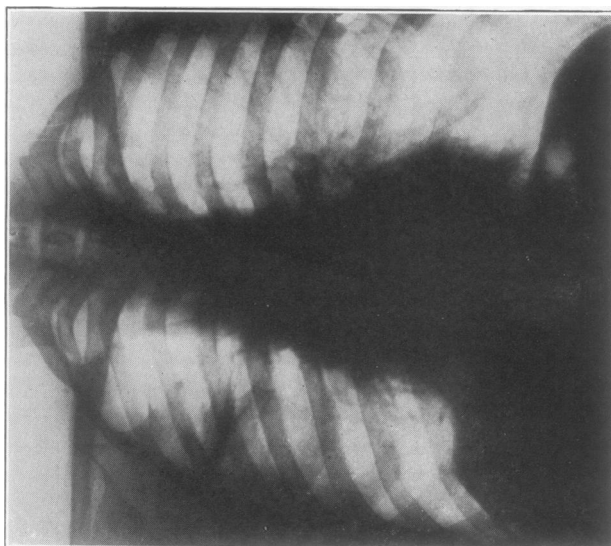


(b)



(c)

PLATE XXIII.—(a) A radiograph of a giant bullous cyst simulating a chronic pneumothorax. (b) The same case as in (a); the bulla has increased in size and caused symptoms suggesting a tension pneumothorax. The huge cyst was removed easily and satisfactorily by operation. (c) Gross destructive or bullous emphysema of the left lung simulating chronic pneumothorax in a man aged 56. The bullae were removed at operation with an excellent result.



(b)

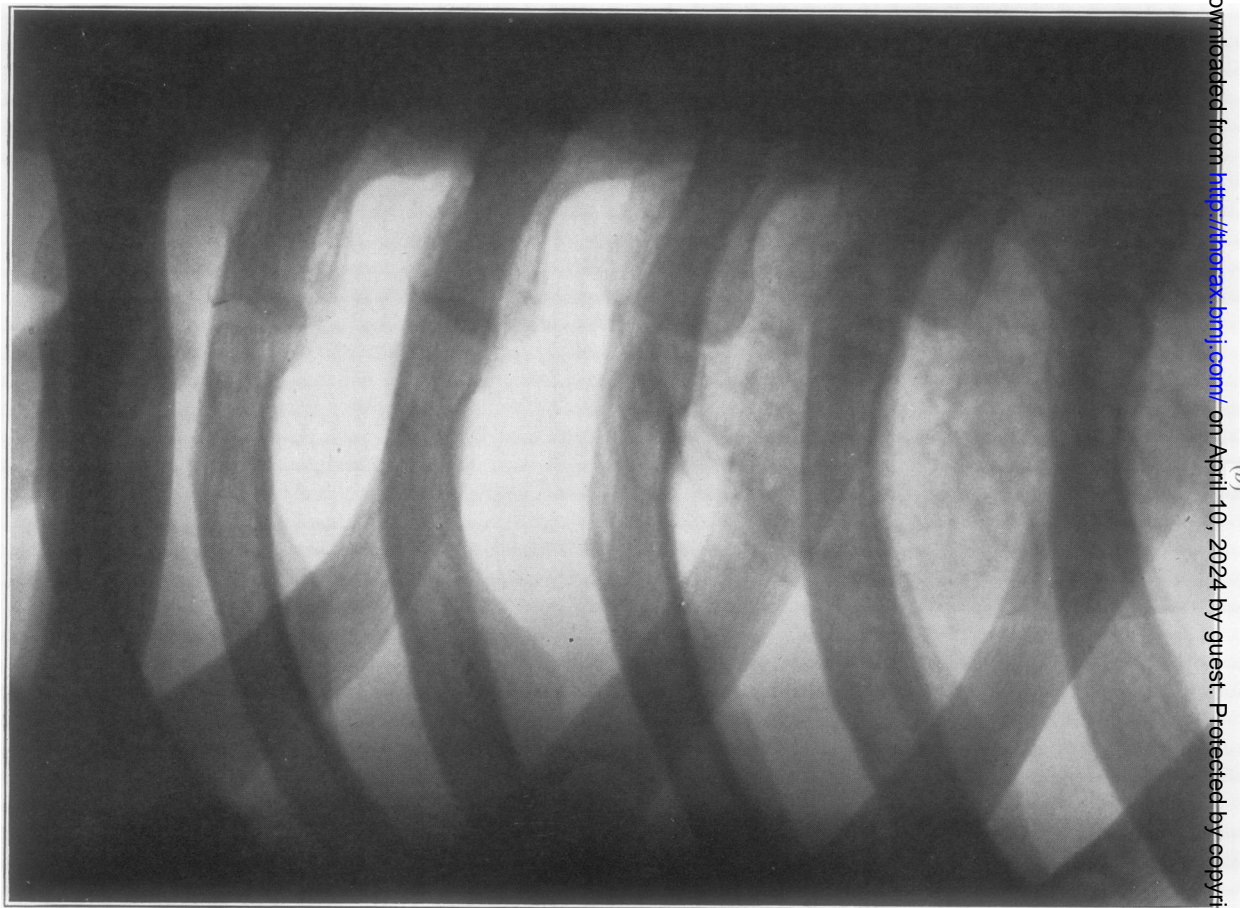
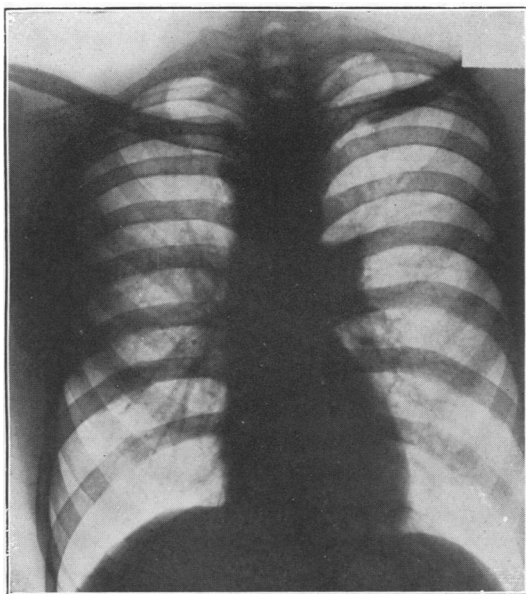
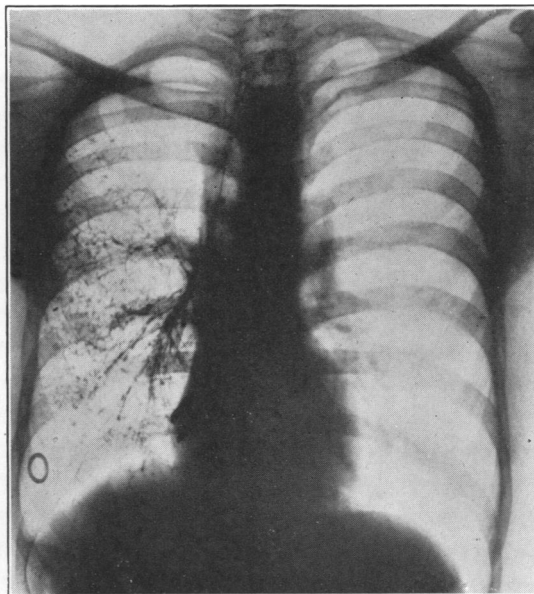


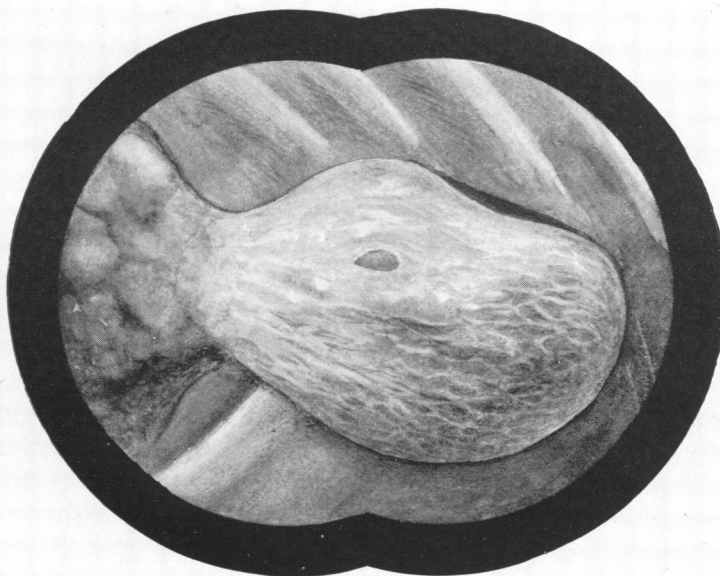
PLATE XXIV.—(a) Radiograph from a case of recurrent pneumothorax in a young man; a small apical bulla is shown, partly hidden by a rib. It was confirmed at thoracoscopy. (b) Radiograph from a case of recurrent pneumothorax in a young man; a small calcified focus and an adjacent scar can be seen in the right upper lobe.



(a)

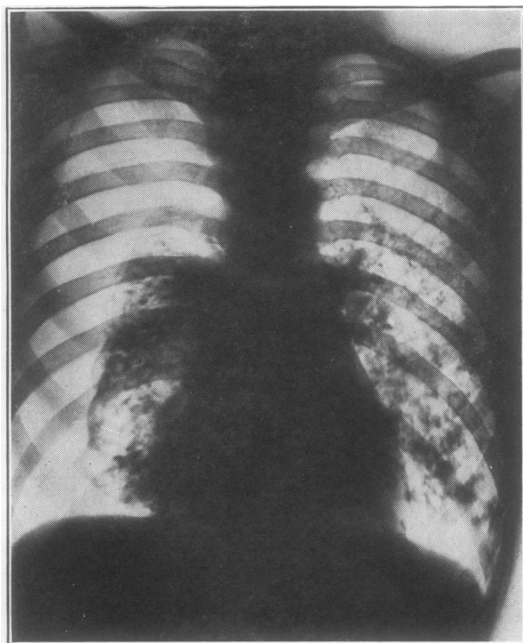


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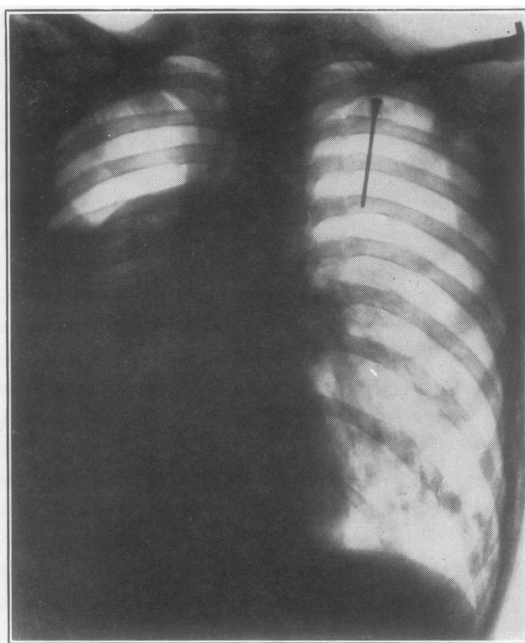


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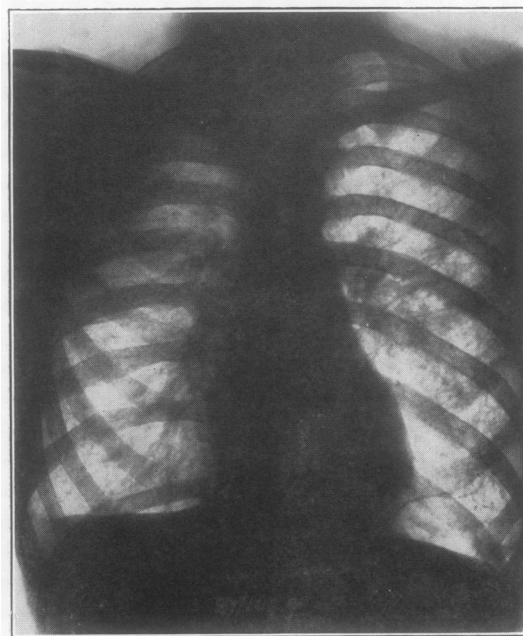
PLATE XXV.—(a) Radiograph from a case of recurrent, alternating pneumothorax in a young man. (b) Right bronchogram of the same patient as in (a); shrunken, atelectatic and bronchiectatic middle and lower lobes are revealed. (c) Thoracoscopic view of a large bulla to show a perforation.



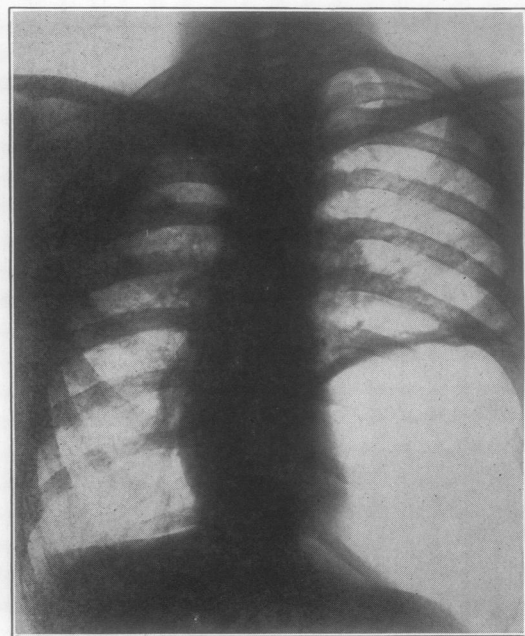
(a)



(b)



(c)



(d)

PLATE XXVI.—(a) Bilateral congenital polycystic disease in a young man; a right pneumothorax is present. (b) The same case. A right pleurodesis has been induced. A left tension pneumothorax has supervened, and an indwelling needle was inserted in order to save life. (c) The same case. The right lung has expanded but a cap of pleural thickening or fluid still remains; the left lung has also fully expanded after pleurodesis. Note the thin-walled larger cysts at the left base. (d) The same case. The cysts at the left base have now become grossly distended so as to simulate a localized tension pneumothorax. They subsided spontaneously.

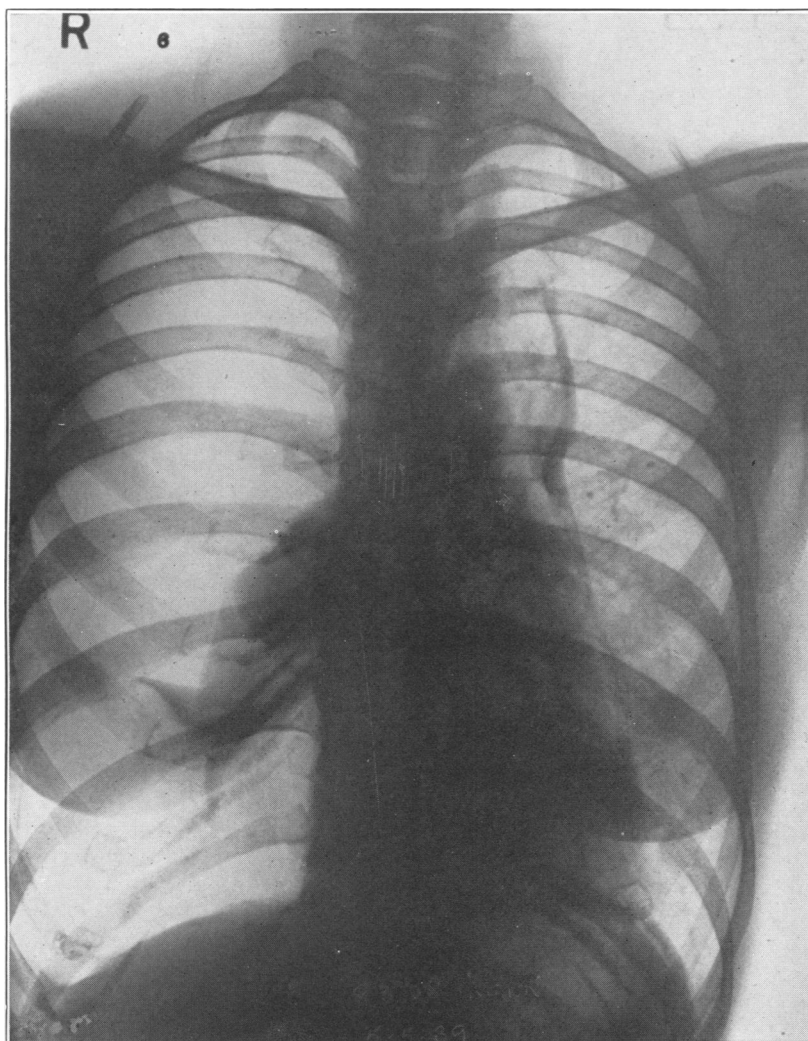


PLATE XXVII.—Radiograph of a right tension pneumothorax which had recurred five times and then became chronic. A huge mediastinal hernia is present. The edge of a large thin-walled cyst can be seen above the right diaphragm.

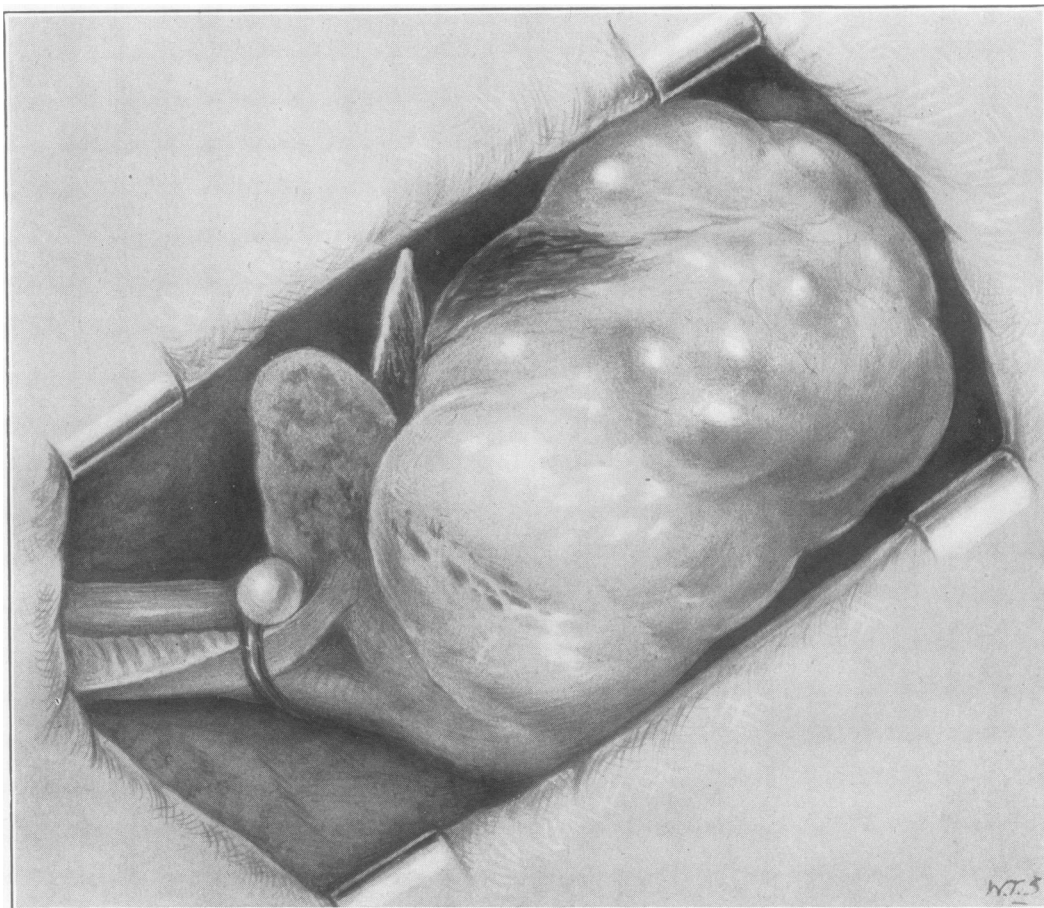


PLATE XXVIII.—A woman, aged 30, with chronic spontaneous pneumothorax; showing the appearances seen at thoractomy.

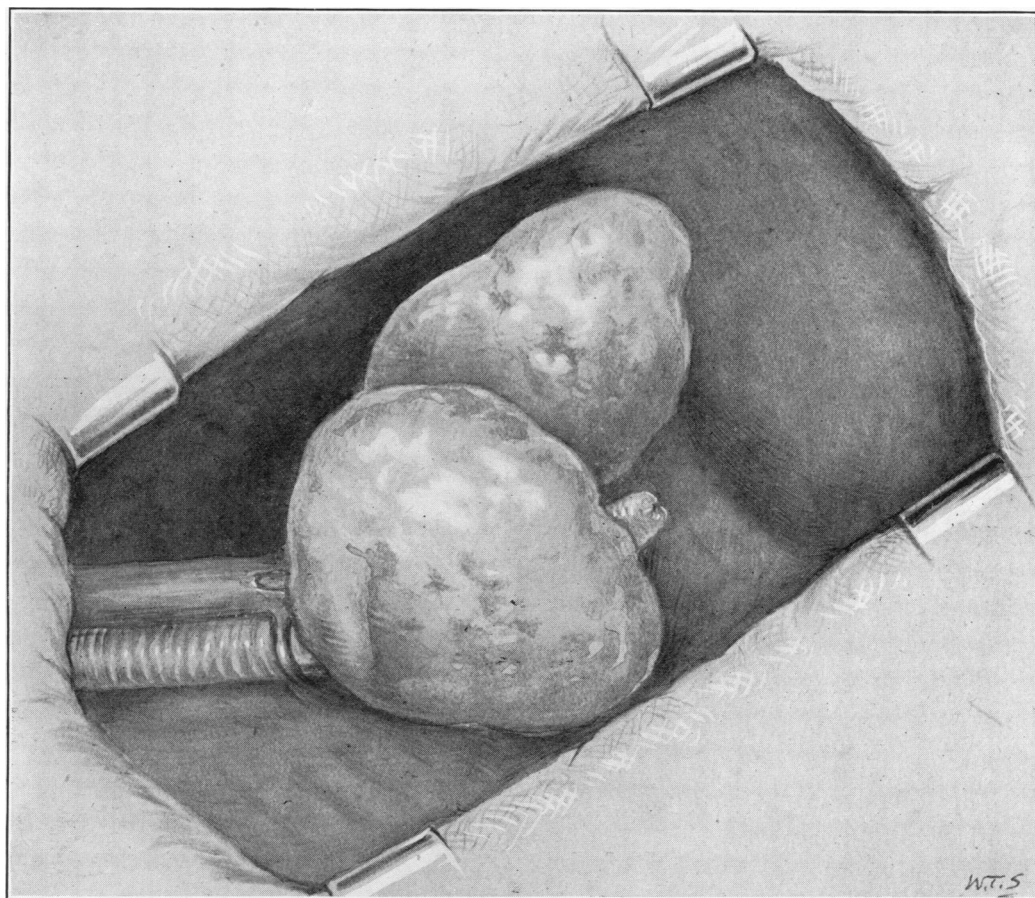


PLATE XXIX.—Showing the cystic lower lobe. (Same patient as in Plate XXVIII.)

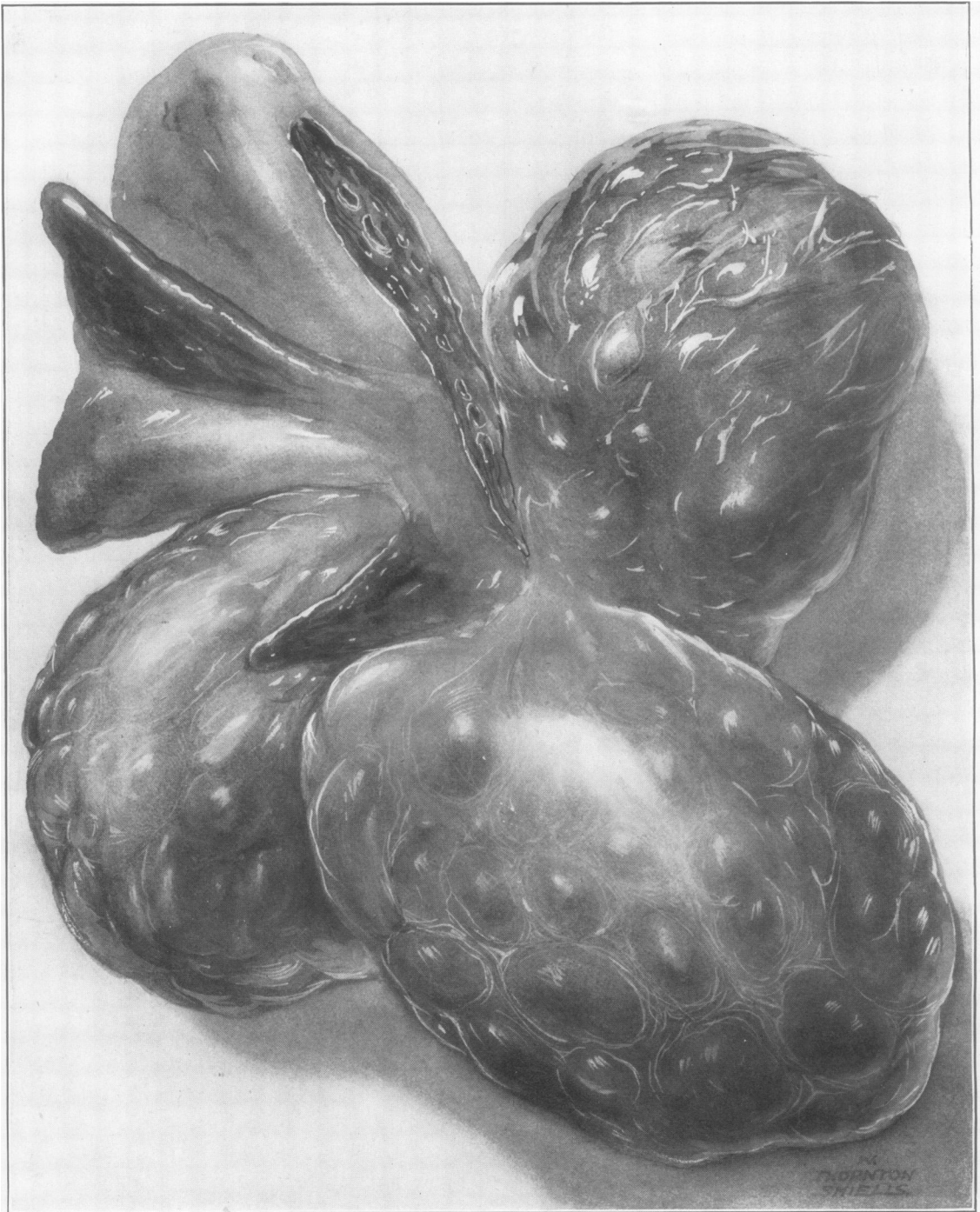


PLATE XXX.—Showing the expansion of the upper and middle lobes that began as soon as the cystic lower lobe had been removed and the partial volvulus corrected. (Same patient as in Plates XXVIII and XXIX.)

information he could give was that some six months earlier he had noticed a vague discomfort in the affected side, and during the last few years he had occasionally found he had been unable to take a deep breath. This pneumothorax was still present four months later and was then treated by pleurodesis. It is by no means uncommon to observe a small pneumothorax on the "good" side which has given no indication of its presence, a chronic or recurrent pneumothorax on the other side dominating the clinical picture.

A striking feature of the severe cases of chronic pneumothorax is loss of weight. This is best illustrated by Plates XXIIb and c, and XXII, which show a patient (a woman aged 26) who had had a chronic pneumothorax for seven months; she suffered from chronic bronchitis and asthma. Her weight dropped from 7 stone 4 pounds to 4 stone 13 pounds, a fall of 2 stone 5 pounds. As will be seen from the photographs, she regained her lost weight as soon as expansion of the lung was achieved. Incidentally, a year after the chronic attack on the right side she suffered a spontaneous pneumothorax on the left side; it is just as well that her right side had been treated and not left to take care of itself.

*Methods of investigation.*—It is scarcely necessary to do more than mention the importance of taking a careful history of previous respiratory disease. Inquiry about tuberculosis is not likely to be omitted, but it is important to remember to ask specifically about asthma; information about attacks in childhood is often not volunteered. Evidence of lower respiratory infection, including bronchitis or bronchiectasis, must be sought. Twenty patients in my series gave a history of chronic bronchitis. In a further five, bronchiectasis was present.

Plain radiography may show nothing more than the pneumothorax, especially if the lung is completely collapsed. The opposite lung may show emphysema; it should always be carefully inspected so that a shallow pneumothorax is not overlooked.

The first thing to consider is whether the condition is a true pneumothorax or whether it is a giant cyst or bulla simulating one. This may at times be very difficult. Plate XXIIIa and b show an example in which differentiation was not easy, although one felt fairly confident the condition was a giant cyst. It will be noted that in Plate XXIIIb there is a great increase in size of the air-space; this occurred in conjunction with an acute attack of bronchitis and closely simulated a tension pneumothorax; the patient was admitted to hospital as an emergency. Presumably the cough-

ing from the bronchitis had caused the cyst to become violently distended. In this case the cyst seemed to arise in connexion with the upper lobe; at operation it was found to spring from the lowest lateral fringe of the lower lobe. Plate XXIIIc shows another example of what might be diagnosed as a tension pneumothorax; this patient also was extremely dyspnoeic. At operation he was found to have two enormous bullous cysts; the lung expanded to fill the chest immediately they were removed.

This recognition of giant bullae or cysts is of great practical importance. It has already been mentioned that the radiographs of some examples reported in the literature as bilateral chronic pneumothorax strongly suggest bilateral apical bullae. This is so in a case recorded by Lewis (1933); in the case report it is stated that needles were inserted in both "pneumothoraces" to observe the pressures, and the patient died suddenly and unexpectedly ten hours later; there was no autopsy. It is almost certain that an autopsy would have revealed bilateral apical bullae, each with a puncture and accompanied by tension pneumothorax on one or both sides. In fact death in such a case should not be "unexpected": if a needle is inserted, a tension pneumothorax is almost certain to follow. The cyst itself usually develops as a result of a check-valve action, and if it is punctured it may continue to deliver air into the pleura with each breath, unless the pleura happens to be completely obliterated. This danger associated with puncture of a bullous cyst is by no means generally appreciated. If it is decided that diagnostic puncture is needed, provision should be made for treatment of the tension pneumothorax that may follow. It is even better, in a case of doubt, to be prepared to do a thoracotomy at once, if radiographs show that a pneumothorax has been caused, without waiting for a severe tension pneumothorax to develop; an event which may be hazardous in the extreme to many of these patients and may be slow to respond even to prompt treatment with an indwelling needle and a water-seal.

It is most important to inspect the plain radiographs, in a case of true pneumothorax, for evidence of cystic disease or bullae in one or both lungs. Failure to recognize the presence of large removable cysts may result in prolonged and needless invalidism. Plate XXIVa shows a small apical bulla in a case of recurrent bilateral pneumothorax in a young man; the bulla was confirmed at thoracoscopy. Experience shows that it is exceptional to detect small apical bullae on plain radiography although they may more often be demonstrated on tomography. Tomography may also reveal other-

wise hidden cystic disease or bullae in one or both lungs; it should not be omitted although it may not help. The plain radiographs should also be inspected carefully for evidence of apical scarring or of old calcified foci, both of which may be significant and causal (Plate XXIVb).

Bronchograms are often revealing and, although perhaps not necessary as a routine, are usually desirable. On three occasions they have revealed quite unsuspected bronchiectasis.

Plate XXVa and b shows such a case, a young man of 21 who had had four attacks of spontaneous pneumothorax on the right side and three on the left; he had virtually no cough or sputum. The very shrunken atelectatic right lower lobe was overlooked in the plain films. It is a fair assumption that the considerable compensatory emphysema of the right upper lobe may have rendered it more liable to give way and leak; this does not, however, explain the attacks on the left side, which showed no bronchiectasis. Bronchograms may also be of help in differentiating between a pneumothorax and a giant bulla; in one case they gave conclusive indication of a pneumothorax; conversely the information they have given has been at times uncertain.

Bronchoscopy is not needed as a routine but is sometimes helpful. In one case considerable rotation and obstruction of the main bronchus was caused by a large cyst moving about inside a huge tension pneumothorax with a large mediastinal hernia.

Intrapleural pressure readings may be valuable in demonstrating conclusively the presence of a fistula, as shown by rapid return of the pressures to the previous reading after withdrawal of air. Most cases of chronic pneumothorax will have had repeated aspirations of air and pressure readings in an attempt to secure lung expansion. Estimation of the oxygen, nitrogen, and carbon dioxide content of the pleural air may also indicate a fistula; this has been done in a few of my patients.

The most valuable and often conclusive examination is *thoracoscopy*, and this should never be omitted; in a bilateral case it may be permissible to waive the examination on *one* of the sides. It is a sound principle throughout the whole of medicine and surgery to examine by direct inspection whenever possible; here we have an example of pleural disease, readily amenable to inspection, and yet thoracoscopy has been used only relatively recently and has still by no means become a routine. With its aid it is often possible to make a complete and certain diagnosis at once; the information thus given may be negative but will nevertheless be useful. Moreover, in many cases it is

the medium of successful treatment in that the first steps towards an artificial pleurodesis can be taken. The information afforded by thoracoscopy will be described in the later parts of this paper. Plate XXVc shows a large bulla with a perforation disclosed at thoracoscopy.

*Types of case.*—Analysis of seventy-one cases investigated shows that about nine groups or types

TABLE IV  
SPONTANEOUS PNEUMOTHORAX: AETIOLOGY

	Type of case	Number
1	Generalized emphysema, 12 cases ..	
	Bullous emphysema 13 ..	
	Total ..	25
1a	Asthma and bronchitis with emphysema ..	8
2	Large solitary bullae or cystic disease ..	11
3	Diffuse polycystic disease ..	3
4	Small bullae (mostly apical) ..	15
5	Apical scar ..	6
6	Leak or tear seen ..	4
7	Areas of "cuckoo-spit" ..	4
8	Various ..	4
	(Tooth extraction 1 ..	
	Staphylococcal abscess 1 ..	
	Drainage of empyema 1 ..	
	Tuberculous pleurisy 1) ..	
9	No cause ..	6

can be identified, although there is some interdependence between them (Table IV). Fig. 1 illustrates these groups diagrammatically.

Although some of these types may have a common mechanical factor, they differ pathologically and illustrate the rich variety of disease conditions of the lungs represented in chronic and recurrent spontaneous pneumothorax; the condition, far from being an entity, is a natural event in a considerable number of lung diseases. It will be simpler to consider the significance of these various conditions when discussing the mode of production of the pneumothorax and the reasons for chronicity and recurrence.

*Mechanics of production and chronicity of pneumothorax.*—A number of authors have contributed interesting, useful, and informative discussions on the mode of production of "pneumothorax simplex": notably Kjaergaard (1932 and 1935), Perry (1939), Ornstein and Lercher (1941–2), and authors who have written on isolated cases or the comparatively rare cases that come to autopsy.

The easiest and most obvious explanation, and the one most generally accepted, is rupture of a small emphysematous bulla or cyst. There have been sufficient post-mortem demonstrations of ruptured bullae in fatal cases to make it certain

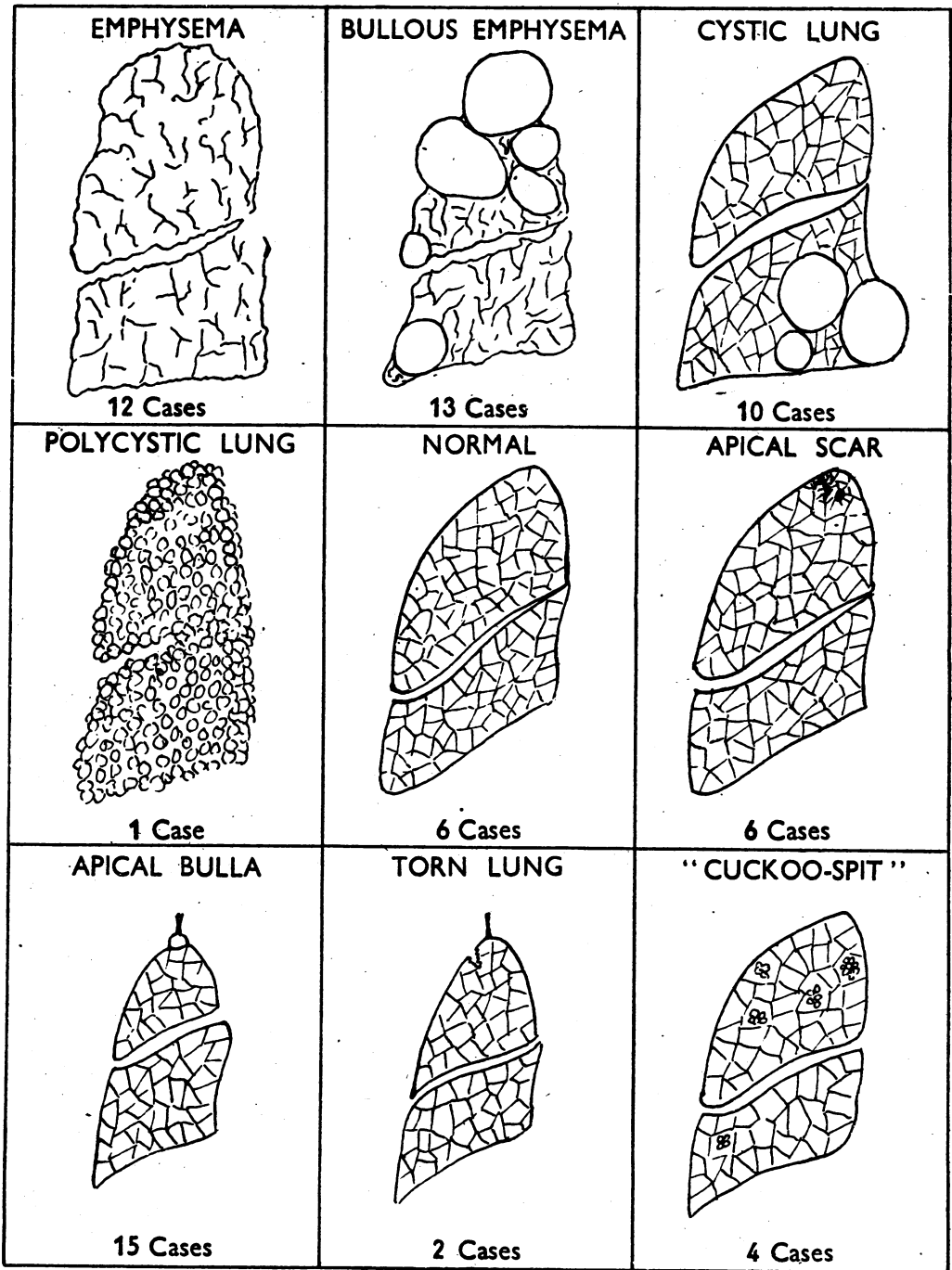


FIG. 1

that they can be the cause. A tear or rupture has been observed in only four of my cases, but a small bulla of the characteristic type has been observed in fifteen cases, a number sufficiently large to be significant. Kjaergaard (1932) has discussed the formation of these small bullae and the valvular mechanism that may be demonstrated in their basal parts. Two illustrations in his paper show this valvular mechanism in cysts taken from post-mortem specimens in which the bullae were found accidentally and had not ruptured to cause a pneumothorax. It is easy to see how such a bulla may be progressively distended by each act of respiration until it bursts; this bursting can occur while the patient is at rest, as indeed happens; the rupture is also liable to be precipitated by any act of straining or violent exercise. According to Kjaergaard the credit for first describing this mechanism belongs to Fischer (1922), and to his pupil Hayashi (1915), who described three cases. Orth had already pointed out that there must be an obstruction to the outlet of air from these bullae as it is difficult or impossible to force the air out of them by pressure. Hayashi demonstrated at the base of a bulla a valve-like structure which was formed of deformed atrophic lung tissue.

Kjaergaard mentions two types, in one of which there is no scar-tissue in the base of the bulla, which abuts directly on healthy or emphysematous alveoli, and another in which the bulla rests on a layer of scar tissue.

This mechanism of production and of rupture of the bulla also offers a simple explanation of why the pneumothorax may remain chronic or may recur; indeed, when one considers the valve mechanism it is strange that spontaneous expansion of the lung occurs so often.

The isolated apical bullae or cluster of small bullae in an otherwise normal lung are generally accepted as being due to healing of an old tuberculous lesion. In some cases (six in this series) a scar may be present without any demonstrable or visible bulla, and it is a matter of assumption that the escape of air has come from a smaller emphysematous area or minute bulla formed secondary to this scar tissue. This is a very reasonable assumption and also introduces the question that will be referred to again later, whether, even when an unruptured bulla of moderate size is observed, it is the actual cause of the leak, since the latter may well have occurred from a nearby, easily overlooked, smaller area of dilated alveoli.

These areas of so-called "local emphysema" lead naturally to a consideration of true emphysema as a cause of pneumothorax. A number of authors have referred to the fact that pneu-

mothorax is a relatively rare complication of emphysema. Kjaergaard has suggested that the communication between the bullae and the adjacent alveoli in emphysema is not usually valvular but is wide and free and that this may explain the rarity of rupture. Nevertheless, if rupture does occur it would be anticipated that it would be a more serious condition with a greater tendency to chronicity and recurrence. This supposition receives support from my cases in which generalized emphysema was present in twelve instances; it is more understandable and significant that gross bullous emphysema was present in a further thirteen, making a total of twenty-five cases. The differing role of emphysema in pneumothorax simplex and in the chronic and recurrent groups is further reflected in the greater representation in the higher age groups (Table III). The fact that eight of these patients also suffered from asthma and chronic bronchitis is of further significance, and it is also noteworthy that one of these patients was aged 17, two were in the second decade, and three were in the thirties. In a bare inspection of the age incidence they would not be differentiated from the cases of benign pneumothorax in young adults.

When gross bullous disorganization of the lung accompanies generalized emphysema, the nature of the bullae is certain. There is less certainty of the nature of the large bullae or cysts that may occur in otherwise healthy lungs. It is easy to class them all as congenital, and there is little doubt that many of them are. If other congenital anomalies, such as aberrant rib formation, are observed, this gives strong support to the congenital origin. In others there is no such ready confirmation and the association of irregular clusters of bullae of various sizes with dense interlacing bands of scar tissue and dense pleural plaques strongly suggests that they are the end result of some severe destructive inflammatory process in the lungs. They may even be gross examples of post-tuberculous scarring such as is commonly seen in smaller foci at the apex. Multiple cysts occurring without fibrosis are more likely to be truly congenital.

These cysts or bullae occur in either the upper or the lower lobe. Even if the cysts themselves are not congenital they may arise from a congenital weakness of structure of the lungs that favours their production and therefore their continued increase in size and final rupture. In one of my patients, a woman aged 23, a number of clusters of very thin-walled bullous cysts were present in the lower lobe, and at thoracotomy a very striking phenomenon was seen; the cysts

were not tense but were lax and collapsed completely on expiration and became distended with inspiration. It required little imagination to see how a sudden cough, strain, or violent exertion could lead to their rupture.

Allison (1947) has recently pointed out that certain of the giant cysts or bullae are caused by a defect in the wall of a bronchus of moderate size and that the hole can be seen in the floor of the cyst.

In addition to single congenital cysts, diffuse cystic or polycystic disease may be present; this may be unilateral or bilateral. Such was the case in the patient whose radiographs are shown in Plate XXVI. This patient came into hospital with a pneumothorax on the right side (Plate XXVIa); it was the eighth on this side, and he had had two on the left side. In spite of the small cystic nature of the disease, bronchograms showed no bronchial dilatation, and at thoracoscopy the lung could be seen studded with innumerable thin-walled cysts of varying size, but all small. Clearly the cystic degeneration or malformation affected chiefly the alveolar tissue or bronchioli.

Artificial pleurisy was induced with silver nitrate on the right side, and while this was proceeding he developed yet another spontaneous pneumothorax on the left side, making eleven attacks in all. This was a tension pneumothorax, and only prompt insertion of a needle attached to an under-water seal saved his life (Plate XXVIb). After the right lung had expanded he suffered yet another attack on the left side and this was then treated with silver nitrate. The condition after bilateral pleurodesis can be seen in Plate XXVIc; it should be noted that some larger cysts are present at the left base. A most interesting and significant phenomenon has since been observed on the left side. The large cystic condition at the left base increased in size, and on one occasion when he complained of distress and shortness of breath a radiograph (Plate XXVIId) showed gross distension of the cysts in the left lower lobe. In a later radiograph these became smaller, and since then they have contracted still more so that the present appearance resembles that seen in Plate XXVIc. This is clear proof that the cysts were responsible for the recurrent pneumothoraces, and suggests that these big basal cysts were the cause of the left tension pneumothorax. Once his pleura had been obliterated they were unable to rupture, and so they distended to a huge size and presumably may do so again. The former spontaneous pneumothoraces may have acted as a safety-valve and spared the lungs. It may become necessary to operate and resect this

cystic area at the left base in order to spare him progressive compression of the relatively healthy lung above.

This progressive distension after pleurodesis has been seen in two other cases in this series. One was a patient who had a small apical bulla about 2 cm. in diameter. At a recent follow-up this was noticed to have grown to some 6 cm. in diameter. Presumably the pleurodesis spared him a succession of recurrent pneumothoraces; the bulla is as yet too small to be of significance. In the other patient, a woman aged 30, who had had three attacks on the right side over a period of twelve years and whose last attack had lasted six months, thoracoscopy revealed a moderately large thin-walled cyst in the right lower lobe. After some hesitation it was decided to try the effect of pleurodesis rather than lobectomy. After pleurodesis this cyst not only persisted but grew larger. As she complained of pain and discomfort and of recurrent pyrexial attacks with bronchitis, the lobe was removed. She has since been quite well.

These experiences and observations indicate the natural history of the condition and the fact that radical treatment will usually be needed when a cyst is present. Kjaergaard (1935) amplifies his earlier paper by describing two cases studied post mortem in which a pneumothorax was caused by rupture of congenital valve vesicles on the surface of the lungs; in both cases cysts were present deep in the lungs as well as on the surface. The condition was comparable with the case illustrated in Plate XXVI. Of great interest in connexion with cystic disease is the occurrence of *familial pneumothorax*, a number of examples of which are recorded in the literature and are detailed by Kjaergaard. Götzsche (1933), for instance, describes a remarkable series of five in the same family. Bachmann (1940) reports the occurrence in a father and a daughter. Two patients in my series had near relatives (in one case a sister) who had had a spontaneous pneumothorax. The assumption is that the attacks are due to rupture of hereditary congenital lung cysts.

It is surprising that a tear or hole was seen in only four cases, but in many there were sufficient adhesions present to render complete examination impossible. These adhesions themselves may in fact be a cause of chronicity or recurrence, in that they may cause the lung to tear as it collapses and the tear is then held open. This has actually been observed at thoracoscopy; and in one case, in spite of the application of silver nitrate, the lung would not expand; the adhesion was accordingly divided and the lung at once expanded. If a controlling adhesion is seen next to an area of

scarring or bullous change, it should be divided so as to allow a tear or rent to become relaxed and to heal.

In four of my cases I was able to observe a remarkable condition that has not been described before, although at least one author has hinted at it. In none of these four could bullous or cystic change be seen either radiographically or at thoracoscopy; one patient (a man aged 58) had generalized emphysema; another (a man aged 34) had bronchiectasis and mild emphysema; a man aged 32 had no other evidence of abnormality in the lungs; and the fourth (a man aged 35) had emphysema, asthma, and bronchitis. The most careful inspection of the pleura at thoracoscopy failed to show any rupture or leak, but a number of scattered foci could be seen in which a few tiny air bubbles (each less than a millimetre in diameter) could be seen on the lung. It is not uncommon to see small air bubbles on the moist lung surface in a pneumothorax, but when the patient was instructed to hold his nose and blow up his cheeks these clusters could be observed time and time again to increase not only in size but also in the number of bubbles. Each cluster would grow so as to form a tiny area resembling the froth one often sees in plants in early summer and which is known as "cuckoo-spit." I have applied this name to the condition. These areas are caused by a minute alveolar leak; this leak is certainly in some cases only into the subpleural tissues; but, with an unsupported pleura, rupture can occur and an intrapleural leak follows. These foci are numerous and probably changing, and the observation strongly suggests that in certain cases of chronic and of recurrent pneumothorax in which no definite or gross leak, such as a ruptured bulla, can be demonstrated, the mechanism of escape of air is multifocal, changing from time to time, and it is so minute as to escape detection except with the closest inspection with a thoracoscope held almost up to the lung surface. It is doubtful if the mechanism could be observed in the post-mortem room, and it may explain cases such as those described by Rolleston (1900), Priest (1937) and others in which death occurred from pneumothorax and yet a most careful inspection failed to reveal any abnormality in the lungs.

This mechanism may also be the one actually responsible even when a large bulla or cyst is seen, because I have often observed that the bullae are tense and intact, not collapsed and leaking. Pitt (1900), in addition to describing the post-mortem finding of a torn adhesion attached to a torn bulla in a fatal case, mentions another fatal case: that of a man aged 54 who had chronic bronchitis and

asthma and whose lungs contained many bullae, but no actual hole could be demonstrated even though the bronchi were carefully distended with water.

Schmincke (1928) reports an autopsy on a young man who died of bilateral spontaneous pneumothorax and had many emphysematous blebs in both lungs. He believed that a peripheral zone of embryonal tissue persisted which did not differentiate into alveoli; he describes and depicts such an embryonal area which in some areas did not progress to its final development into mature alveoli. This observation seems to support the conclusion I have come to that in certain cases the incidence of the spontaneous pneumothorax is due not to any gross disease, either focal or general in the lungs, but to a congenital (or possibly acquired) defect of quality that renders the pleura liable to leak or rupture easily in many isolated, minute, and changing places. In other words these patients have "leaky lungs" or "porous pleura."

This would seem to be the only explanation for the six cases in my series in which no abnormality could be observed or demonstrated either radiographically or at thoracoscopy. In one (a woman aged 35 who had three attacks on the right side, four on the left, and also a chronic pneumothorax on the right lasting six months until obliterated by pleurodesis) I inspected the pleura four times with a thoracoscope and tried a variety of ways, such as coughing, straining, and applying constant suction, to try and demonstrate a leak. The lung seemed perfectly healthy.

That a defect in the quality or strength of the lung tissue probably exists is shown by another case, that of a woman aged 44 who had had a pneumothorax for four months. Fig. 2 shows the appearance of bullous or cystic change observed at thoracoscopy. A small hole, which may have been caused by the local anaesthetic needle, was seen in the upper lobe. As the lung was being painted with a swab moistened with silver nitrate the swab-holder slipped and fell gently a distance of 2 or 3 cm. on to the surface of the lung; another hole appeared at once. No normal lung would rupture in this way, and it is beyond reasonable doubt that the lung tissue, including the pleura, was extremely fragile. Incidentally the lung was very slow to expand in this case; several applications of silver nitrate were needed, and it took six months to fill the chest.

This defect in the tensile strength of the lungs can well be due to imperfection of the elastic tissue, which may vary considerably in quantity and is noticeably absent or defective in emphysema. The formation of bullae is probably closely

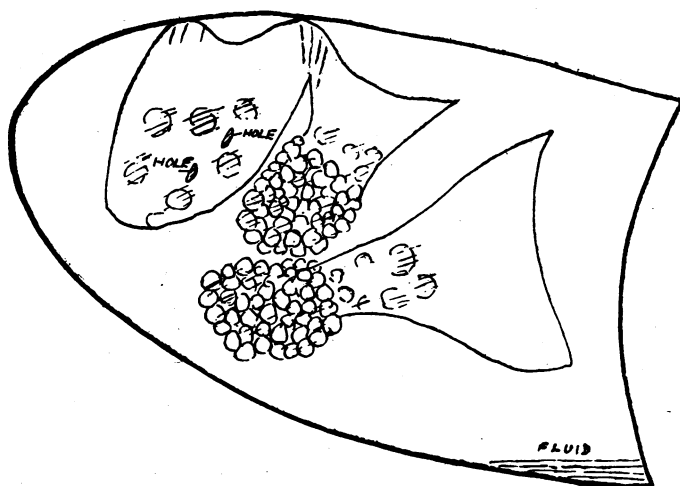


FIG. 2

connected with rupture of the elastic supporting and strengthening layer. Zahn (1891) measured the combined thickness of the alveolar wall and pleura and showed that whereas in the normal it is 0.13 to 0.24 mm., in emphysematous lungs it may be as little as 0.05 or even 0.03 mm.; he suggested that this atrophy of the visceral pleura might be one cause of pneumothorax.

*Summary of the causation of pneumothorax.*—The cause of pneumothorax, considered with particular reference to recurrence and chronicity, may lie in any one of several anatomical sites. It may be within the pleura, as when an adhesion causes the lung to tear and holds the rent open. It may be pleural as when the elastic supporting layer is defective in quality or quantity. It may be subpleural, when there is rupture associated with bullae arising either in connexion with an old scar or with destructive emphysema; subpleural leakage may also occur from defective alveoli, possibly congenitally maldeveloped. The major disease may lie in the alveoli throughout the lung, as in generalized emphysema, or it may be in the bronchioli or smaller bronchi as in congenital cystic conditions. Finally the larger bronchi may be responsible, as in the case of giant valvular bullae resulting from defects in the wall of the bronchus itself. Chronic diseases of the bronchi, such as chronic bronchitis, asthma, and bronchiectasis, also play their part.

#### TREATMENT

In the past the treatment of chronic and recurrent pneumothorax has been governed largely by *laissez faire*; or perhaps, equally unsatisfactory,

a mistaken assumption of an underlying tuberculous basis has resulted in the patient being condemned to wasting many weary months in bed. Even to-day patients stand in peril of being sent to a sanatorium. Conservative expectant management of a first attack of "pneumothorax simplex" is sound: the lung usually expands quickly and safely in its own time. If the rate of expansion is slow and prolonged and the condition passes into the stage of chronicity, or if relapses occur, then there can be no question that active treatment is needed. It is wasteful of the patient's time and of valuable beds to keep him immobile when the cause of his disability rests on purely mechanical factors that can be corrected. I appeal, therefore,

for active treatment of these cases.

All good treatment is founded on a knowledge of the cause of the disease, and often rests largely upon a consideration of the morbid anatomical changes present. It follows that treatment should not be empirical, for the analysis of these cases shows that a number of different causes may be acting. Each case must be fully investigated in the manner already detailed, and thoracoscopy in particular should never be omitted.

The most important first step is the recognition or exclusion of localized large cystic or bullous disease, as distinct from the simple small apical bulla. It is useless to persist in conservative treatment if a large cyst or bulla is present; it is also wrong to rely upon pleurodesis, because the cyst remains and may increase in size. The correct thing to do is to excise the cyst or cysts either by lobectomy, or, if they are conveniently sited, by local resection. I have had to do this in eight cases, which, so far as I know, is the largest number treated in this way.

Bigger (Horsley and Bigger, 1937) describes a case in which he resected a small, leaking emphysematous bulla in a case of chronic pneumothorax. Sycamore (1936) and later Tyson and Crandall (1941) report a single case of resection of a small, unruptured bulla in a man aged 30 who had had five attacks of spontaneous pneumothorax. Hoyer and Clagett (1946) performed middle lobectomy for cystic disease associated with chronic pneumothorax of six months' duration in a man aged 41. My own first case was operated upon by right lower lobectomy in May, 1939, and is so rich in features of practical

value and interest that it deserves recording in more detail.

Mrs. S. M., aged 30, was first taken ill in February, 1934, with an attack of pain above the right breast for three weeks; a few months later she had a second attack, and in December, 1935, a third one, in which she was dyspnoeic. The dyspnoea persisted, and two months later she had a further relapse and the presence of a spontaneous pneumothorax was recognized for the first time. A diagnosis of tuberculosis was made, and a four months' pregnancy terminated; she was kept in bed until May, by which time the right lung had expanded. In July, 1937, she had another slight attack of pain, and in April, 1938, when she was eight months pregnant, she had yet a further attack of severe dyspnoea with fever and cough. The dyspnoea persisted and was very severe when her child was born in June. After this she continued an invalid existence with a troublesome cough and crippling dyspnoea; her weight fell by a stone and a half.

In April, 1939, she was seen by Dr. C. H. Hoyle, who noted that she was so short of breath as to be unable to converse for long at a time. Her vital capacity was 1,000 c.cm. and she had a right total pneumothorax with a large mediastinal hernia. Dr. Hoyle recognized what had been consistently overlooked up till then, that the outline of a thin-walled bulla or cyst could be made out in the lower part of the chest (Plate XXVII). This could be seen to move medially and laterally on inspiration and expiration, or with change of position.

At thoracoscopy the presence of a large thin-walled bullous cyst was confirmed; it sprang from the lower lobe and had a white, opaque, smooth surface with a scar-like patch near the centre. Nearby two smaller bullae could be seen.

At bronchoscopy the carina was deformed to a Z-shape and the right bronchus so flattened and distorted, as if compressed and rotated forwards and downwards to the left, that the bronchoscope could not be passed into it.

Thoracotomy was performed in May, 1939, and a large tri-lobed cystic condition was displayed in the lower lobe; the huge mediastinal hernia allowed the cyst to fall forwards medially so that the right bronchus was twisted through a right angle (Plate XXVIII). A condition of partial volvulus was thus produced, and this explained the bronchoscopic findings. The upper and middle lobes did not aerate, even with considerable pressure by the anaesthetist. The lower lobe was removed with the contained cysts, and as soon as this was done the partial volvulus was corrected and the upper and middle lobes at once began to aerate (Plate XXIX). A small cyst about 2.5 cm. in diameter attached to the hilar region of the upper lobe was removed. The main cystic mass is shown in Plate XXX. She made an uninterrupted recovery from the operation, although the upper and middle lobes seemed to be unable to fill the whole hemi-

thorax; for, though they expanded to the chest wall, some mediastinal shift to the right occurred and has persisted.

The change in her condition after this operation was dramatic; she soon resumed a busy normal life, and within five months regained her lost weight. Two years later she went through a normal pregnancy and delivery. During the war she looked after three evacuated children in addition to her own family. She was also able to resume singing, which had been quite impossible while she had her pneumothorax.

She continued well until September 10, 1947, when she experienced pain in her left chest; later dyspnoea occurred and she was found to have a spontaneous pneumothorax on the left side. When the radiographs of her earlier illness were being reviewed it was noticed, for the first time, that she had had a small pneumothorax on the left side during convalescence from her operation. This had been quite symptomless, but it emphasizes how easily a shallow pneumothorax can be missed unless it is deliberately looked for; this should always be done on the contralateral side when a spontaneous pneumothorax is present.

Thoracoscopy revealed two small bullae, one attached to the upper lobe and about 1 cm. in diameter; the other attached to the lingula and with a haemorrhagic wall. Both were painted with swabs of 20 per cent silver nitrate solution, which was also applied to the surface of the upper and lower lobes. An obliterative pleuritis was thus set up and the lung expanded after two aspirations of air and fluid.

This case has many points of interest; the long history of recurrent attacks (at least five in all) over a period of five years had resulted in much suffering and disability and had brought the patient to a state of invalidism. Because of an erroneous diagnosis of tuberculosis, her child had been sacrificed. The policy of *laissez faire* in regard to the pneumothorax is typical. She had been seen by a number of physicians over several years, and the presence of the cyst had been overlooked in the radiographs: this would not have mattered if she had been submitted to thoracoscopy, for the cysts must then have been revealed. The successful treatment by lobectomy is of interest for, so far as I know, this is the first occasion on which this operation was used in the treatment of chronic or recurrent pneumothorax. Hoyer and Clagett's (1946) case was not operated on until 1945, by which time I had performed lobectomy on six more such cases. Finally, there is the development of the contralateral spontaneous pneumothorax and its treatment by the induction of a chemical pleuritis.

I have, in addition to these eight cases, performed lobectomy or local excision of giant bullae or cysts in a further six cases without pneumo-

thorax, although several of these simulated chronic pneumothorax. It would seem that the very large cysts are not so liable to leak or rupture as the smaller ones, possibly because they receive support from their firm apposition to the chest wall. Their very size protects them.

When the presence of a cyst or large bulla has been excluded the logical treatment would appear to be pleurodesis; that is, obliteration of the pleural space by some artificial means so as to prevent relapse, or to achieve expansion of the lung in a chronic case.

Table V shows the methods of treatment used in this series. For various reasons, such as refusal,

TABLE V  
SPONTANEOUS PNEUMOTHORAX: TREATMENT

<i>Lobectomy</i> .. .. .	8
<i>Pleurodesis</i> .. .. .	53 patients
11 bilateral .. .. .	64 sides
Silver nitrate .. .. .	52 patients
9 bilateral .. .. .	61 sides
Poudrage .. .. .	3 sides
<i>Cauterization of adhesion</i> .. .. .	2 patients
<i>No treatment</i> .. .. .	12 patients

no treatment was given in twelve patients. In two cases adhesions were divided. In fifty-three patients pleurodesis was done; eleven of these were bilateral cases, and so sixty-four sides were treated in this way. Poudrage (using iodized talc powder, after the method of Bethune, 1935) was used in three cases, but in fifty-two silver nitrate was used. Two patients had poudrage on one side and silver nitrate on the other and were emphatic that the silver nitrate was less disturbing. It has also the great advantage that after the initial application at thoracoscopy further treatments can be given very simply by injection while the patient is in bed. Moreover, in bilateral cases it is not obligatory to thoracoscope both sides and a simple intrapleural injection can then be given on the second side. Poudrage requires thoracoscopic application in the theatre.

I first used a solution of silver nitrate to produce pleurodesis in January, 1936, in a case of chronic pneumothorax. Adams (1933) described its sclerosing action when applied to bronchial fistulae, and it occurred to me that it might be equally effective in closing the hypothetical alveolar-pleural fistula. Actually no hole could be seen, but the lung was swabbed with a solution of 20 per cent silver nitrate and a progressive obliterative pleurisy followed. As a result of this I used it in other cases of chronic and recurrent

pneumothorax and also in producing pleurodesis before lobectomy (Brock, 1942). I have since learnt that Morlock described its use in a case of chronic pneumothorax in 1933; he used 5 ml. of a 1 per cent solution.

According to Hennell and Steinberg (1939), the idea of induction of chemical pleuritis in cases of chronic or recurrent pneumothorax first occurred to Spengler (1901). In 1906 Spengler suggested using 0.5 per cent silver nitrate; in 1919 he advised using 30 per cent glucose solution for the same purpose; but it was not until 1923 that he reported his first clinical case. In this he used 30 ml. of a 30 per cent solution of glucose in repeated doses and obtained a cure in four months. In his report he suggested that either larger amounts of 30 per cent (up to 100 ml.) or smaller amounts of 50 per cent should be used. Kenner (1932) succeeded with 20 ml. of 0.5 per cent silver nitrate after failing with 30 per cent glucose.

Harvey (1938) reports two cases of spontaneous pneumothorax in asthmatics; in one the pneumothorax had been present for three months and was treated with 40 ml. of 50 per cent glucose, which was administered twice; on each occasion severe pain and a febrile reaction was caused.

Hennell and Steinberg report five cases of chronic or of recurrent pneumothorax treated by the induction of a chemical pleuritis; this is the longest series recorded up till now. Three of their patients were treated with iodized poppy seed oil; one was treated with a hypertonic glucose solution, but this failed, and oil was then used; the fifth was successfully treated with hypertonic glucose solution. They state that it is best to use a 67 per cent (saturated) solution in doses of 50 or 60 ml. A 50 per cent solution may be inadequate, but the saturated solution is more likely to be effective and is just as safe. They have used as little as 25 ml. and as much as 60 ml. with equally satisfactory results; the size of the pneumothorax has been the rough guide to the amount used; 50 ml. was the average dose. When a glucose solution failed, iodized oil succeeded, but with a much more acute reaction, with more constitutional symptoms, and more fluid.

Hennell and Steinberg state that their review of the literature disclosed reports of no more than twelve cases of spontaneous pneumothorax treated by induction of chemical pleuritis. The largest single series before their five cases comprised three cases and was reported by Schott (1934, 1935), who used 0.5 ml. of oil of turpentine in one case and a 50 per cent solution of glucose in two cases.

Other oily preparations have been used in isolated cases by various workers; gomenol in olive

oil (Chandler, 1939) or merely plain olive oil has succeeded. I have used gomenol in olive oil without success on two patients and have then succeeded with silver nitrate. So far no author has described a sufficiently comprehensive series treated with simple oil to prove the claims of this substance. Iodized oil, as used by Hennell and Steinberg, seems to provoke just as severe and painful a reaction as silver nitrate. The effectiveness of hypertonic glucose solutions is also in some doubt; several injections are needed, and they are certainly painful and disturbing (Kjaergaard); some authors state that morphia should be given before the glucose injection.

Another alternative is the use of venous blood; I do not know what quantity is needed to ensure total obliteration, but Watson and Robertson (1928) record a case in which 300 ml. of blood was used.

While the method of pleurodesis by a chemical irritant is undoubtedly practical and efficient, I do not press the claims of silver nitrate against other substances; but I have found it so effective in treatment that I have been loth to change. The pain and general reaction caused are the one serious objection to its use. I am planning to try copper sulphate as an alternative, but to produce a satisfactory obliterative pleurisy a fairly high degree of acute pleural inflammation must be invoked, and this will inevitably cause pain and discomfort. Hypertonic glucose solutions, iodized oil, and blood also cause similar severe pain and general disturbance. The amount of pain caused by silver nitrate varies; it may be severe and require morphia for its control; it may be relatively slight and be relieved by a simple analgesic such as aspirin. When the silver nitrate is applied directly to the lung under thoracoscopic control no pain is caused unless the solution comes into contact with the parietal pleura. If it is injected, pain is more likely to occur.

For injection into the pleura I use 5 or 10 minims of a 10 per cent solution; for direct application to the lung at thoracoscopy three or four small swabs are used, dipped in a 20 per cent solution. A febrile reaction follows, rising and falling over about five days and reaching a maximum of 100° to 101° F.; an effusion of varying size accompanies it. Sometimes a superadded spontaneous pneumothorax occurs, due to superficial necrosis, but this has never caused any anxiety or been more than temporary. The results have been extremely satisfactory in all the 61 cases (total number of sides) in which it has been used. In only two has the rate of expansion been slow, but further injections and patience have succeeded.

The method must be used intelligently and carefully. Many people seem to imagine that all that is necessary is to inject the solution into the pleura and do little or nothing else. First of all it is necessary to ensure that active silver nitrate reaches the pleura, and this is done by inserting a needle and then injecting the solution from an all-glass syringe; a glass-metal syringe must not be used. Next, air and liquid must be aspirated intelligently to get the pleural surfaces in apposition so that they will adhere while they are inflamed. If the pleural surfaces are left separated by liquid, they may become fibrous and inert by the time the liquid has been absorbed. Moreover it is asking too much to expect total obliteration of the whole pleura with one injection of silver nitrate; this may occur, but it is uncertain. After four weeks an attempt should be made to reinduce a pneumothorax, and if a pocket is obtained a further injection should be made. Unless the pleura is tested in this way relapse may follow.

In cases of chronic pneumothorax it is usually not possible to aspirate liquid to achieve rapid lung expansion, and slower obliteration must be expected. In such cases it is best to achieve a summation of effect by repeating the injection at ten- or twenty-day intervals, aspirating air and liquid meanwhile, until obliteration is achieved. Some of my colleagues tell me they have greatly accelerated the rate of lung expansion by introducing a small tube through the thoracoscopic cannula and leaving it in place for twenty-four or forty-eight hours so that continuous suction can be applied.

#### FOLLOW-UP

Of the fifty-nine patients in whom active treatment was carried out, eight had lobectomy or local resection of a large cyst (one case), and fifty-three were treated by pleurodesis; two of the lobectomy patients underwent pleurodesis as well (one on the lobectomy side, one on the opposite side), and this explains the apparent discrepancy in the figures. One of the lobectomy patients died three months after operation from a tension spontaneous pneumothorax (which was unfortunately not diagnosed) on the opposite side; the remaining seven patients are alive and well. The patients treated by pleurodesis have been followed up to date, with the exception of eight who have been lost sight of after periods ranging from six months to two years. In all these eight cases the state of the pleura had been satisfactorily tested after pleurodesis in an attempt to confirm obliteration; one of these, a man who had had bilateral pneumothoraces and who refused to allow the left side to be obliterated,

had a further attack on the left side during the two years he was observed; he has not been seen since 1942. None of the remaining forty-five patients, all of whom have been followed up to date, has had a relapse. Four have died; one, a boy aged 12 years at the time of his chronic bilateral pneumothoraces due to congenital polycystic disease, died of acute pulmonary tuberculosis two years after the onset of the pneumothoraces. The remaining three patients died from bronchopneumonia, or acute bronchitis and cardiac failure at four years, eighteen months, and six months after the pleurodesis. Two of these had suffered for years from asthma, chronic bronchitis, and emphysema, the third suffered from chronic bronchitis and emphysema.

The treatment of a disabling chronic pneumothorax or a troublesome relapsing or recurrent pneumothorax by the pleurodesis given by silver nitrate is one of the most satisfactory and effective methods we have at our disposal, and I can confidently recommend it as the method of choice once gross cystic disease has been excluded. It is almost entirely free from risk; the principle of production of a chemical pleurisy is a sound one; the major objection of the painful reaction that it can cause may well be answered by the introduction of a kinder pleural irritant. The fundamental principle, namely that of the production of pleurodesis, will, however, remain.

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