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SPONTANEOUS PNEUMOTHORAX*

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The escape of air into the pleural space was recognized long before Itard in 1803 applied the name of pneumothorax to this condition. Boerhaave as long ago as 1724 described a fatal case of bilateral leak of air into the pleurae due to rupture of the oesophagus. Houghton in 1832 noted the beneficial effect of spontaneous pneumothorax on some cases of pulmonary tuberculosis. The application of artificial pneumothorax to the treatment of this disease and the experience of the effects of open and closed chest wounds gained in the war have increased our knowledge of the effects of pneumothorax and led to notable advances in treatment, while the developments in x-ray technique have simplified the problems of diagnosis.

Until recently there have been many misconceptions about the causes of spontaneous pneumothorax and their relative frequency. The striking, almost catastrophic, results of this condition in advanced pulmonary tuberculosis, and especially when a valvular opening is formed, have dominated the clinical picture given in textbooks, and led to a preponderating role being given to this disease in its aetiology—to such an extent that in many less severe and dramatic cases latent or healed pulmonary tuberculosis has been invoked as the cause on very slender or even hypothetical evidence. It should be generally recognized that pneumothorax may occur with comparatively slight symptoms at the onset, though these may be ingravescent and progress to the more typical severity in a few hours. It is certain that incomplete spontaneous pneumothorax is often diagnosed only after x-ray examination, and it is therefore probable that many cases are not recognized or even suspected. It is also becoming apparent that spontaneous pneumothorax arises not infrequently in apparently healthy persons of any age in whom after careful investigation no evidence of tuberculosis or emphysema is found. The forms we are specially concerned with are those in which air enters the pleural space from some spontaneous tear in the visceral pleura, and for the purposes of this discussion we may consider:

1. Pneumothorax in the apparently healthy—pneumothorax simplex.
2. Pneumothorax in the course of pulmonary tuberculosis.
3. Pneumothorax from other causes.

Pneumothorax Simplex

This subject has attracted less attention in this country than in France, Denmark, and America. It is one which merits discussion, if only to draw attention to the facts that it is more common than is generally supposed and that it is usually of benign character.

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An interesting and valuable monograph on this subject was published in English in 1932 by Hans Kjaergaard¹ of Copenhagen. He studied fifty-one patients, with a total of sixty-one attacks. He finds that pneumothorax simplex arises in patients apparently perfectly healthy without evidence of either tuberculosis or emphysema, and that they usually recover spontaneously. It is commoner in males than in females; Kjaergaard found thirty-six men to fifteen women. It can occur at any age from infancy to advanced life, though it is more frequent in early adult life—so much so that in France it is often referred to as “pneumothorax des conscrits.” Kjaergaard noted that the onset was usually sudden and that strain or accident is not common. My own experience confirms this, though I have had two patients in the same family in whom it occurred while straining at stool, and one in whom it followed a fall while mountaineering. In several instances I have seen it in association with asthma, although Kjaergaard would not include these in his group if there were much evidence of emphysema. One case in a schoolboy began with sudden pain while playing tennis.

Little is known of the pathology of benign or simple pneumothorax. Many writers, chiefly on theoretical grounds, have regarded it as due to rupture of an emphysematous bulla near a small area of healed subpleural tuberculosis. It is certain that the majority of these cases show no clinical or x-ray signs of tuberculosis, and do not develop such afterwards. Kjaergaard, quoting Hayashi and Fischer, believes the weak point at which rupture takes place is a distended vesicle near scar tissue, with a valvular opening to a bronchiole which allows air to enter but not to escape; but he has also found rupture in distended vesicles in emphysema.

Kjaergaard's classification into five groups—partial, “coat-shaped,” total with little or no displacement, total with moderate displacement, and valvular or tension pneumothorax—seems unnecessarily complicated. I suggest the following: (1) partial, where the collapse of the lung is limited by adhesions or by rapid closure of the opening in the pleura; (2) complete, when the lung retracts completely with or without mediastinal displacement; (3) valvular or tension pneumothorax, where the opening is valvular and the intrapleural pressure becomes greatly raised; (4) bilateral pneumothorax, which may be simultaneous or successive; (5) haemopneumothorax.

SYMPTOMS AND SIGNS

The symptoms in these cases vary greatly. In partial pneumothorax there may be only slight pain and shortness of breath with or without cough. These symptoms may increase and become severe if the pneumothorax progresses to complete collapse of the lung, and still more

so if the opening is valvular, when alarming symptoms may develop, with cyanosis, restlessness, anxiety, and eventually collapse with cardiac failure, unless relief is afforded. There is as a rule no disturbance of temperature, though there may occasionally be a slight rise for a few days. Cough is not marked, and is generally short and dry. In a few cases some blood-tinged mucus may be expectorated. In haemopneumothorax the symptoms are often severe and suggestive of internal haemorrhage or of an acute abdominal catastrophe.

The physical signs may be so slight as to escape notice, or may be typical of complete pneumothorax with marked indications of mediastinal displacement. In partial cases diminished movement with tympanitic percussion note or with obliteration of cardiac or liver dullness, and weakened air-entry, should suggest the nature of the condition. Examination by x rays is crucial, and it is important to take oblique or lateral as well as antero-posterior views, or the condition may be overlooked in partial cases. The films also show the situation of the air and the degree of lung collapse. As a rule there is no pleural reaction and no fluid except, in a few cases, a very small pool at the costophrenic angle or at the lowest part of the pleural space.

The diagnosis of this condition has to be made from:

(1) Large emphysematous bullae. In these, with x rays lung markings can usually be seen, and they are often multiple. (2) Subphrenic pneumothorax. X -ray examination will show the relation of the air to the diaphragm. (3) Congenital cysts. Diagnosis may be difficult, though the failure of the air to absorb may arouse suspicion. It is possible that some cases recorded as chronic pneumothorax may have been of this nature. (4) Hernia of the stomach through the diaphragm. This may only be differentiated with certainty by x -ray examination.

The course of these cases is usually benign, and the lung may expand fully in two or three weeks in partial degrees, and within six weeks in complete pneumothorax. In valvular or pressure cases the course may be longer. In a few instances the condition becomes chronic, and air may remain in the pleura for months or even years. These cases of chronic pneumothorax are probably due to the failure of the opening in the pleura to close, since removal of air under these conditions does not help. As a rule the lung expands even after many months. The liability to recurrence seems considerable, though the extent may vary in different attacks. Watson and Robertson² record a case with fourteen separate attacks on the two sides and on one occasion a bilateral partial collapse. Cases of bilateral pneumothorax are uncommon, and, of course, in simultaneous pneumothorax one or both are partial in patients who recover. Patients with bilateral pneumothorax may cause some anxiety, especially if one or other side has a valvular opening. They usually recover, but cases may also be recurrent.

Spontaneous haemopneumothorax is generally serious and often fatal; no lesion may be discovered; and the condition may, like pneumothorax simplex, be the result of a ruptured valve vesicle or emphysematous bulla.

TREATMENT

In partial cases no treatment is necessary save rest for a few days. In complete pneumothorax, if the opening closes, re-expansion may be rapid. It is advisable for the patient to rest in bed or in his room for two or three weeks, and the progress of absorption should, if possible, be watched by x -ray examination. If there are symptoms of severe pain or dyspnoea at or shortly after the onset, some analgesic remedies may be needed, and it may be desirable to take the pressure in the pleural cavity. If this is positive, air may be removed by a reversed artificial pneumothorax apparatus or, in case of urgency, by a

cannula or large needle with a rubber tube attached, the distal end of which is placed under water. If the dyspnoea is recurrent, the latter may be the better method, since the cannula can be left in for some hours or days to prevent the pressure becoming positive. Some authorities advise against removal of air unless symptoms of distress arise, on the ground that the tear is more likely to heal if the lung is compressed. In cases in which absorption does not take place it has been recommended to put in air to collapse the lung further in order to favour closure of the tear in the pleura. In recurrent cases attempts have been made to produce adhesion by injecting the patient's own blood or a 30 per cent. solution of grape sugar into the pleura. In haemopneumothorax haemostatic measures may be necessary, and possibly removal of some of the blood from the pleura by aspiration.

Spontaneous Pneumothorax in Pulmonary and Pleural Tuberculosis

Spontaneous pneumothorax is certainly a common event, and often a very serious one in the course of pulmonary tuberculosis. Kingston Fowler³ found sixty-five examples in 1,000 necropsies at the Brompton Hospital. Since it is not invariably fatal, it is clear that the incidence of this complication is more than the post-mortem figures suggest, and any sudden accession of pain, associated with dyspnoea and distress, should suggest this possibility and lead to clinical and x -ray investigation.

The results in this disease vary most strikingly, and we may classify the cases for purposes of discussion as follows:

1. Cases of quiescent or arrested disease leading to partial, and even complete, collapse of one lung without pleural reaction. The course is similar to that of pneumothorax simplex, from which it can only be differentiated by the most careful study, including clinical, radiological, and haematological investigation, and perhaps tuberculin tests. Patients should be treated as those with pneumothorax simplex, but should be carefully watched for a long period.

2. Cases of advanced disease. In these death occurs within a few hours or days from shock, exhaustion, or cardiac failure. It should be borne in mind that in some patients with severe symptoms soon after the onset the recognition of the valvular nature of the pneumothorax and appropriate treatment may relieve distress, prolong life, and even lead to recovery in apparently hopeless cases. The treatment is therefore to combat collapse, to relieve anoxaemia and dyspnoea, and to remove air, if there is positive pressure, by the methods already mentioned.

Between these extremes various conditions result, owing to secondary reactions of varying gravity—namely,

3. It is probable that some degree of spontaneous pneumothorax from lung rupture arises not infrequently in the course of therapeutic pneumothorax. Hutchison and Blair⁴ noted this seven times in the course of treatment of 120 cases. They describe a typical temperature chart with a sudden rise and gradual staircase fall, associated with signs of mediastinal displacement and an alteration in pleural pressures. This accident seems especially apt to happen in bilateral therapeutic pneumothorax, and may lead to grave symptoms, particularly if the perforation is valvular. In treatment the pressures should be watched, with x -ray control, and the degree of pleural reaction carefully noted, in order to determine the spacing of subsequent refills. A valvular opening with the development of pressure may require very careful treatment and frequent or continuous removal of air.

4. The perforation of the visceral pleura may heal, but the pleura may become infected with tuberculous lesions

and an effusion of clear fluid form. Such an event may be the first indication of the presence of the disease. If, after careful examination, it is clear that the opposite lung is not extensively affected, it may be advisable to continue the pneumothorax therapeutically, treating the patient in the same way as if the fluid had appeared in the course of a therapeutic pneumothorax.

5. The perforation may heal, but definite tuberculous pus may develop, resulting in a true tuberculous pyothorax or empyema. This is a graver condition, and one in which the choice of treatment is often a responsible matter of great difficulty. The object is to secure re-expansion of the lung, though the extent of disease may be such as to render this unlikely. The most hopeful method is to aspirate and wash out at intervals with some mild antiseptic such as flavine or Carrel-Dakin solution, x-ray examination being used to see if re-expansion of the lung is taking place. Sometimes a phrenic block or evulsion may prove helpful. In some cases in which there is no temperature reaction with the purulent fluid in the pleura the fluid may be left, and aspirated only if it causes distress. Occasionally the lung slowly expands. Another measure which has been suggested is the so-called "disinfecting oleothorax." Ray W. Matson⁵ recommends complete removal of the pus, followed by irrigation of the pleural cavity, after which a massive injection of 5 per cent. gomenolized paraffin, equal to the amount of pus removed, is introduced. If these measures fail surgical treatment should be considered, such as intercostal tube drainage with slight continuous suction, water-sealed drainage, or thoracoplasty in several stages. The decision in regard to thoracoplasty should not be too long postponed lest the patient's general condition deteriorate so much as to render it impracticable.

6. If the rupture occurs over or near a cavity the pleura may become infected with septic organisms in addition to the tubercle bacillus, and a septic pyopneumothorax result. A similar condition may obtain from the rupture of a tuberculous empyema or pyothorax into a bronchus or pulmonary cavity. This is the most serious of all forms of spontaneous pneumothorax, and may be rapidly fatal. On the other hand, it may be relatively benign in course, especially if localized. The treatment most likely to help is to aspirate and wash out unless the patient is too ill and obviously sinking, intercostal drainage as for pyothorax being employed later. If the effusion still remains septic, rib resection with water-sealed but not open drainage may be necessary, with a view to thoracoplasty later to close the pleural cavity and arrest the lung disease.

Spontaneous Pneumothorax in Non-tuberculous Conditions

Air may enter the pleura from rupture of an empyema causing a bronchial fistula, or by perforation or local destruction of the pleura by gangrene, abscess of the lung, septic infarct, or new growths of the lung, pleura, bronchi, or mediastinal structures.

It is necessary to recognize that the gas separating the pleural surfaces may arrive there by channels other than directly from the lungs and air passages—namely, (1) through the oesophagus in cases of ulceration, new growth, or trauma; (2) from the alimentary tract in cases of rupture of a subphrenic abscess or of a dysenteric or other liver abscess through the diaphragm; (3) from outside by perforating wounds or from rupture of a chest-wall abscess or of an empyema necessitatis; and (4) as a pathological rarity from its formation by gas-producing organisms in empyema.

In nearly all of these cases a septic pyopneumothorax results, often with grave symptoms at once. Physical signs may be characteristic, but x-ray investigation, showing a fluid level or levels in the pleura, with collapsed

lung, may first reveal the condition. In many of these cases the outlook is hopeless owing to the nature of the primary condition, and palliative measures alone are practicable. In others, the treatment is surgical on the same lines as those just mentioned for infected tuberculous pyopneumothorax, though where the primary disease is abdominal special operations may be necessary.

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SUBSTANCES PROMOTING NORMAL AND ABNORMAL GROWTH*

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Among the many phrases that pass through the mind as one meditates about the remarkable phenomena which living things present to us, none has a more characteristic note than that title which Aristotle prefixed to one of the most interesting of his writings, *περὶ γενέσεως καὶ φθορᾶς*, or in Latin more ominously, *De Generatione et Corruptione*. In English this is usually translated *Generation and Destruction*, but there is an old phrase, more consonant with the feeling of the Greek "On Coming to Be and Passing Away." In this symposium, by good fortune, we shall be concerned entirely with certain of the agencies which are involved in the coming-to-be of living organisms, and we shall leave out of account that process of dissolution which in a few short moments can destroy what took, not a few months, but many years, to build. We shall hear something of the numerous chemical substances which in recent years have become known to us as having powerful and sometimes very specific effects upon various elements in this building process.

At the outset, however, a few words must be said about the title of the symposium. If we define growth as the increase in total weight or dry weight of a cell, a tissue, or an organ, we shall be perfectly correct but not very interesting. As soon as we acquire a close acquaintance with the processes of normal development, whereby the finished animal or human organism comes to be, we realize that normal growth is often if not generally accompanied by differentiation. We may call differentiation the sum total of all the changes by which a highly heterogeneous system of organic forms originates from the relative homogeneity of the egg or zygote. These changes produce ever higher levels of internal organization. They are perhaps more interesting than the simple growth processes, because more mysterious.

It is easier for us to picture how one cell can produce another exactly like itself, or how molecule may be added to molecule within a growing cell, than how regional changes occur. How does histological differentiation set in at the right time and place? How do organic forms arise? What exactly is going on at a moment when we can say "In a few minutes a minute depression will appear on the floor of the archenteron, and this will

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