Spontaneous Pneumothorax (1944)

Pages
36

Size
8.5 x 10

ISBN
0309347203

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SPONTANEOUS PNEUMOTHORAX

I.

Introduction

Pneumothorax may be of endogenous origin, that is, air may leak from the lung through the visceral pleura into the pleural space, or it may be of exogenous origin, air may reach the pleural space through an opening in the chest wall and parietal pleura. Clinically, pneumothorax of endogenous origin is met in a great variety of curcumstances, the most important of which are: (1) traumatic, e.g., following bronchoscopic examination, intra-tracheal anesthesia, automobile accidents with broken ribs puncturing the lung, violent artificial respiration, attempts at anesthesia of the stellate ganglion, severe coughing spells associated or not with infectious disease like whooping cough, influenza, etc., needling of the lung, etc., (2) rupture of congenital sub-pleural vesicles, congenital sub-pleural cysts, emphysematous "valve vesicles" or scar tissue vesicles, (3) rupture or perforation of tuberculous lesions of the lung, (4) a large miscellaneous group including neoplasms of the lung, infarcts of the lung, abscesses, etc. Pneumothorax of exogenous origin is most frequently the result of deliberate injection of air into the pleural space for the treatment of pulmonary tuberculosis. The term <u>induced pneumothorax</u> is preferable for this type of pneumothorax and not <u>artificial pneumothorax</u> which has, however, the advantage of being time-honored and generally understood. <u>Therapeutic pneumothorax</u> is also a convenient and accurate term. In case of hydrothorax or cases

without hydrothorax induced pneumothorax may also be used for diagnostic purposes to secure better visualization of the lung and parietal thoracic wall. Pneumothorax of exogenous origin is frequently due to external violence such as bullet wounds. Here air may reach the pleural space from without as well as from within, that is, from the wounded lung. It is obvious, therefore, that the term traumatic pneumothorax is applicable both to pneumothorax occurring from external violence and pneumothorax occurring from internal damage to the lung and visceral pleura.

Although the adjective spontaneous is not accurately descriptive, the term spontaneous pneumothorax has from long usage come to mean the sudden entrance of air from the lung into the pleural space through an opening or openings, the result of disease or congenital or acquired defect of the lung or pleura and not due to external violence.

The lung may partially or completely collapse. The opening or openings through which air passes from the lung into the pleural space may be few or many, small or large, close promptly with collapse of the lung (closed pneumothorax), remain temporarily or permanently patent (open pneumothorax), or periodically permit air to enter the pleural space but not to return into the lung (valvular pneumothorax). The absence or presence of pleural effusion, the nature and the amount thereof, will depend upon the lack of infection of the pleura or its degree at the time of rupture of the lung or subsequently in the case of persistent fistulae. In spontaneous pneumothorax in the tuberculous,

infection of the pleura is the rule. In spontaneous pneumothorax of apparently healthy persons, even though the collapse of the lung may recur frequently or be persistent over several or many years, infection of the pleura does not occur because the lung is healthy; rupture has been due primarily to anatomical imperfections and not to active disease (infectious process).

II.

Classification

Spontaneous pneumothorax may be most usefully divided into:

I. Spontaneous pneumothorax in tuberculous persons.

II. Spontaneous pneumothorax in apparently healthy persons.

1. Simple pneumothorax
2. Recurrent pneumothorax
3. Persistent pneumothorax
4. Tense pneumothorax
5. Hemo-pneumothorax

III.

Spontaneous Pneumothorax and Tuberculosis

It is not planned to give here a full discussion of spontaneous pneumothorax in tuberculous persons, but merely a summary of fundamental differences between this dramatic episode in tuberculous persons and previously apparently healthy persons.

It was formerly thought that spontaneous pneumothorax was nearly always due to an active tuberculous infection of the lungs. In 1932 Fishberg expressed the general opinion when he wrote that 80 per cent

of all spontaneous pneumothoraces were due to tuberculosis. Additional experience gained largely through the wide spread use of artificial pneumothorax in the treatment of tuberculosis and recognition of the value of the x-ray in the study of chest disease, would indicate that these figures were too high at the time and are certainly too high today after the reduction in both mortality and morbidity rates. It is most important to appreciate that spontaneous pneumothorax often occurs in persons who never have had tuberculosis, as evidenced by negative tuberculin tests, good history and negative chest x-rays, do not have tuberculosis at the time of the lung collapse and subsequently never develop tuberculosis.

Between July 1937 and April 1944, 992 patients were discharged from the National Jewish Hospital in Denver. Among those patients classical spontaneous pneumothorax due to ulceration of a caseous lesion through the visceral pleura occurred only once in the course of an active progressive pulmonary tuberculosis in the absence of an accompanying artificial pneumothorax. Spontaneous pneumothorax occurred only six times as the result of the rupture of an emphysematous vesicle in a lung in which the tuberculosis was well controlled (Kaufman). According to Powell, about 6 per cent of the fatal cases of tuberculosis at the Brompton Hospital at London died with pneumothorax. At the Montefiore Hospital, New York, spontaneous pneumothorax constitutes about 3 per cent of the immediate causes of death among tuberculous patients. Fishberg says: "In many hospitals for the tuberculous we meet with cases of sudden death during the night.

Some of these are due to copious internal pulmonary hemorrhage, but in most the cause is pneumothorax, which killed before aid could be summoned by the patient." Watson and Robertson (1928) estimate that spontaneous pneumothorax occurs at some time in 2 to 4 per cent of all cases of pulmonary tuberculosis.

In the tuberculous, spontaneous pneumothorax may develop as the result of (1) ulceration into the pleural space of a progressive lesion, (2) tear of the pleura by the pull of an adhesion between lung and chest wall, (3) rupture of a sub-pleural vesicle which may or may not have developed as the result of healing of a localized tuberculous lesion. In the latter case, the tuberculous lesion may be completely obsolete and the lungs otherwise entirely free of active or inactive tuberculosis. It must not be forgotten that spontaneous pneumothorax may develop in a tuberculous person by a mechanism of non-tuberculous origin, rupture of an emphysematous bleb or tear of adhesions.

Finally, spontaneous pneumothorax occurs in the tuberculous infrequently on the same side as an induced (artificial) pneumothorax as the result of (1) wound of the lung at initial treatment or during refills, (2) tear of adhesions, (3) ulceration of a caseous lesion, (4) rupture of an air vesicle.

Occasionally, a valve-like tuberculous endo-bronchial lesion may lead to an obstructive atelectasis of the whole lung, that is, air passes out of the lung easily but slowly and with difficulty or not at all into the lung. Brantigan, Hoffman and Proctor (1942) point out that in induced pneumothorax after a closed pneumonolysis, endo-

bronchial lesions may produce a check valve type of obstruction that results in rapid re-expansion of the lung and loss of the pneumothorax space. Under similar conditions, endo-bronchial lesions may cause complete occlusion and atelectasis. Bronchoscopic examination is therefore indicated in (1) all cases of recurrent pneumothorax as well as all cases of persistent pneumothorax, (2) cases of artificial pneumothorax when the lung re-expands with great rapidity and collapse is maintained with great difficulty, (3) cases of artificial pneumothorax when the lung after cessation of refills shows no indication to re-expand, (4) cases of spontaneous pneumothorax developing during the course of artificial pneumothorax and in which the collapsed lung gives the appearance of being atelectatic, (5) cases in which following rapid re-expansion of the lung after an induced pneumothorax, it seems necessary to collapse the lung again and yet repeated attempts to do so have been unavailing.

Symptoms and Physical Signs of Spontaneous Pneumothorax in Tuberculous Persons

Although the features of the episode itself are highly characteristic and diagnosis easy, since spontaneous pneumothorax may occur at any moment in the course of pulmonary tuberculosis, when disease is much or little, rapidly progressive, quiescent, arrested, or long since healed, when death seems inevitable shortly, or recovery just around the corner, symptoms and physical signs will vary with the setting.

The onset is sudden with pain of more or less severity in the side of the chest, back or shoulder, with great anxiety, shortness of breath, tachycardia, signs of shock, pallor, cyanosis, clammy skin,

falling temperature and blood pressure. If death does not come quickly from shock and embarrassment of respiration and circulation, the patient rallies and within twenty-four hours begins a septic type of fever. The pleura is heavily inoculated with tubercle bacilli and often with organisms of mixed infection. A tuberculous pyo-pneumothorax or mixed infection pyo-pneumothorax is the rule. Physical signs vary with extent of collapse of the lung, presence or absence of interstitial emphysema, height of intra-pleural pressures and displacement of mediastinal structures. Physical signs in spontaneous pneumothorax in tuberculous persons are fundamentally the same as in spontaneous pneumothorax of non-tuberculous persons. (Fig. I)

In a less unfortunate few, the infection of the pleura is not overwhelming, the perforation is small and soon heals. Constitutional symptoms severe at first gradually subside, a large effusion fills up the side, the air in the pneumothorax space is ultimately absorbed, a pleural effusion is left, which requires aspiration only if pressure symptoms develop. With the passage of time, the fluid is absorbed and the lung re-expands wholly or in part. Some mediastinal displacement and retraction and limitation of movement of the chest wall on the affected side may remain. If the involvement of the lung was not too extensive, its long continued collapse may lead to its healing and recovery will be complete.

Twenty years ago, in the vast majority of instances spontaneous pneumothorax in tuberculosis was fatal within a few months if not

days or weeks. Today the skilled thoracic surgeon saves many of these patients. Much will depend upon the treatment given or not given by the first doctor to see the patient after the perforation has taken place. Treatment is directed toward relief of pain, shock, anoxia and a struggling heart. Aspiration of air from the pleural space either periodically with the pneumothorax apparatus or continuously with water seal and cannula between the ribs is life-saving, for the moment, at least. The general practitioner and the general surgeon must realize, often do not, that tuberculous pyo-pneumothorax is a different disease from non-tuberculous pyo-pneumothorax. Recovery is greatly influenced by the general condition of the patient, the extent of tuberculosis in the other lung as well as the collapsed lung, the degree of soiling of the pleural space, the size of the perforation, the development of a permanent broncho-pulmonary-pleural fistula, the presence and degree of interstitial emphysema. If the fistula is large the rapidly accumulating pleural effusion loaded with tubercle bacilli and other organisms is drained through the lung, threatening bronchogenic spread, exhausting the patient with the unending cough and profuse expectoration. In this much-dreaded complication of pulmonary tuberculosis, nice judgment is needed to know what to do and when to do it. In selected cases, a small catheter between the ribs will provide external drainage, stop bronchial drainage and tide the patient over the crisis. Thoracoplasty is usually inescapable, life-saving, not always successful. Differential diagnosis is summarized in Fig. II.

IV.

Spontaneous Pneumothorax in Apparently Healthy Persons

A. Simple (benign, idiopathic or temporary) spontaneous pneumothorax may be defined as pneumothorax occurring suddenly in previously healthy persons. The incident is accompanied by little or no fever, pleural effusion is infrequent and when present very small in amount. Physical signs are summarized in Fig. I. The lung re-expands in two to six weeks, recovery is complete and without residual ill effects. The other lung as well as the re-expanded lung show no evidence of active pulmonary tuberculosis. The sputum is negative for tubercle bacilli, the tuberculin test is frequently negative. Finally, during subsequent years, active pulmonary tuberculosis develops in these patients no more frequently than in any other similar group. The pneumothorax is produced by leakage of air into the pleural space through one or more small openings between lung and pleura due to rupture of one or more emphysematous blebs (bullae, air vesicles, etc.) or to tear of the lung by unusual strain or adhesions between lung and chest wall. Some of these bullae are doubtless due to congenital defects of lung or pleura. (Ross and Fullerton, 1939.)

General Instructions for the care of patient with spontaneous pneumothorax of non-tuberculous etiology. The patient should be hospitalized promptly. If fever is present or dyspnea marked, absolute bed rest is imperative. Later, the patient may be up and around the ward quietly until the lung re-expands. Straining at stool,

lifting heavy objects, awkward positions, severe coughing paroxysms, ascent above 4000 feet in a plane should be avoided. Re-expansion of the lung may be checked by fluoroscopic examination at appropriate intervals. The possibility of pulmonary tuberculosis must not be overlooked. The sputum should be searched by all approved methods for tubercle bacilli, x-ray films of the re-expanded lung as well as the other lung should be scrutinized with great care for minimal lesions of tuberculosis. A tuberculin test should be done.

Treatment of simple pneumothorax. In these cases the lung usually re-expands promptly, often with astonishing rapidity in a few days or at the most in a few weeks. Occasionally, air may be aspirated from the pneumothorax space at short intervals (daily or every few days) and re-expansion of the lung thereby hastened. For fear of enlarging the opening into the pleural space or encouraging its persistence, aspiration of air must be undertaken cautiously, small amounts of air (200-500 cc.) only withdrawn at one sitting and high negative pressures avoided. In any form of pneumothorax, leaks (fistulae) may be discovered by the simple procedure of withdrawing small amounts of air with the aid of the pneumothorax apparatus in reverse and carefully noting whether the expected increasingly negative intra-pleural pressures are maintained (no leak) or slide back toward neutral. The test may re-open a healing fistula.

The treatment of cases in which the lung again collapses spontaneously after partially re-expanding, which may happen more than once, is discussed under Recurrent Pneumothorax.

B. Spontaneous pneumothorax which recurs after complete re-expansion of the lung is called Recurrent Pneumothorax. Although, in the vast majority of instances the lung re-expands spontaneously and collapse does not recur, in about 10 to 20 per cent of cases, collapse of the lung does recur one or more times after short or very long intervals of time, usually on the same side, occasionally on the other side. Rarely, collapse of both lungs occurs simultaneously, infrequently collapse of the lung is persistent. (Bittorf, one patient with lung still collapsed after 25 years; Perry, one patient with lung still collapsed after 20 years.) In 1938 Trudeau said he had a patient who had had spontaneous pneumothorax 12 times on the left side and 16 times on the right.

Since recovery is unilateral cases is certain, opportunities at the post-mortem table to study with exactitude the method or methods of production of this form of pneumothorax have only rarely been afforded. Up to 1932 Kjaergaard could find reports of only 6 autopsies. However, x-ray study (fluoroscopy and roentgenography), thoracoscopic observations and thoracotomy have yielded individually or collectively in a small number of cases valuable information in the form of ruptured blebs, bullae or vesicles and pleural adhesions presumably tearing the lung. One way or another the exact site of perforation has been demonstrated in life many times.

Just as repeated or continuous traction on the edges of any wound will delay or prevent its healing, so in persistent or recurrent pneumothorax, closure of a pulmonary-pleural fistula, especially one located near adhesions, will be delayed or prevented by respiratory

diaphragmatic movements transmitting traction through the collapsed lung by means of adhesions to the diaphragm on the one hand and adhesions to the chest wall on the other hand. After severing pleural adhesions a low positive pressure induced pneumothorax will permit the lung to contract or relax and close the fistula with consequent healing. If pleural adhesions cannot be severed, temporary phrenic nerve paralysis and resultant rise of the diaphragm may relax the lung sufficiently to permit healing of the fistula. (Fig. 3.)

In the cases in which x-ray or thoracoscopy demonstrates a single emphysematous bleb, probably responsible for collapse of the lung, or pleural adhesions, surgical intervention (thoracotomy) to excise the bulla or sever the adhesions (at thoracoscopy or thoracotomy) would appear justified in spontaneous pneumothorax which repeatedly recurs. Although the number of cases so treated, reported and unreported, is small and lapse of time short since these methods of treatment have been undertaken, and finally, even though the probability of recurrence more than twice is not great, surgical approach to this problem seems logical and justifiable in the type of case described in this paragraph.

Chemical pleuritis. In cases of recurrent pneumothorax in which presumably responsible vesicles (blebs or bullae) or pleural adhesions have not been demonstrated by x-ray or thoracoscopy, thoracotomy may still be tried in the hope of finding responsible and correctable lesions. When thoracotomy has been unsuccessful or much more frequently in place of any surgical procedure, the production of an aseptic or

"chemical pleuritis" has been tried in the hope of producing adequate and persistent adhesions between lung and pleura sufficient to prevent repeated collapse of the lung. The list of chemical irritants instilled into the pleura for this purpose includes hypertonic glucose solutions (30-67.5 per cent), hypertonic saline, 0.5-1.0 per cent silver nitrate, glycerine, formaldehyde, oil of turpentine, gomenol in olive oil, lipiodol, mineral oil, India ink, etc. Some of these preparations have been tried only experimentally. Particulate matter such as plain or iodized talc, powered or in suspension, has been used for the same purpose. All chemical installations are usually painful, sometimes extremely so, often unavailing, and are frequently accompanied by more or less severe though transient constitutional symptoms and pleural effusions. Their ultimate effects are unpredictable.

Although poudrage has been used successfully more than once, it has two great disadvantages: it places irremovable particulate matter within the pleural space and it has occasionally caused a severe pleuritis with such thickening of the visceral pleura that re-expansion of the lung was impossible. Ross and Fullerton at post-mortem found the pleura after poudrage the site of a formidable foreign body reaction about one centimeter in thickness. The patient's own blood (200-300 cc.) may be injected into the pleural space, as recommended by W. B. Porter, and successfully tried by Watson and Robertson (1928) and others. Unfortunately, this method is not without danger. Organization of heavy deposits of fibrin on the visceral pleura may pre-

vent re-expansion of the lung. (See discussion of Hemo-pneumothorax.) The same objection, risk of visceral pleuritis and encasement of the lung in an undistensible envelope, applies to the use of fifty per cent glucose installations. The risks of this constrictive visceral pleuritis may doubtless be minimized by resorting to chemical pleuritis only in rare instances, making the injections when the lung is on the way out, that is, not far from the parietal wall of the chest in its re-expansion, and aspirating remaining air rapidly and immediately following the chemical injection. In other words, in producing a chemical pleuritis, it is vitally necessary to get the visceral and parietal layers of pleura in contact at the earliest possible moment. Gomenol is recommended by Matson, but has the disadvantage of being non-absorbable. Scarification of the parietal pleura or rubbing the pleural surface with gauze have received clinical trial but have not proved satisfactory.

Occasionally, the lung re-expands partially and then collapses again. This may happen more than once. For fear of aggravating the leak, aspiration of air should be scrupulously avoided in these cases, and resort had to induction and maintenance for several weeks of a low grade positive pressure pneumothorax by injecting air instead of withdrawing it, to assist healing of the leak. In a few cases, temporary phrenic paralysis to relax the lung will close the leak. Obstinate cases will have to be treated by thoracotomy as outlined previously.

To Summarize: Conservatism in the treatment of recurrent <u>spontaneous pneumothorax</u> is recommended. Many cases are not greatly

inconvenienced by the episodes of collapse of the lung, and, therefore, no interference of any sort whatsoever is justified. The cases most amenable to surgery are those with a single point of attack: adhesions, a single bulla or very few emphysematous bullae.

C. Persistent or chronic pneumothorax are applicable terms when collapse of the lung persists months without the lung showing any or but slight inclination to re-expand. The responsible lesion is most frequently a persistent fistula or persistent fistulae between the lung and the pleural space. Fundamentally, these fistulae develop in the same manner as the leaks that produce temporary pneumothorax. They are simply larger and possibly for that very reason remain patent.

Treatment is directed towards the healing of these openings, after which the gases in the pneumothorax space are steadily and rapidly absorbed with coincident re-expansion of the lung.

Endo-bronchial lesions causing obstructive atelectasis of the collapsed lung and therefore preventing its re-expansion are usually tuberculous in nature and, therefore, not responsible factors in the type of persistent pneumothorax under discussion in this paragraph. Nevertheless, bronchoscopic examinations should be made in any case.

Various chemical solutions have been injected intrapleurally in persistent pneumothorax as in recurrent pneumothorax, to cause or to hasten healing of the fistulae by the production of an aseptic pleuritis. In view of the uncertainties, frequest unfortunate reactions to these chemical installations and occasional occurrence

of a disastrous constrictive visceral pleuritis, it is preferable when experienced surgical assistance is available to resort without too much delay to surgery. (See section of Recurrent Pneumothorax.) Although chemical pleuritis may be used by an experienced thoracic surgeon in carefully selected cases of Recurrent Pneumothorax, it is definitely contra-indicated in Persistent Pneumothorax. At open thoracotomy, opportunity is afforded for excision of valve vesicles, severing of presumably culpable pleural adhesions, the detection, excision or repair of accurately identified fistulae.

D. Tense Pneumothorax is that form of pneumothorax in which a valve-like fistula permits the development of high intra-pleural pressures. The movement of the affected side is greatly restricted, the intercostal spaces on the affected side filled out, the percussion note is hyper-resonant, liver dullness or precordial dullness disappears, the mediastinum displaced toward the opposite side, the diaphragm on the affected side pushed downward, the breath sounds absent or diminished. The patient is cyanotic, dyspneic, in great respiratory and circulatory distress. Constitutional symptoms are often present, such as high fever and tachycardia.

Pulmonary interstitial emphysema progressing to mediastinal emphysema, to retro-peritoneal emphysema and generalized subcutaneous emphysema may or may not be associated with spontaneous pneumothorax. They are more frequently associated with that form of spontaneous pneumothorax which occurs during or in association with artificial pneumothorax. Mediastinal emphysema should always be suspected

when subcutaneous emphysema is detected in the episternal notch or above one or both clavicles. For complete elucidation of pulmonary interstitial emphysema (P.I.E.) and its relation to pneumothorax, the contributions of the Macklins and L. Hamman should be studied. Briefly, damage to the pulmonary parenchyma permits a leakage of air into the pulmonary interstitial spaces. This air in peribronchial, perivascular and septal spaces works toward the hilus of the lung and into the mediastinum. The x-ray will easily reveal air under the skin and in fascial planes in the neck, thoracic wall, etc. Less easily recognized will be the evidence of air as air streaks along the blood vessels in the lung, air in the interstitial space between the pleural folds and the pericardium along the left border of the heart and in the lateral view in the anterior mediastinum. Largely as the result of coughing or straining, gas pressures build up in the mediastinum and impede the return flow of blood to the heart through low pressure venous channels. The final effects of interference with blood flow in capillaries and small vessels in the lung and obstruction of return flow to the heart in the mediastinum are great and increasing embarrassment of respiration and circulation. The patient will die unless the emergency is recognized and effective treatment promptly instituted.

Ascent to high altitude would doubtless be quickly and completely disastrous in this type of pneumothorax. On the contrary, prompt placement in a high pressure chamber might give immediate relief. (Boyle's Law - inverse relationship pressure and volume of a gas.)

Repeated aspiration of sufficient quantities of air to restore the mediastinum to normal position and to relieve pressure on mediastinal structures is often sufficient. In more severe cases, continuous relief of pressure may be secured by needle or cannula between the ribs attached to a water seal. Air may be aspirated hazardously by needle or syringe from the mediastinum in the episternal notch or the first and second parasternal interspaces.

More simply and much more safely, relief may be secured by incisions above one or both clavicles for spontaneous egress of air or for aspiration of air by a small cup and suction. After the emergency has passed, the lung usually re-expands. If it does not, the cause of failure to re-expand and therefore the treatment are as outlined under Persistent Pneumothorax.

In any type of pneumothorax, cyanosis or any other sign of anoxia, calls for the prompt use of oxygen. In Tense Pneumothorax, the inhalation of 100 per cent oxygen for 24 to 72 hours will not only relieve anoxia but will hasten the absorption of gases in the interstitial spaces and the reduction of the volume of gas in the pneumothorax space.

E. Simultaneous Bilateral Spontaneous Pneumothorax. Collapse of one lung by sudden change of intra-thoracic pressures may lead to collapse of the other lung. In only 25 per cent of cases of spontaneous collapse of one lung does sudden severe effort immediately precede the event. Conceivably, the additional respiratory burden suddenly thrown upon the other lung may lead to rupture of emphy-

sematous bullae or tear of pleural adhesions. Obviously, with collapse of both lungs, ventilation and oxygenation are reduced to the asphyxial point. Continuous aspiration of air from both sides of the chest in the hope of partially re-expanding one or both lungs with administration of 100 per cent oxygen may be life-saving. Death comes swiftly in these cases, often before medical assistance can be secured. Simultaneous bilateral spontaneous pneumothorax carries a high mortality rate (Perry: 50 per cent death rate in 20 collected cases).

V.

Spontaneous Hemo-pneumothorax

In Spontaneous Hemo-pneumothorax after perforation of the lung both air and blood, often in considerable volume, leak into the pleural space. The fundamental cause of this form of pneumothorax, a tear of pleural adhesions or rupture of a bulla, is the same as in all other forms of non-tuberculous spontaneous pneumothorax. In the one instance, blood vessels are torn and, in the other, they are not. Ruptured bullae have been found repeatedly in persons dead of hemo-pneumothorax. Bullae have been demonstrated roentenologically during life in other instances. Finally, the successive occurrence of spontaneous pneumothorax and hemo-pneumothorax in the same patient strongly indicate a common etiologic factor. It is further significant that the walls of bullae, usually quite avascular, are sometimes surprisingly vascular. (Mazzei and Pardal, 1934.) It has been emphasized by Hartzell in his fine

review of this subject in 1942, that even at post mortem it has not always been possible to identify the bleeding point. During life it has been impossible to determine whether the bleeding came from the lung and presumably the lesser circulation or from the parietal wall of the chest and therefore the systemic circulation. It is to be noted that bleeding from the lung into the pleural space might come either from a bronchial artery and systemic circulation or the lesser circulation; in either case collapse of the lung might automatically stop the bleeding. In the case of bleeding from torn adhesions, the hemostatic influence of lung collapse would somewhat depend upon whether the lung was entirely separated from the chest wall or only partly separated from the chest wall or only partly separated with the tear located at the base of adhesions still attached to lung and parietal wall of the chest. R. C. Matson, 1939, thinks bleeding from adhesions comes largely from collaterals with the intercostal vessels. The fact that the chest cavity is not often suddenly filled with profuse hemorrhage but that blood seems to accumulate slowly over hours or several days suggests on the one hand that bleeding comes from the low pressure lesser circulation, and on the other hand that respiratory movements of the lung and diaphragm by traction on the torn lung or adhesions prevent healing of the bleeding point and favor continued oozing of blood into the pleural space. After partial re-expansion of the lung and absorption of part or all the blood in the pleural space, spontaneous collapse may recur with fresh bleeding.

In all of Hartzell's 43 cases (40 collected, 3 added), tuberculosis was adequately excluded as a cause of the spontaneous collapse

and the associated bleeding. Once again, it should be emphasized that perforation of a tuberculous lung leads to pyo-pneumothorax almost invariably and excessively rarely to hemo-pneumothorax. Hemo-pneumothorax in tuberculosis occurs usually when the disease is obvious and far advanced, and only very rarely then. However, a few instances are recorded of severe bleeding into the pleural space in tuberculous persons following artificial pneumothorax. In 1919 Heise and Krause reported a case of fatal hemo-pneumothorax following artificial pneumothorax in a man with rapidly progressing caseous pulmonary tuberculosis. Hartzell observed two patients with intra-pleural bleeding after pneumothorax refills at the Cleveland City Hospital. One patient made a good recovery, the other some weeks later developed spontaneous pneumothorax on the same side with mixed infection empyema. Weiner and Jackson report spontaneous bleeding and collapse of a lung under treatment by artificial pneumothorax in a tuberculous patient who had had thoracoplasty on the other side.

Of the 43 patients with hemo-pneumothorax, whose cases are reviewed by Hartzell, 14 died, 5 of these within twenty-four hours of hospital admission and 4 more within the week. With only one exception, all of the 43 were men. In 41 accurately recorded cases, the ages of 36 patients were between 20 and 40 years. In all 43 cases and in the 14 fatal cases, one side of the chest was not involved significantly more often than the other. Hemo-pneumothorax is therefore a much more serious affair than simple pneumothorax. In the main, the <u>clinical picture</u> differs in the following very important particulars:

(1) the pain at onset is much more severe and (2) is occasionally referred to the epigastrium or right hypochondrium suggesting an "acute belly", especially when accompanied by (3) nausea and vomiting and (4) signs of shock, pallor, sweating and tachycardia. If the patient rallies, fever is higher than with simple pneumothorax. The essential diagnostic features are: (1) the sudden onset in a previously healthy person, or in a tuberculous person taking artificial pneumothorax, of (2) marked anemia (internal hemorrhage) and (3) signs of hydro-pneumothorax with the effusion more rapidly accumulating than would be expected of an inflammatory exudate. The discovery of blood at thoracentesis clinches the diagnosis. A red cell count may be done on the removed fluid and compared with a count on the blood made at the same time.

In the type of hemo-pneumothorax here under discussion, infection of the pleura does not occur, usually the temperature returns to normal, the blood is absorbed as well as the air in the pleural space and the lung re-expands, recovery is complete. Occasionally, the blood clots in the pleural space, masses of fibrin form on the visceral pleura and on the parietal pleura, the lung cannot re-expand, fever continues, the patient remains very ill or dies. Only a formidable operation, thoracoplasty, can save the patient. Sometimes the blood is more or less completely absorbed, and the lung completely re-expands but with the passage of time heavy deposits of calcium accumulate in a thickened pleura in large plaques very conspicuous in the x-ray film. A calcified pleural plaque is most frequently due to a preceding hemothorax or hemo-pneumothorax.

Treatment of Hemo-pneumothorax. Since death is due mainly to internal hemorrhage and embarrassment of respiration and circulation, treatment is directed towards relief of high intra-pleural pressures, prevention of further bleeding and restoration of depleted blood volume. Since high intra-pleural pressures must have some hemostatic effect and since the low blood pressure from hemorrhage must discourage further bleeding, nice judgment is required to decide when to leave the patient alone and when to aspirate air, blood, or air and blood from the pleural space and transfuse. In general, repeated small transfusions when the red cell count and hemoglobin values are near 50 per cent are safer than not transfusing. The longer the interval since the onset of the collapse and bleeding the safer the transfusion.

If other blood or plasma is not available, blood in the pleural space may be withdrawn, air replacing it coincident with withdrawal and this blood used for transfusion. The patient must be under constant observation and repeated blood counts and blood pressure determinations made. The patient should be in an oxygen tent until the emergency has passed. Even more than in other forms of pneumothorax, transport by plane to high altitudes is dangerous.

Summary

1. Hemo-pneumothorax is due to ruptured bullae or torn adhesions.
2. Reported cases indicate a much greater frequency among males than females.
3. The essential diagnostic criteria are:
    1. sudden onset in a previously healthy person
    2. signs of hydro-pneumothorax

3. rapidly accumulating effusion
4. signs of internal hemorrhage: shock, pallor and anemia

4. The acute onset with shock and pain in the epigastrium resemble closely an acute abdomen.
5. When a patient has lost much blood, it is probably safer to use repeated small transfusions than to do nothing.

VI.

Spontaneous Pneumothorax and Aviation

It is not intended here to discuss the surgical aspects of evacuation of cases of traumatic pneumothorax, the result of bullet wounds, etc., from combat zones, but merely to review certain physiological principles and physical laws fundamental to the intelligent handling in peace or in war-time of pneumothorax whether spontaneous, artificial or traumatic.

According to Boyle's Law, temperature remaining unchanged, the product of pressure and volume of an ideal gas is constant. In other words, if the pressure increases, the volume of the gas decreases; if the pressure decreases, the volume of the gas increases. This relationship for increasing pressures is shown in Table I. A similar table may be constructed for decreasing pressures. (Table II.)

Table I.

Pressure (P)	1	2	3	4	6
Volume (V)	12	6	4	3	2
$P \times V$	12	12	12	12	12

Table II.

Pressure (P)	1	.5	.33	.25
Volume (V)	12	24	36	48
P x V	12	12	12	12

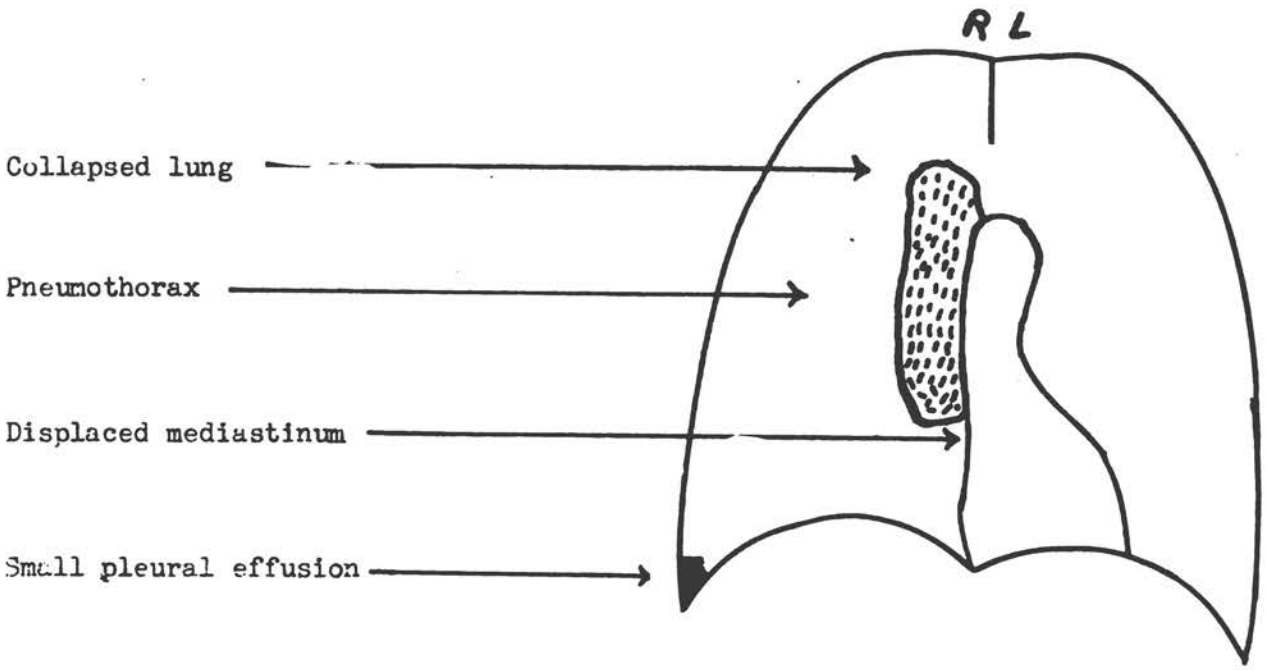
Obviously, the volume of gas will increase with altitude, that is, with falling barometric pressure. At 18,000 feet the barometric pressure, 380 mm. Hg., is one half the sea level pressure and one volume of gas will be doubled; at about 34,000 feet altitude the volume of the gas is quadrupled and so forth. Gases in the body, e.g., in a pneumothorax or in the intestinal tract, are saturated with water vapor at a pressure of 47 mm. of Hg., accordingly the change with altitude in volume of gas in a pneumothorax may be calculated by a formula in which the old pressure less the water vapor pressure is divided by the new barometric pressure less the water vapor pressure. It should be noted that the water vapor pressure is unchanged by variations in atmospheric pressure. A man at sea-level with complete collapse of one lung has a pneumothorax volume of about 3000 to 5000 cc. If we estimate the average barometric pressure at Denver, altitude 5,280 feet, as 600 mm. Hg., the change in volume of this gas in the chest may be calculated as follows:

$$\frac{760 - 47}{620 - 47} = 1.24$$

Every 1000 cc. of gas at sea level will try to occupy a volume of 1240 cc. at Denver. Three thousand cc. of gas, if the intra-thoracic

environment permitted, would assume the volume of 3720 cc. If the pneumothorax was of recent development and the patient already in some distress from displacement of the mediastinum, the expanding gas in the chest might add greatly to his distress if not actually jeopardize his life. Doctors who live at sea level are learning not to give a "refill" to a pneumothorax just before the patient takes the train for higher altitudes. If a patient must be evacuated by air, medical and nursing personnel must be instructed in the simple procedures, previously outlined, to relieve increasing intra-pleural pressures. A needle between the ribs, long enough to reach the pleural space, not long enough to wound the lung, is sufficient for the emergency.

Fig. I. Physical Signs Pneumothorax



On the affected side

1. Restricted movement
2. Prominence whole side
3. Filling out of intercostal spaces
4. Hyper-resonance
5. Disappearance hepatic dullness
6. Mediastinal displacement
7. Absence breath sounds
8. Amphoric qualfity breath sounds
9. Metallic sounds
10. Coin sound
11. Succussion or splash (hydro-pneumothorax)

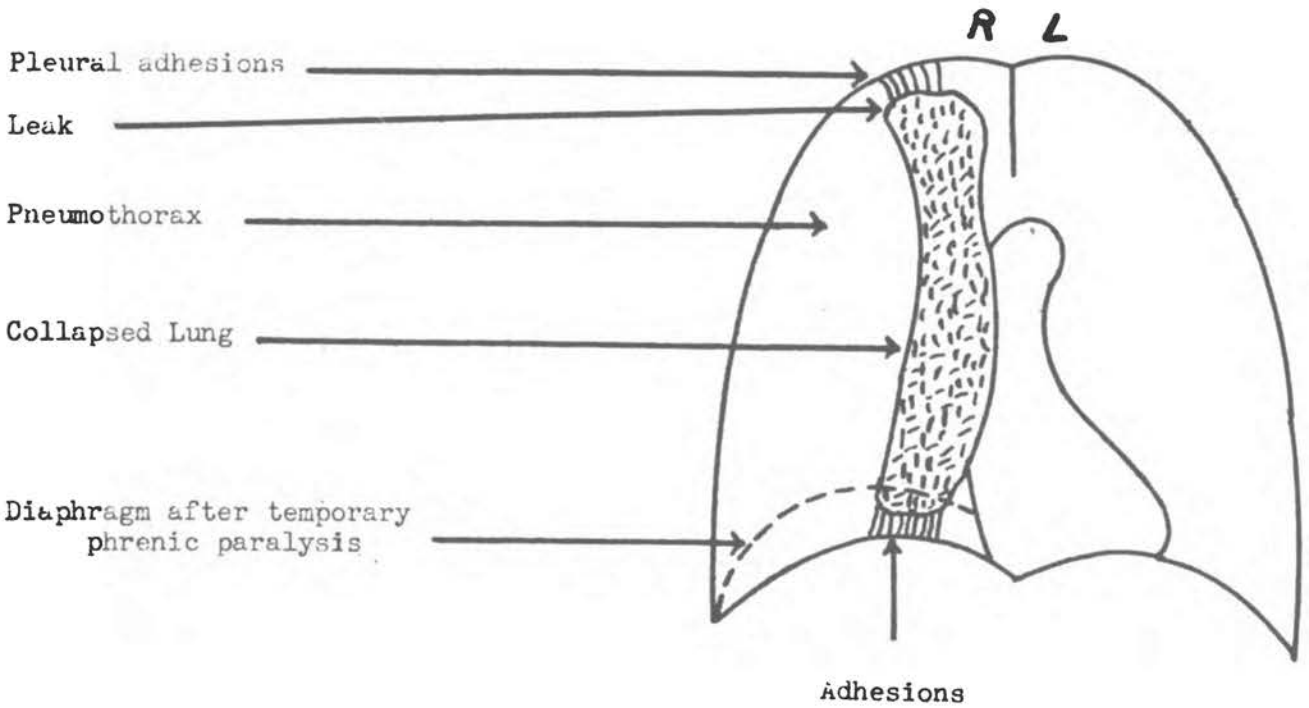
Fig. II. SPONTANEOUS PNEUMOTHORAX

Differential Diagnosis

		Tuberculous	Non-Tuberculous
1.	Previous condition of lungs	Tuberculous	No tuberculosis, emphysematous blebs or pleural adhesions
2.	Condition of lungs at time of collapse	Tuberculous	No tuberculosis
3.	Subsequent condition of lungs	Progressive disease, usually	Tuberculosis does not develop
4.	At time of collapse	Severe constitutional symptoms, fever, etc.	Constitutional symptoms absent or slight
5.	Pleural effusion	Pyo-pneumothorax the rule	Little or no pleural effusion
6.	Fistula	Rarely heals	Usually heals
7.	Re-expansion of the collapsed lung	Rare	Usual
8.	Recurrence of collapse	Very rare	10 - 20 per cent
9.	Surgery	Usually necessary	Elective if recurrent or persistent pneumothorax
10.	Prognosis	Very bad	Excellent

Fig. 3.

Diagram to illustrate influence of the movements of the diaphragm on the maintenance of a leak and the effect of temporary phrenic paralysis in relaxing the lung and closing the leak.



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