Current Practice

RESPIRATORY TRACT DISEASE

Spontaneous Pneumothorax: Diagnosis and Management

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General Considerations

Spontaneous pneumothorax may occur at any age, but it is most common between 20 and 40 years, and is much more frequent in men than in women. It is very unusual before the age of 20 years. There is a second group of cases in the United Kingdom in older patients, associated with chronic bronchitis and emphysema. This association is partly responsible for the excess of men over women with the condition.

When considering management it is important to bear in mind that spontaneous pneumothorax may be a recurrent disease and that it often occurs as a bilateral condition. The frequency of recurrence is reported as varying from 5% to 50% in different series, and the average is probably about 30%. Bilateral spontaneous pneumothorax is reported as occurring in about 10% of patients, though simultaneous bilateral pneumothorax is fortunately extremely rare.

For many years rupture of a tuberculous focus was considered to be the most frequent cause of a spontaneous pneumothorax, but this is no longer true, for the most common cause nowadays is the rupture of a small sub-pleural bleb or bulla, producing a breach of the lung surface which is often single but may be multiple. Usually there is no generalized bronchial or pulmonary disease, though spontaneous pneumothorax may be associated with almost any pulmonary disorder, and extremely rarely it may arise from rupture of the oesophagus.

The breach in the lung surface may seal off as the lung collapses, when healing will take place rapidly—the so-called *closed* pneumothorax—and re-expansion of the lung will occur spontaneously. Where there is a large rent with an associated large bronchial communication the air-pressure in the pleural space remains near atmospheric on inspiration and expiration—the *open* pneumothorax. In these circumstances spontaneous healing is unlikely. Sometimes a small breach in the lung surface acts as a one-way valve, which allows the accumulation of a large amount of air in the pleural space, and the intrapleural pressure may rise and produce considerable collapse of the lung—the *tension* pneumothorax. This may produce gross mediastinal displacement with compression of the opposite lung and the great vessels, leading to circulatory collapse and asphyxia. Tension pneumothorax is a medical emergency demanding urgent treatment.

Diagnosis

The diagnosis of spontaneous pneumothorax may often be made from the history of the illness and the physical signs elicited, but confirmation is usually sought by radiographic examination. The diagnosis is sometimes made in the course of routine radiography—when the pneumothorax is usually small. Fortunately, when the diagnosis has to be made urgently so that appropriate treatment may be instituted immediately, the clinical features are usually sufficiently gross to be readily recognized.

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Symptoms

Though gradual onset of dyspnoea is sometimes observed, the onset of symptoms is usually sudden and is rarely associated with strenuous movements or exercise of any kind. Symptoms vary greatly in severity. If a small amount of air enters the pleural space of a healthy adult with good lung function there may be no symptoms and the diagnosis may be made only on routine radiographic examination. The leak of a similar amount of air in a patient with severe chronic bronchitis, emphysema, or asthma may produce marked respiratory distress. The patient may complain of the sudden onset of severe pain in the chest on the affected side: shoulder pain is not uncommon and rarely there may be central chest pain. This is usually followed rapidly by a sensation of shortness of breath varying in degree with the size of the pneumothorax and the prior state of the lungs and bronchi. A healthy young adult may complain only of shortness of breath on exertion. There may be a slight dry cough. The patient often becomes anxious. He may complain of—or his relatives may observe—a clicking sound over the front of the thorax.

Signs

Patients with severe respiratory distress have an anxious appearance, may be cyanosed, have tachycardia and hypotension, and the respiratory rate is increased. On examination of the chest, rib movement is found to be diminished or absent on the affected side, which may be held in a position of inspiration. There may be evidence of mediastinal displacement, the trachea and apex beat being displaced away from the side of the pneumothorax. Surgical emphysema may be present in the neck, where it will give rise to a characteristic crackling feeling on palpation. Percussion will reveal a hyper-resonant note, and in severe cases the note may be tympanitic. The breath sounds are diminished to a varying extent and may be absent when a large pneumothorax is present: faint amphoric breath sounds may be heard. When the pneumothorax is on the right side, the hyper-resonance may extend well down over the liver dullness. The vocal resonance is usually diminished, and if the air is under tension the "bell" or "coin" sound may be heard. High-pitched tinkling accompaniments may be heard when a small hydrothorax is present.

It must be emphasized that these signs are present only in classical examples of pneumothorax and that small amounts of air in the pleural space may be impossible to diagnose clinically. Rarely evidence of a "noisy" pneumothorax may be observed. The patient or a relative may notice the noise, which may be heard by the physician even without the aid of a stethoscope.

The sound is variously described as clicking, crunching, grating, or crackling; it is synchronous with the heart beat, and varies with respiration, often being louder in expiration; and it may be present only in certain postures, especially on leaning to the left side. The pneumothorax in the presence of such a sound is usually of small degree and is almost invariably left-sided. It may be difficult to detect radiographically.

There may also be evidence of the disease on which the pneumothorax is superimposed—fever, cough, sputum, and haemoptysis in tuberculosis or lung abscess; wheeze and inspiratory and expiratory rhonchi in bronchial asthma; or clubbed fingers and lymphadenopathy in bronchogenic carcinoma. Spontaneous pneumothorax due to a ruptured tuberculous cavity, and less commonly to a ruptured lung abscess, may lead to the rapid accumulation of fluid in the pleural space, and signs of this fluid may be present in addition to those produced by the pneumothorax. Splashing may be heard on succussion of the chest, though this sign may also be produced after a marked haemorrhage into the pleural space.

The radiographic diagnosis of spontaneous pneumothorax is usually reasonably straightforward. The outline of the collapsed lung is almost always clearly defined and no lung markings are observed peripheral to it. A small pneumothorax (especially if it is present on the mediastinal surface of the lung) may easily be overlooked, however, unless careful inspection is made. Radiographic examination by films taken in full inspiration and expiration and by screening is helpful in difficult cases. A fluid level may be present. It is sometimes difficult to differentiate between a localized pneumothorax and a large bulla or a large cavity.

Although it is not uncommon for a small pneumothorax to pass unrecognized, there is rarely difficulty in the diagnosis of an acute spontaneous pneumothorax where there are characteristic symptoms, provided that careful examination of the chest is carried out. Where there is severe pain, particularly if it is accompanied by a degree of shock, acute abdominal or cardiac emergencies may be simulated, but the rapidity of the onset of severe dyspnoea is a helpful distinguishing feature. A superficial resemblance to pleurisy may give rise to diagnostic difficulty, but again careful physical examination should be able to differentiate the condition. The signs associated with clicking or noisy pneumothorax may be confused with pericarditis. As with many conditions, awareness of the possibility of its occurrence is important in the diagnosis of spontaneous pneumothorax, particularly in emphysematous patients, in whom the diagnosis may be very difficult to make without radiographic examination.

Management

There is wide divergence of views regarding the management of uncomplicated spontaneous pneumothorax, varying from observation alone to the carrying out of bilateral pleurectomy for a unilateral lesion. There are cogent arguments against pursuing a wholly conservative approach as routine management. The behaviour of a spontaneous pneumothorax is unpredictable, and a deterioration in the situation because of a further leak of air into the pleural space may be potentially dangerous, particularly in emphysematous patients. It also means that an unnecessary delay has occurred in the patient's treatment, giving rise to prolongation of hospitalization. Furthermore, a free pleural space is liable to become infected. Conservative treatment often is synonymous with a prolonged period of hospitalization and absence from work, and the end-result may be an unexpandable lung: but that is not to say that there is no place in management for a conservative approach. The choice of treatment is also influenced by other factors—the history of recurrent pneumothorax, either ipsilateral or bilateral; the presence of underlying lung disease; and the presence of fluid (which may be blood) in the pleural space.

Emergency Treatment

The need for emergency treatment outside hospital is fortunately very rare, but very occasionally—in patients with asthma or gross emphysema, for instance, and in tension pneumothorax—urgent treatment is required. The most convenient method of dealing with the situation is to insert a needle—one the size of a blood transfusion needle would be appropriate—through the second or third interspaces in the mid-clavicular line on the affected side, when air will escape from the pleural space. Before the needle is inserted a finger-cot should be tied tightly around its collar, a V-shaped cut being made in the rubber with a pair of scissors; the cut rubber acts as an effective valvular mechanism until definitive treatment may be given. Oxygen should be administered, and further emergency treatment may also be required where haemopneumothorax has occurred with severe bleeding.

Elective Treatment

Each physician and surgeon has his own view regarding the method of choice in the treatment of spontaneous pneumothorax. The author has tried to present as balanced a view as possible, though in some respects it is necessary to be didactic.

Conservative management by expectant observation is appropriate only in the management of a small pneumothorax in the previously healthy young adult. A small pneumothorax in this context is usually defined as one in which the lung is less than 20% collapsed. The progress of the condition should be observed by radiographic examination every 3 to 5 days until complete expansion has occurred. Prompt radiographic examination is indicated if the symptoms increase in severity, and active treatment is necessary if the pneumothorax increases in size. The patient's activities are usually limited during the period of observation, though strict rest in bed is not necessary. Even with a small pneumothorax, conservative treatment is inappropriate in the management of patients who have impaired respiratory reserve or who have a history of contralateral pneumothorax. The risks of conservative management have already been stated.

Aspiration of air through a needle is less commonly practised than it used to be. A needle of the Foster-Carter type is inserted into the intrapleural space and is connected to a water-seal drainage system by rubber tubing. The indwelling needle may be given additional stability by being passed through a large rubber cork applied to the thoracic wall, the cork being securely strapped to the chest wall. Such needles are, however, of too narrow a bore to secure rapid expansion of the lung, are liable to become blocked easily, and, most important of all, are readily dislodged and may be responsible for a further rent in the lung, with disastrous consequences. Where a small pneumothorax exists some authorities recommend aspiration of air through a needle which is left *in situ* for a short period, the intrapleural pressure being measured at the end of aspiration. It is, however, impossible to aspirate all the air without serious risk of damaging the lung. It is the author's opinion that there is no place for needle aspiration of air in the management of spontaneous pneumothorax except in situations where it is totally impossible to employ closed-tube thoracostomy with a rubber catheter.

The insertion into the pleural space of a rubber catheter connected to a water-seal drainage system is widely accepted as being the method of choice in the management of a large pneumothorax, and in other circumstances—poor respiratory reserve, bilateral pneumothorax—where rapid expansion of the lung is desirable.

Under local anaesthesia, a trocar and cannula is thrust into the pleural space through the second intercostal space in the mid-clavicular line. The fourth or fifth intercostal space, just

behind the anterior axillary line, may be chosen if there are adhesions between the lung and the thoracic wall, or for cosmetic reasons. The trocar is withdrawn and a self-retaining Malecot catheter (size 22–28), stretched on an introducer, is passed through the cannula. The latter is threaded off the catheter, the introducer being released. The catheter is then gently withdrawn until the expanded portion is just within the pleural space, and attached to a water-seal drainage system which allows air to leave the pleural space but prevents it from re-entering.

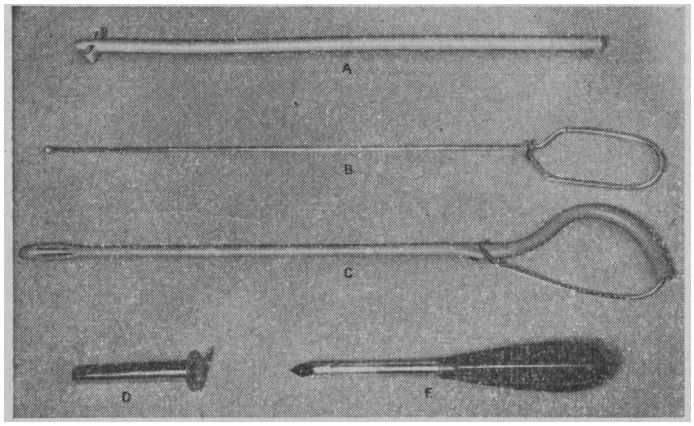

FIG. 1.—Instruments used in intercostal drainage. A, Self-retaining Malecot catheter (size 22–28). B, Introducer. C, Catheter stretched on introducer. D, Cannula. E, Trocar.

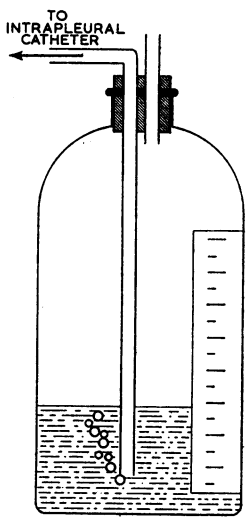

FIG. 2.—Water-seal drainage. The intrapleural catheter is connected to the longer glass tube, thus permitting escape of air under positive pressure. When negative pressure develops, water enters the longer glass tube and the height of the water column is a measure of the intrapleural pressure. The capacity of the drainage bottle should be three to four litres.

Some controversy exists regarding the wisdom of using suction. To be effective, suction must remove air faster than it leaks into the pleural space from the broncho-pleural fistula, and as even a small fistula may leak many litres a minute suction is usually reserved for desperately ill patients with anoxia, when very rapid expansion of the lung is desirable, or for cases where expansion is not satisfactory after 24 to 48 hours. A small electric pump is applied to the outlet of the drainage bottle, and, starting with a negative pressure of about 2 cm. Hg, suction is increased gradually to 10 cm. Hg. Breathing and coughing move the column of water in the inlet tube unless the tube becomes blocked by fibrinous exudate. A second tube may be inserted, usually in a lower intercostal space, if the rate of expansion of the lung is unsatisfactory. The tube or tubes are usually left *in situ* for 24 hours after the lung is shown to be re-expanded, and the fistula closed, though some authorities recommend waiting seven days in all cases. The tube is withdrawn usually under sedation with pethidine or an equivalent drug. Closed-tube thoracostomy is successful in a very high proportion of patients, and has the additional advantage that should the lung collapse again soon after the tube is withdrawn there has usually been a sufficient reaction at the site of intubation for adhesions to have taken place locally, thus preventing a total collapse.

Open thoracotomy is indicated in patients who have unexpanded lungs due either to a persistent fistula, or to a fibrous cortex over the lung, or to both; in haemopneumothorax; and in recurrent pneumothorax. The extent of the operation will depend upon the nature of the problem as defined at thoracotomy. The parietal pleura is stripped in order to produce an extensive raw surface to which the visceral pleura can adhere. Sometimes it is found possible and appropriate to suture the leaking bulla or bullae, and in a few cases of localized disease it may even be considered advisable to remove a segment or lobe. The operation of pleurectomy for recurrent spontaneous pneumothorax is uniformly successful and the patient is usually in hospital only a few days.

In some centres irritant substances are instilled into the pleural space with the aim of promoting an inflammatory reaction of the pleural surfaces, in the hope that they may become adherent. The substances commonly used are (*a*) 10 ml. of 1% camphor in oil, (*b*) 0.5 ml. of 5% silver nitrate, (*c*) a few ml. of the patient's own blood, (*d*) 2 ml. of 25% suspension of kaolin, and (*e*) 2–5 g. of 1% iodized talc. The introduction of some of these substances frequently produces severe discomfort and is followed by a period of fever. More important, there is little evidence that the recurrence rate is significantly reduced by these methods, with the exception of poudrage by talc scrupulously carried out through two intercostal cannulae. The instillation of irritant substances is, in the author's opinion, justifiable only for patients whose condition does not warrant open thoracotomy and pleurectomy.

Complications

The main complications of spontaneous pneumothorax are infection of the pleural space (tuberculous or staphylococcal), haemorrhage, atelectasis due to plugging of bronchi by secretions, and recurrence of the pneumothorax. Haemorrhage into the pleural space is sometimes profuse, and the patient may be profoundly ill, for the bleeding is at systemic pressure. Treatment is by means of formal thoracotomy, at which the clot is removed, bleeding controlled, and the cortex peeled off the lung. Blood transfusion may be required. Clot-dissolving enzymes are so rarely successful that they are considered unnecessary in management. Atelectasis may occur at any stage in the management of spontaneous pneumothorax, especially in patients with asthma or bronchitis. Physiotherapy and, if necessary, bronchoscopy should be instituted if atelectasis occurs.

There is some difference of opinion regarding the timing of pleurectomy for recurrent pneumothorax. The extreme view is sometimes taken that bilateral pleurectomy should be carried out after the first incident of spontaneous pneumothorax. It would seem reasonable to recommend pleurectomy if three ipsilateral episodes have occurred. The occurrence of a contralateral pneumothorax is obviously an indication to carry out pleurectomy with the minimum of delay. It must be remembered, however, that in the grossly emphysematous patient thoracotomy and pleurectomy may be too hazardous a procedure, and recourse may have to be made to irritant substances despite their disadvantages.

Other Aspects of Management

It is important to remember the value of oxygen therapy, particularly for patients who are severely ill with tension pneumothorax, and for patients with impaired lung function due to emphysema—the usual precautions being taken in the latter

group to avoid over-administration of oxygen. Morphine should be given to allay pain and anxiety, again with the proviso that it should be given with caution in patients with chronic lung disease. Appropriate antibiotic therapy should be given immediately in the presence of pulmonary infection.

Though they are rare causes of spontaneous pneumothorax, where tuberculosis or bronchogenic carcinoma is considered possible, appropriate steps—sputum culture and cytology and bronchoscopy—should be taken. It is customary, therefore, for patients with spontaneous pneumothorax to be kept under supervision for 12 months with radiographic examination at 4–6-month intervals, even though it is exceedingly rare for any abnormality to develop if the radiograph at the time of the spontaneous pneumothorax was normal.

ANY QUESTIONS?

We publish below a selection of questions and answers of general interest.

Goitre in Pregnancy

Q.—*What is the likely effect of pregnancy on a simple euthyroid goitre in a young woman who is now four months pregnant? The goitre is much smaller after nine months' treatment with thyroxine sodium 0.1 mg. twice a day. Should this dosage be changed?*

A.—Goitres are frequently found in pregnant women, but the gland usually decreases in size after the pregnancy. Several factors are responsible for the development of the goitre. In pregnancy renal iodide clearance is twice the normal, causing a lowering of the plasma inorganic iodine and an increase in the thyroid-iodide clearance. Crooks *et al.*[1] have suggested that this induced iodine deficiency might be a major cause of pregnancy goitre.

Increased oestrogen levels during pregnancy cause a rise in the concentration of circulating thyroxine-binding-globulin (T.B.G.).[2] The initial effect of this elevation would be to lower the free thyroxine which is in equilibrium with bound thyroxine leading to stimulation of the thyroid gland via pituitary thyroid-stimulating hormone (T.S.H.). Increased secretion of thyroid hormone then restores the equilibrium with higher levels of bound thyroxine on T.B.G. and normal levels of free thyroxine, the patient remaining euthyroid at the expense of increased thyroid activity.

Without treatment pregnancy is likely to aggravate an existing goitre. In the patient mentioned the definite reduction in the size of the gland after treatment with L-thyroxine suggests that the goitre was mediated by increased T.S.H. stimulation in response to a lowered thyroid output from the gland. The thyroid reserve is likely to be reduced in this case and may be unable to meet the increased demands of pregnancy. It would probably be advisable to increase the dose of L-thyroxine to 0.3 mg. a day over the period of the pregnancy.

REFERENCES

1 Crooks, J., Aboul-Khair, S. A., Turnbull, A. C., and Hytten, F. E., *Lancet*, 1964, **2**, 334.
2 Tata, J. R., *Brit. med. Bull.*, 1960, **16**, 142.

Oral Contraceptives and Fibroadenomata of Breast

Q.—*Is it wise for a woman aged 42 with a history of fibroadenomata of the breast to take oral contraceptives?*

A.—Fibroadenomata of the breast are not a contraindication to the use of oral contraceptives—indeed, their use may lead to objective improvement in the mastopathy. Theoretical considerations would suggest that a formulation containing a low rather than a high dose of oestrogen would be preferable.

The question of whether it is wise for any woman aged 42 to use oral contraceptives is, of course, another matter entirely, and could be argued at great length. The most important points are that oral contraceptives provide the maximum protection against unwanted pregnancy, evidence for harmfulness continues to be unconvincing, while the risks, physical as well as mental, of an unwanted pregnancy at this age are far from merely conjectural.

Chediak Test for Syphilis

Q.—*What is the Chediak test for syphilis?*

A.—The Chediak test for syphilis is a flocculation test which has the advantage over similar tests that it can be performed on dried blood.[1–3]

REFERENCES

1 Ghosal, D. K., and Bhattacharyya, A. K., *Indian J. Derm.*, 1964, **10**, 23.
2 Olansky, S., Harris, A., Vinson, H., Bossak, H. N., and Portnoy, J., *Publ. Hlth Rep. (Wash.)*, 1952, **67**, 563.
3 Harris, A., Olansky, S., and Vinson, H., ibid., 1952, **67**, 572.

Gout and Allergic Manifestations

Q.—*Is there any relationship between gout and recurrent angioneurotic oedema, or is their occurrence in a patient of mine merely fortuitous?*

A.—Hyperuricaemic gout is not associated with a tendency to allergic manifestations in general or angioneurotic oedema in particular. It is therefore unlikely in this case that the recurrent angioneurotic oedema has any connexion with the patient's gout.

However, there are two other possibilities. First, the treatment which the patient is receiving must be considered. While probenecid and colchicine are not considered to be drugs which cause allergic reactions,[1] acetylsalicylic acid may do so in up to 0.2% of patients.[2] This would appear to be a personal idiosyncrasy and is not infrequently associated with asthma, or may occur in a patient already suffering from asthma. Such an allergic response to aspirin usually develops when it is first used, but this does not exclude the subsequent appearance of hypersensitivity. Phenylbutazone may cause skin rashes but not usually urticaria or angioneurotic oedema.[3]

Secondly, attacks of angioneurotic oedema itself may conceivably precipitate attacks of acute gout, as may any "stress," the classical example being in the post-operative period.[4] This may be due to as yet unexplained metabolic changes, mild dehydration (as with the use of diuretics, although the thiazides also have an inhibiting effect on the tubular secretion of uric acid[5–7]), or to the "stress" effect on the body producing a similar effect to the withdrawal of A.C.T.H.[8] which can exacerbate attacks of gout. Antihistamines, which may be being used for the angioneurotic oedema, are not among the drugs which are associated with hyperuricaemia.

REFERENCES

1 Goodman, L. S., and Gilman, A., *Pharmacological Basis of Therapeutics*, 2nd ed., 1955. Macmillan, Toronto.
2 Meyler, L., *Side-effects of Drugs*, 4th ed., 1963. Excerpta Medica Foundation, Amsterdam.
3 Von Rechenberg, H. K., *Phenylbutazone*, 2nd ed., 1962. Arnold, London.
4 Copeman, W. S. C., *Textbook of the Rheumatic Diseases*, 3rd ed., 1964. Livingstone, London.
5 Borhani, N. O., *Ann. intern. Med.*, 1960, **53**, 342.
6 Demartini, F. E., Wheaton, E. A., Healey, L. A., and Laragh, J. H., *Amer. J. Med.*, 1962, **32**, 572.
7 Oren, B. G., Rich, M., and Belle, M. S., *J. Amer. med. Ass.*, 1958, **168**, 2128.
8 Gutman, A. B., and Yü, T. F., *Amer. J. Med.*, 1950, **9**, 24.

Prednisone and Skeletal Growth

Q.—*What is the mechanism by which prednisone retards skeletal growth in children? Is there anything that can be done to counteract this effect in cases in which continued administration of corticosteroid is necessary?*

A.—The mechanisms whereby prednisone and related corticosteroid hormones retard skeletal growth are probably closely related to the antianabolic effects of these hormones. Cortisol interferes with protein synthesis and also accelerates protein catabolism and conversion into carbohydrate (gluconeogenesis).

When long-continued administration of steroids is necessary the smallest dose capable of achieving the desired effect should be used. If in the management of a child with rheumatoid arthritis or severe chronic asthma it is decided to embark on long-continued steroid therapy—never an easy decision—the aim should be not necessarily to abolish all symptoms but to relieve them sufficiently to enable the child to lead a full and active life. A dose of prednisone not exceeding 5 mg. per sq. m. body surface is unlikely to lead to significant retardation of linear growth. When steroid therapy is stopped growth is resumed at a normal or even accelerated rate.

Therapy with one of the protein anabolic drugs has been suggested to counteract the antianabolic effect of the steroid hormones. Because the anabolic agents are androgens,