to physicians and laboratory workers alike the necessity for bacteriological control of enteric fever.

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COLLAPSE THERAPY IN BRONCHIECTASIS

A WARNING

BY

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Collapse therapy has earned a great reputation in the treatment of pulmonary tuberculosis. A bronchiectatic cavity, on the other hand, differs from a tuberculous one in that it is lined with epithelium, and, however long two epithelial surfaces are in contact, they will never unite. In bronchiectasis, therefore, the procedure cannot be regarded as a curative, but only as a palliative, treatment. Collapse therapy in bronchiectasis possesses, however, a disadvantage which I do not think is fully appreciated by those who advocate it—namely, that it greatly diminishes the possibility of a successful lobectomy, should this become necessary. In the modern one-stage lobectomy it is essential to obtain an early expansion of the remaining lobe, and so obliterate the pleural space before infection can set in. A paralysed diaphragm removes the principal factor in expanding the lung, and a prolonged artificial pneumothorax so anchors the remaining lobe that it will not expand quickly enough. I would like to quote two unfortunate experiences which have prompted me to give this warning to others.

Case 1.—A woman, aged 28, was admitted to St. Andrew's Hospital, Bow, on January 11th, 1933. Seven years ago she had a so-called pneumonia on the right side, followed by an empyema, which healed with rib resection and drainage. Since that time she had had a persistent cough, with about one ounce of offensive sputum every morning. Four years ago she started to spit up blood (about four ounces at a time), and this recurred three or four times every year. By 1932 the condition had become much worse, and the bleeding was more frequent. Her sputum was negative for tubercle bacilli. Lipiodol demonstrated severe bronchiectasis in the lower lobe. In November, 1932, a right phrenic evulsion was performed, but following this there was no improvement in the symptoms. In January, 1933, I saw her for the first time. She was then having severe haemoptyses every week, was anaemic, and it was obvious that something had to be done to try to relieve her condition. On February 8th I performed a right lower lobe lobectomy. Owing to the previous empyema there was a mass of adhesions round the lower lobe, particularly on the costal surface, but by entering the endothoracic space I managed to strip the lobe free. There were adhesions in the interlobar fissure, but none over the upper lobe. The diaphragm was absolutely rigid and board-like, and there was very little shift of the mediastinum. During removal of the lower lobe a little pus was spilt into the pleural cavity. At the end of the operation the chest was drained by a closed waterlocked drainage. The upper lobe in this case failed to expand, and the patient died on the eighth day after operation from a generalized infection of the right pleural cavity and a bronchopneumonia on the opposite side.

Normally, the upper lobe expands to fill the whole hemithorax within the first day or two after operation, according to physical signs, *x* rays, and post-mortem observations. This case, I think, illustrates that with a paralysed diaphragm one has removed the principal factor in expanding the lung,

and there is extreme risk of the patient dying from an infection of the pleural cavity.

Case 2.—A girl, aged 19, was admitted to St. Bartholomew's Hospital on September 21st, 1933, under Dr. F. G. Chandler. At the age of 3 she had had double pneumonia, and since then had had cough and sputum. In 1929 lipiodol demonstrated a bronchiectasis in the left lower lobe. She then had several ounces of offensive sputum daily. An artificial pneumothorax was therefore induced, and maintained with monthly refills of 500 c.cm. There was at no time any pleural effusion. The pneumothorax succeeded in reducing her sputum to a few drachms of inoffensive sputum daily, but by 1932 this had started to increase again. The lung was almost completely collapsed, except for a small part situated behind the heart shadow. Lipiodol demonstrated dilated bronchi in the collapsed portion of the lung. By September, 1933, she was producing one ounce of sputum in the twenty-four hours. A lobectomy was therefore advised, and was performed on October 17th. On opening the chest I found that the whole pleural cavity was lined by a thick white membrane, which completely obscured the anatomical landmarks. This was stripped off the lower lobe and a left lower lobe lobectomy performed, but with some difficulty owing to adhesions in the interlobar fissure. After removing the lobe I attempted to decorticate the white membrane off the upper lobe. This, however, was only partially successful, as it was firmly adherent in one place. At the end of the operation the chest was drained with a closed waterlocked drainage. This patient now (December, 1933) has a complete empyema on the left side, and there is practically no expansion in the remaining upper lobe. I fear that we will therefore have substituted a large chronic empyema for a left basal bronchiectasis.

In an artificial pneumothorax of more than a year's duration, even in those cases where there is no pleural effusion, the lung is likely to be encased in a white membrane and expansion of the remaining lobe therefore severely hampered. In future, before performing a lobectomy in a case of artificial pneumothorax, I shall inspect the pleural cavity with a thoracoscope.

DISCUSSION

After a *phrenic evulsion* for a basal bronchiectasis there may be a temporary improvement, but subsequently, when the diaphragm has completely atrophied, the patients return to the same, or a worse, condition. In cases of dry bronchiectasis, suffering from haemoptysis only, I have seen a definite improvement in symptoms following paralysis of the diaphragm. It is, however, advisable first to try a temporary paralysis, by crushing the nerve, to see if this is going to be effective in controlling the bleeding.

Artificial pneumothorax frequently fails owing to extensive pleural adhesions, but when a complete collapse is obtained the symptoms may disappear, as in those cases quoted by Dr. F. G. Chandler and referred to by Dr. Beaumont in the correspondence in the *British Medical Journal* of October, 1933. The collapse must, however, be maintained by refills for the rest of the patient's life, and, should oblitative pleurisy develop, with subsequent expansion of the lower lobe, one has probably burnt one's boats as far as lobectomy is concerned.

Thoracoplasty for basal bronchiectasis has, on the whole, given extremely bad results, and involves the sacrifice of a whole lung for a basal lesion, which is obviously wrong. But this operation still has a place in the treatment of unilateral bronchiectasis, when all the lobes are grossly affected and there is a large quantity of sputum. By means of a very extensive resection it is possible in these cases to reduce the sputum from, say, 20 ounces to one or two ounces; occasionally, they may even be sputum-free, but in the majority it recurs again—although never, of course, to the same extent as before operation.

Thus the choice of therapy in basal bronchiectasis seems to me to rest between (a) posture and bronchoscopy, and

(b) lobectomy, which in selected cases carries a mortality of between 8 and 10 per cent.

CONCLUSION

The object of this paper is to point out that, when collapse therapy is applied to cases of bronchiectasis, it must be appreciated that such therapy may exclude the possibility of a subsequent lobectomy, should it become necessary.

Memoranda

MEDICAL, SURGICAL, OBSTETRICAL

HERNIA OF THE LUNG

This condition, although rare, is nevertheless of sufficient clinical interest to justify recording the details of a case which came under my notice. Most surgical textbooks give little more than a passing reference to the subject.

CASE HISTORY

A miner, aged 30, was injured while at work in the pit on January 28th, 1929, being struck heavily on the back by a loaded "tub." He was admitted to the General Infirmary at Leeds under the care of Mr. J. F. Dobson soon after the accident. He was found to have a fracture of the body of the right scapula, fractures of the second, third, and fourth right ribs in the antero-lateral wall of the thorax, and a considerable subcutaneous tearing of the fibres of the overlying pectoralis major. He was treated by the application of strapping, and was discharged to the care of his panel doctor ten days later. Soon after his arrival home he had what was described as an attack of acute bronchitis.

He was next seen in May, 1930, in the out-patient department, complaining of persistent pains and grating sensations over the right side of the chest, together with a spasmodic, unproductive cough. On examination there were harsh rhonchi and pleural friction sounds at the right base. X-ray examination of the chest showed malunited fractures of the second, third, and fourth ribs on the right side; the lungs were clear save for some increase in the root-shadows. In July, 1930, he was noticed to have a definite defect in the right chest wall and pectoralis major just above and to the outer side of the right nipple; the defect measured about three and a half inches in its vertical diameter and two and a half inches transversely. Through this defect a soft, crepitant mass appeared on deep inspiration, and, to a greater degree, on coughing; this was obviously a hernia of the lung. A corset with a pad to fit the defect was supplied.

Early in 1931 the man was given light work in the lamp-room at the colliery; he carried on this work satisfactorily for about two or three months, but subsequently he said he was unable to do any kind of work. In November, 1932, he was seen again; the corset controlled the hernia satisfactorily, but he complained of much spasmodic cough and shortness of breath. The hernia was no larger than when it was first seen, and there was no other abnormality to be detected in the chest on physical examination. X-ray examination of the chest showed nothing abnormal save the malunited fractures of the ribs.

COMMENTARY

Aetiology.—Hernia of the lung, or pneumatocele, may be of congenital or acquired type. The congenital herniae occur at the site of developmental defects in the thoracic parietes, and usually are incompatible with life. The acquired herniae may be of traumatic origin, or may follow some pathological process in the chest wall, or may even arise spontaneously. Most commonly they are of traumatic origin, following such injuries as stab and gunshot wounds which damage intercostal muscles or nerves, or the ribs themselves; some follow non-union or malunion of fractured ribs. The traumatic type of hernia

may appear soon after injury, or, in what Morel-Lavallée¹ termed the "consecutive" type, the hernia appears some considerable time after the injury, presumably on account of the yielding of scar tissue. Spontaneous hernia of the lungs occurs in such people as glass-blowers and wind-instrument players; it has also been recorded as occurring during labour. While traumatic cases may arise at any situation in the chest wall, spontaneous herniae usually are found anteriorly near the sternum, where the external intercostal muscle is replaced by membrane. At the corresponding place posteriorly the weak area is guarded by the mass of erector spinae, although Wightman² recorded a case of spontaneous pneumatocele in a trombone player beneath the trapezius and erector spinae, about one and a half inches to the right of the seventh dorsal spine. Sometimes the hernia protrudes into the neck through a defect in Sibson's fascia, and rarely into the abdomen through a defect in the diaphragm. Pathological causes include caries of the ribs, empyema necessitatis, lung abscesses, and the extensive operations of Estlander and Scheede, but such herniae are very uncommon. Most pulmonary herniae occur in males, usually under the age of 45.

Prognosis.—Inflammatory changes may lead to the formation of adhesions between the sac and its contents, with resulting irreducibility; strangulation is rare, and spontaneous cure practically never occurs.

Treatment.—This must depend on the type of hernia and the cause. A hernia through a gap in Sibson's fascia is probably best treated conservatively; Chorel³ treated one such hernia by means of a pad and bandage. In cases of hernia through the bony chest wall treatment of the cause, such as caries of a rib, may result in cure. In traumatic and spontaneous cases palliative treatment may suffice; this involves the fitting of a corset with an obturator to fit the defect in the chest wall. It may also be necessary in such cases to modify the patient's occupation, although the disability when following occupational injuries sometimes diminishes in a remarkable fashion when the claim for compensation has been settled. Operative treatment should be directed in the first place to the treatment of any pathological process in the chest wall. Cure has been reported in cases of cervical pneumatocele after packing the hernial sac with iodoform gauze. Simple excision after ligation of the sac has been performed on several occasions. Montgomery and Lutz⁴ turned an osteochondral flap upwards from the fifth rib on a periosteal hinge to close a gap in the fourth intercostal space in a case of pneumatocele following a stab wound; this operation resulted in cure, and for herniae through the bony parts of the thoracic parietes this type of repair seems to be the most practicable.

The case recorded above is clearly of the consecutive type; eighteen months elapsed between the date of the accident and the appearance of the hernia. As a result of the chronic cough the scar tissue in the chest wall and pectoralis major yielded, allowing the hernia to appear. As far as the treatment is concerned the defect in the chest wall is rather large for satisfactory closure by means of an osteochondral flap; the hernia is controlled adequately and comfortably by the corset.

I am indebted to Mr. J. F. Dobson for permission to publish this case.

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