and he felt bound to stand still for a few minutes, after which he was able to carry on again. The pain had never occurred at night, but was definitely produced more easily after food. The attacks had gradually got more frequent, though not more severe, and the exercise of quiet walking was sufficient to bring one on. Latterly he had had two or three attacks a day.

On physical examination the heart was not enlarged. An x-ray outline was taken, and this showed a heart of normal shape, but the aortic shadow was wider than normal. The heart sounds were clear, though the first sound was rather high-pitched; the aortic second sound was not accentuated. The arteries were not thickened, and the blood pressure was 120/70. The urine was normal, the Wassermann reaction negative, and no abnormal signs were present. Another electro-cardiogram was taken (Fig. 2), and this showed a marked difference in the "T" complexes.

FIG. 2.

Whereas in the former one all the T's were positive, in this second one T2 is barely positive and T3 is definitely negative.

A month after this examination he fell down dead when getting into a tram-car.

Post-mortem Examination.

The post-mortem examination was performed at the Bristol Royal Infirmary by Dr. A. D. Fraser, and by his kindness one of us (C. B. P.) was able to examine certain organs in detail.

The subject was a well-nourished male about 40, and there were no external evidences of trauma or of disease. The lungs were normal in appearance, as also were the abdominal organs, with the exception of the aorta and kidneys. The aorta in its thoracic part showed slight atheroma, but the atheromatous change was very marked in the abdominal portion. There were several atheromatous ulcers, and in one or two places calcification had occurred. On section the aortic lesions show pronounced intimal thickening. In most places the media is the seat of replacement fibrosis. The intimal lesions in some parts show extensive fatty degeneration, and around the fat deposits are a certain number of mononuclear cells with a foamy cytoplasm, apparently engaged in phagocytosis of the fat. This is the only cellular reaction to be seen in the lesion.

The heart was somewhat enlarged, and on palpation the coronary arteries were definitely hard and thickened. It was injected with a barium gelatin emulsion, through the coronary arteries, according to the technique described by Gross,¹ and x-ray photographs were taken.

The skiagram of the injected heart shows intense vascularization of the left ventricle. This increase in vascularity of the left side compared with the right corresponds to a very extreme degree of the "age change" described by Gross, although in this case it is much more marked than one would expect at twice the age. At the age of 40 the increase of the blood supply of the left ventricle over the right is normally only just becoming obvious. In both the right and left coronary areas there are obstructions to the circulation.

All four valves were normal in appearance. On microscopical examination the myocardium shows areas of dense fibrosis. The coronary arteries are in many places almost completely occluded by massive intimal thickening, similar in type to that occurring in the aorta. In places this intimal thickening has been vascularized by new capillaries. Both internal and external elastic laminae are, in places, broken up, and the media shows some increase in the amount of fibrous tissue it contains. There appears to be some increase in the adventitial layer of fibrous tissue.

The kidneys were normal in size and appearance. There was very little abnormality in the small renal vessels; the renal artery shows medial fibrosis, but no intimal change.

The cause of death would appear to have been impairment of the coronary circulation due to degenerative changes in the coronary arteries. The pathological process corresponds with that of a premature senility of the arterial tree, with a particular incidence on the coronary arteries. There is no evidence of any infective factor. The pathology of the coronary artery lesions resembles that in the case described by Perla and Seligman,² except that

in their case the lesion was by no means confined to the coronary arteries.

We may also refer to a recent paper from this laboratory in which Hughes and Perry³ report the case of an infant, aged 7 weeks, who died suddenly, and was found at the necropsy to exhibit advanced degenerative changes in the coronary arteries.

REFERENCES.

- ¹ Gross: *The Blood Supply of the Heart*.
- ² Perla and Seligman: *Arch. of Path.*, vii, 1, 55, January, 1929.
- ³ Hughes, F. W. T., and Perry, C. B.: *Bristol Med.-Chir. Journ.*, 1929, xlv, 173, p. 219.

THE USE OF SURGERY IN PULMONARY TUBERCULOSIS.*

BY

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IN the treatment of pulmonary tuberculosis, as in that of all other diseases, the simplest methods, carrying the least risk, must be tried first and continued as long as they show a possibility of succeeding. The simplest treatment for many patients with pulmonary tuberculosis is sanatorium treatment, being generally without risk. In a few cases, however, there is the danger that haemorrhage may curtail life before healing takes place under sanatorium regime. Again, sanatorium treatment may be inadequate on account of the acuteness of the disease, weak powers of resistance, recurrence of infection, or because the character of the changes, whether the result of the tubercle bacilli, or of the secondary infection, or of the resulting fibrosis, is such that healing cannot occur without the application of radical additional aid.

In this article it is proposed to consider the problems of how to diminish the relative virulence of the tubercle bacilli to the resistance of the individual, and of how to reduce the anxieties and dangers of haemorrhages: how, further, the healing processes and the eradication of the infectivity of the individual can be hastened when the time factor is of importance: and finally, how we can overcome the persistent infections of single large or of multiple small cavities in the parenchyma or in the dilatations of the bronchi.

Where one lung is involved the object is achieved if collapse can be readily secured, but this is not always possible. If the second lung is implicated, the radical treatment of the more seriously affected side may have to be greatly modified, and may even be impracticable.

The simplest, but not always the easiest, method of collapse is that of artificial pneumothorax. The purpose here, however, is to discuss those surgical methods which may be accessory or alternative to artificial pneumothorax—namely, hemidiaphragmatic paralysis by phrenic evulsion, thoracoplasty, multiple neurectomy, and extrapleural pneumolysis. If it were always possible to get a perfect collapse with artificial pneumothorax, and if there were no complications to this method of treatment, there would be less need for these other operations. What often indicates the necessity for the operations of thoracoplasty or phrenic evulsion are the failures to obtain complete collapse by artificial pneumothorax, even with the aid of the cautery for dividing adhesions, and the various complications of pneumothorax.

The Operation of Evulsion of the Phrenic Nerve.

This operation consists in exposing the nerve in the neck, dividing it, and then, by traction on the distal end, drawing out the intrathoracic part of the nerve. In a case of complete evulsion the terminal filaments will be torn out from the diaphragm. It is not advisable, however, in certain cases, such as in those in which the nerve is caught up by thickened pleura, to attempt such a complete evulsion; it is satisfactory if some 10 to 12 cm. of the nerve can be removed.

* Contribution to a discussion at the Medical Society of London, February 10th, 1930.

Evulsion of the phrenic nerve has almost entirely superseded simple section, because, by evulsion, it is almost always possible to destroy the connexion of the accessory phrenic with the intrathoracic part of the nerve, and so interrupt this possible additional source of supply to the diaphragm. The operation is performed under local anaesthesia, except in young children. Under ordinary conditions an incision 2 to 3 cm. long is sufficient. Abnormalities of the phrenic nerve are frequent and varied, and it may be necessary to enlarge the incision slightly in order to make certain of finding and distinguishing the nerve which is to be evulsed. At times also, enlarged glands, or the scar tissue of old glandular infections, interfere considerably with the easy exposure of the nerve.

After evulsion, the dome of the diaphragm is paralysed and the muscle fibres gradually atrophy. Three changes occur in consequence. Where the muscle was in a state of tonic spasm, that spasm disappears; when normal or limited respiratory movements were present, they are abolished or may even be replaced by paradoxical movements, the paralysed dome rising as the unaffected dome descends, and vice versa. The third change is the rise of the dome into the thorax, which gradually increases during the ensuing two or three months as the muscle fibres atrophy.

Each of these changes exercises a favourable effect. The paralysis and loss of tone frees the lung, shrunken by the contraction of the pathological fibrous tissue, from the constant strain of the counter-drag of the muscle; while the rise of the dome, and the paradoxical movements when present, allow of a corresponding partial collapse of the lung, which in turn eases, and may even abolish, the drag of the shrunken lung on the mediastinum and its contents. The partial collapse of the lung is not limited to the base; this is strikingly illustrated in some cases by the great diminution in the size of cavities near its apex.

The relief of such symptoms as pain, aching drag, tightness round the base of the lung, and even of irregular tachycardia, when the pericardium is involved by basal adhesions, is among the most immediate results of hemidiaphragmatic paralysis; the presence of such symptoms is therefore an indication for phrenic evulsion. Hemidiaphragmatic paralysis may have a considerable influence also on certain other symptoms, such as cough and sputum. That form of dry recurrent cough due to irritation of the diaphragm is completely abolished. The bringing up of sputum is facilitated, because the expulsive force of the abdominal muscles acts directly on the lung, and is no longer damped by the semi-rigid, tonically contracted muscle.

Very briefly stated, the indications for phrenic evulsion are:

1. In cases of basal tuberculosis. It can be tried also in cases of basal bronchiectasis, but it is only rarely that any real or lasting success is obtained.
2. In cases of chronic basal effusions with thickened pleura; partly to reduce the space, and partly to relieve the dragging sensation.
3. In cases of more generalized tuberculosis when artificial pneumothorax has been tried and has failed.
4. For the relief of symptoms such as pain, dragging, cough, and tachycardia.
5. As an accessory to artificial pneumothorax when the base of the lung is adherent. It is also of particular value in that it lengthens the intervals between refills. Hemidiaphragmatic paralysis is said to help in preventing effusions or in checking the recurrence of them, but I am not convinced that this is so. Phrenic evulsion can be done to check a basal extension in the opposite lung. It should always be done when a lung, long collapsed by artificial pneumothorax, is being allowed to re-expand, so as to reduce the size of the pleural cavity.
6. As a preliminary to thoracoplasty. The chief object is to reduce the number of the lower ribs which require excising.
7. As a prophylactic measure, to prevent the later development of bronchiectasis in a case with an unresolved basal pneumonia.

Thoracoplastic Operations.

The basis of all thoracoplastic operations is the excision of parts of ribs so as to produce collapse of the chest wall by mobilizing these ribs. This has been found to be less drastic than decostalization—that is, the removal of the whole length of the ribs. One essential condition of the operation is that the posterior line of division of each rib should be made at the level of the transverse processes. Another is that the excision of ribs should be carried up to and include the first, since each rib is fixed to the one above by the intercostal muscles and aponeurosis. When this has been done it will be found (1) that the ribs drop downwards, especially at the posterior ends, so that the cut end of the first rib may now lie opposite the transverse process of the third dorsal vertebra, and the others correspondingly; (2) that the lateral cut end of the ribs has become approximated to the spine; (3) that each of the ribs has become tilted downwards (bucket-handle action). These three actions cause the chest wall to fall mesially towards the mediastinum, so that very considerable collapse of the lung is produced.

According to the original paravertebral, extrapleural thoracoplasty as devised by Sauerbruch, the operation entailed division of ribs one to eleven inclusive. It has been realized more and more in recent years that this excessive resection is not always necessary, and that excision of parts of the first eight, seven, or even six ribs may produce a sufficient collapse to lead to the healing of the tuberculous lesion in upper lobe cases. When discussing apicectomy it will be suggested that a thoracoplasty limited to the first four or five ribs only is as efficacious, and is an operation preferable to apicectomy.

The collapse of an extensive thoracoplasty, and more so of a partial one, is not so complete as is the collapse of an uncomplicated pneumothorax, but it has the great advantage of being constant. Provided, therefore, the collapse by thoracoplasty is sufficient to obliterate existing cavities, it can be equally as efficient as the more complete collapse of pneumothorax.

It should be clearly understood that even the most complete thoracoplastic operation produces no crippling deformity. When the patient is clothed it is impossible to know on which side the operation has been performed. There is no drop of the shoulder, nor is there any limitation of the movements of the arms. When stripped, the alteration in the angle of the axilla is the main indication from the front; there is flattening behind, and the scapula is bedded-in so that the posterior surface is below the level of the spinous processes.

The indications for thoracoplasty are in the main those for artificial pneumothorax, but the contraindications are much more severe. With one exception a thoracoplasty should not be done until artificial pneumothorax has been tried and has proved valueless.

It is sometimes easy to decide that the amount of gas which it has been possible to get into the pleural cavity is insufficient to be of any value; but occasionally it is very difficult to determine when an artificial pneumothorax which is producing a partial collapse and is exercising a slight control on the symptoms should be given up. In such cases the benefit of the doubt may be given to a more prolonged trial of the pneumothorax in the exudative type of case without cavity formation or much secondary bronchial change. Where there is extensive fibrosis, however, with dilatation of the bronchi and bronchioles, or cavities, it is probably a waste of valuable time to continue with the pneumothorax; this should be abandoned in favour of a thoracoplasty.

Occasionally, when the base is well collapsed by artificial pneumothorax, but not the apex, the basal pneumothorax is supplemented by a thoracoplasty of the upper ribs only. When the pneumothorax is done primarily for the control of haemorrhage and is not efficacious, or if a lung, once collapsed, is re-expanding despite pneumothorax or oleothorax, then a thoracoplasty is indicated.

A pyothorax complicating a pneumothorax is always a cause for anxiety; if this pyothorax is secondarily infected it becomes a serious menace to life. It may be possible to control the effusion and the pyrexia by an oleothorax, but, if this fails, a thoracoplasty must be done. The major operation should not be too long deferred, since the pleura,

becoming thickened and rigid, will interfere with the collapse of the chest wall, and the thoracoplastic operation will also be a failure.

There is one type of case for which I consider that thoracoplasty should always be preferred. I refer to these cases of advanced chronic unilateral fibrotic tuberculosis in which there are extensive changes, part cavitation, part great thickening and dilatation of the bronchi and bronchioles. For these cases artificial pneumothorax is only rarely feasible; even where it is, the principal advantage is the building up of the patient's general condition, if necessary, as a preliminary to, and control of the sputum during, the major operation.

The contraindications for thoracoplasty are much more precise than they are for artificial pneumothorax. In the latter the presence of active disease in the other lung is, in itself, no bar; it is rather the anatomical extent of the disease that is the deciding factor. When considering the suitability of a case for thoracoplasty the presence of an active tuberculous lesion in the opposite lung must be regarded as a complete contraindication—with one exception, to be noted directly. It is sometimes difficult, however, to determine whether fine crepitations heard at the apex of the better lung, in which there is a certain amount of fibrosis, are due to active tuberculous disease or to a catarrhal condition. If these signs have been watched over a period of some months and show little variation in area or quality, and the indications in the more seriously diseased lung are very strong, and if, further, after a preliminary phrenic evulsion these crepitations are not deleteriously altered, then a thoracoplasty need not be considered as contraindicated.

The single exception referred to above is in the case of haemorrhage. Bleeding from the more damaged lung, when it is periodically recurring and is of sufficient quantity to endanger the patient's life, and when the fear of it has become an ever-present terror, justifies certain additional risks. In such cases it is permissible, in my opinion, to attempt to control the bleeding by thoracoplasty, when no other means have been found practicable, even if there is a small area of activity in the less involved lung.

Tuberculous enteritis seems to me to be an absolute contraindication. Cachexia, a failing heart, albuminuria, unless transitory, and diabetes, unless well controlled, are also contraindications. Tuberculosis of the larynx is not necessarily a bar unless there is considerable oedema interfering with the airway, or involvement of the epiglottis interfering with nutrition.

Multiple intercostal neurectomy has been tried by J. Alexander in certain cases in which there are contraindications to thoracoplasty. The nerves are resected under local anaesthesia. Slight collapse of the chest wall and immobility are obtained. There has been apparent benefit in some cases, but the number so treated is too small as yet for any definite conclusions to be drawn.

Extrapleural pneumolysis is the local collapse of the lung, which is secured by separating the parietal pleural membrane from the chest wall and by filling the space thus formed with a foreign body to prevent its return. This operation, which may be performed as an independent one, is generally used to induce collapse of the apex of the lung, and it is then called apicolysis. It is often supplemented by hemidiaphragmatic paralysis, and may be used to increase the collapse obtained by a thoracoplasty, especially in cases with thick-walled upper lobe cavities.

Various substances have been employed to fill the cavity between the parietal pleura and the chest wall, so that the collapse of the underlying lung may be maintained. Paraffin, however, may produce an extrapleural effusion, which, finding its way through the divided tissues, escapes and carries the paraffin with it. Fat and fatty tumours may degenerate and form an abscess. I have found a pedunculated graft of the pectoralis major in man, or the breast in woman, the most satisfactory.

As already mentioned, however, when an apical collapse is needed, a very extensive resection of the upper four or five ribs gives a more certain and satisfactory result.

It has been possible to touch the fringe only of the subject in this article. No reference has been made to technique or to the rarer operations.

EFFECTS OF AIR IN THE VASCULAR SYSTEM AND THE MECHANISM OF DEATH.*

BY

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THOUGH some surgeons profess to entertain no fears concerning the evil results of the introduction of air into a vein, most operators believe that such an accident is one to be carefully avoided. Experimental work on the subject was pretty extensive before modern apparatus like the electro-cardiograph could be used for interpretation, but not in recent times. The conclusions arrived at by experiment, though presenting general agreement with regard to salient effects, show certain discrepancies. The literature on the subject is not very extensive. Laborde and Muron¹ injected large quantities of air (500 c.cm. and more) into the jugular vein of the dog at a slow rate without fatal results, whereas rapid introduction of 100 to 200 c.cm. caused paralysis of the heart by distension. Couty² described most of the symptoms observed in the present experiments. The lack of an electro-cardiograph in his and other researches did not permit correct observation and interpretation of the cardiac phenomena. Begouin³ allowed air into the jugular vein in cats and dogs; he also blew it into the vein by the mouth. Withdrawal of air from the ventricle by means of an aspirator was practised with apparent benefit. Depage⁴ says that the entrance of air into veins is not so dangerous as is generally believed. He cites two cases of its occurrence during operation which were cured by artificial respiration, and quotes instances from the work of other surgeons. The three main theories of the mechanism of death—the nervous theory (Morgagni, Bichat), the pulmonary theory (Poisëuille, Bernard, et al.), and the cardiac theory (Magendie, Amussat, et al.)—are discussed. Jung and Auger, in a fairly recent paper,⁵ sum up the general views concerning the mechanism of death, the chief factors being rapid fall of arterial pressure, the entrance of air into the coronary vessels, and ventricular fibrillation. There is no reference to any influence of the respiratory mechanism, except in so far as disturbance of function may be implied as a result of the fall of blood pressure, and in no work up to the present account apparently is there a reference to the use of the electro-cardiograph. Little evidence is provided by the experiments done by myself to favour the view that ventricular fibrillation is a common cause of death after injection of air. The results of these experiments on dogs weighing 10 to 15 kilos are here considered in two chief categories.

A. Thorax Closed.

If 50 c.cm. of air or more be pretty rapidly admitted into the jugular or saphenous vein of a dog with closed thorax and natural respiration a fairly definite train of symptoms is produced—namely, respiratory distress, muscular agitation, polypnoea, marked fall of arterial pressure, and irregular action of the heart. The heart sounds are sometimes audible at a distance from the animal, and have a gurgling character. The electro-cardiogram may exhibit arrhythmia, extrasystoles, inversion of the T wave, etc. A dog thus treated does not necessarily die as a result; in fact, it usually recovers quite fully in the course of twelve to twenty-four hours. If a second large quantity of air be introduced before the effects of the first have passed off the animal may succumb to its influence, and death is generally preceded by a period of apnoea.

In deep anaesthesia the symptoms may be somewhat different from the above. Especially in the case of two dogs under chloralose, there was no marked polypnoea, and apnoea set in early, the effects of one injection being fatal. These two experiments were performed at the Institut Marey, in conjunction with Mr. L. Bull, to whom I am greatly indebted for help and counsel in the taking and interpretation of electro-cardiograms.

The most constant symptom, observed in practically all cases, and varying in degree with the amount of air admitted, is the fall of arterial pressure. When large

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