to an excessive secretion of the intrinsic factor by the stomach, or to hypersusceptibility of the marrow to it, has been mooted by Morris.⁵ Whether phenylhydrazine might have some inhibitory or destructive effect on the intrinsic factor, or on the cells producing it, has not been suggested.

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SOME THEORETICAL ASPECTS OF COLLAPSE THERAPY

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A recent discussion on the results of collapse therapy in pulmonary tuberculosis was remarkable for the lack of attention which was paid to the exact consequences following on the various forms of collapse therapy, when considered in terms of anatomy and pathology. Collapse therapy brings about, primarily, mechanical and anatomical changes, and it is only when these changes react favourably for the individual that there is therapeutic benefit. At present our knowledge of the immunological processes which determine success or failure in the treatment of pulmonary tuberculosis is very slight, and hence a mechanical change may occasionally end in an unexpected result. The following case is a good example.

A man, aged 40, with diabetes and pulmonary tuberculosis in the upper half of the left lung, was admitted to the Brompton Hospital with a view to artificial pneumothorax treatment. While in Brompton the insulin requirement increased, and fresh disease appeared in the other lung, which spread rapidly, in spite of complete rest and large doses of insulin. After the patient had been going rapidly downhill for about six weeks he was allowed to go home, as I thought to die. Six months later he appeared in the out-patient department looking very well. His history and x-ray examination showed that he had had a spontaneous pneumothorax on the left side, with the result that the pulmonary tuberculosis became quiescent, and was so when I last saw him three years afterwards.

Although unforeseen results may occur, it is obvious that the clearer the idea the user of collapse therapy has of the exact anatomical changes he wishes to induce in the lung and of the effects which will follow these changes, the greater the benefit that is likely to accrue. The considerations put forward here are treated from a theoretical point of view, and cases are quoted merely as examples to illustrate the points made. The former may, however, throw some light on the fact that, while collapse therapy is universally considered to be beneficial, the details of its use are the subject of considerable controversy.

EFFECTS OF COMPLETE PULMONARY COLLAPSE

Given a free pleura and a lung without gross fibrosis, low-pressure artificial pneumothorax will produce complete collapse of the lung. The changes that occur cannot yet be said to be fully understood, but the following may be considered as proved.

1. The pulmonary circulation will be greatly reduced and probably cease, though the bronchial circulation will persist.
2. The lymphatic drainage will be markedly slowed.

3. The bronchial movement and internal movement of the lung tissue is greatly diminished and frequently stopped.

4. There is a relaxation of tension on the lung tissue.

PARALYSIS OF THE DIAPHRAGM

Under similar conditions diaphragm paralysis by evulsing or cutting the phrenic nerve cannot be expected to have the same consequences. The pulmonary circulation and lymphatic drainage will probably not be affected; movement of the lung will be only slightly diminished, and this may be at the apex if the "piston" theory is correct. Relaxation of tension will take place owing to the diminution in size of the thoracic cavity, and this will not occur only at the base. In the operation of lobectomy a comparatively large space is left with a pressure which rapidly becomes negative, but within twenty-four hours the space is obliterated by the lung: this is done by the rest of the lung expanding and "flowing" into it. Similarly, if there is any source of tension in the upper part of the lung, the rise of the diaphragm will allow the lung to "flow" upwards, till a pressure equilibrium is obtained. The importance of the relaxation of tension on damaged tissue is very great. Nature's method of dealing with established tuberculous disease may be said to be: to localize it, to get rid of local organisms and the dead tissue formed, and to repair by fibrous tissue. Repair by fibrosis can only take place with difficulty in a rigid structure or in one under tension, and it is in this last stage of natural healing that the relaxation of tension of damaged lung is so important. If, then, the mechanical sequels of diaphragm paralysis fall so far short of those of a complete artificial pneumothorax, the therapeutic effects are not likely to be the same, but not for this reason necessarily useless.

A complete artificial pneumothorax will normally check any toxæmia from pulmonary tuberculosis in the collapsed lung in a fairly short time, and unless some accident supervenes the absence of toxæmia will persist indefinitely, even if the patient is doing moderate physical work. The rapid reduction of toxæmia is probably mainly the outcome of the interference with the pulmonary circulation and lymphatic drainage, and not so much of the reduction in movement, as is sometimes thought. In favour of this are:

1. If the lung is collapsed rapidly it is not at all uncommon to find a dramatic reduction of toxæmia in such a short time that it is difficult to understand how it could come about by lack of movement.

2. In cases in which there is much toxæmia selective collapse, even if anatomically successful, is frequently not so therapeutically.

The slowing of the lymphatic drainage may possibly be of therapeutic importance from quite a different point of view if Wingfield's new theory of the causes of relapse is correct. If the drainage is slowed the bronchial glands will have less tubercle bacilli drained into them and hence be given a greater opportunity of settling down and a diminished probability of discharging tubercle bacilli into the blood stream. Aided by the absence of toxæmia the relaxation of tension will help the repair of destroyed lung tissue and probably lead to the formation of a healed scar.

It cannot be expected that such a result will be obtained by paralysis of the diaphragm, as its mechanical effects will not be such as to cause the disappearance of toxæmia. If, however, the diaphragm is paralysed in a case with little or no toxæmia in which the lesion is trying to heal by fibrosis and contraction, the immobile diaphragm will mechanically assist, and possibly turn the scales in favour of sound healing.

A fresh factor, however, is introduced if the patient is put on strict bed rest after phrenic evulsion. This introduces a small proportion of the results (1), (2), and (3) of the artificial pneumothorax, and therapeutically the issue will be similar to that of a complete artificial pneumothorax, though it will be less rapid and efficient. A diaphragm paralysis, then, is unlikely to do away with toxæmia completely if it is uncontrolled by bed rest. In some cases, however, in which toxæmia can be controlled only by rest in bed a diaphragm paralysis may be able, by its relaxation of tension, to promote healing of the lesion while the bed rest holds the toxæmia at bay, with the result that a permanent reduction or abolition of toxæmia results.

A girl, aged 21, was unsuccessfully treated for two months by bed rest for a mid-zone cavity and infiltration. Although the toxæmia was reduced it was not abolished, and *x*-ray examination showed no improvement. An artificial pneumothorax failed. A phrenic evulsion was performed, and after about a fortnight slight but steady improvement took place, with, finally, complete quiescence and disappearance of the cavity and of tubercle bacilli from the sputum. The diaphragm rise was only 2½ inches after six months, which tends to confirm the presence of adhesions, though they did not apparently interfere with the therapeutic effect.

PLEURAL ADHESIONS

So far I have considered only those cases in which there is a complete freedom from adhesions. The influence of adhesions on cases treated by artificial pneumothorax is well known, ranging from complete frustration of its establishment to no effect in some cases in which the adhesions are thread-like and stretchable. Provided, however, that an artificial pneumothorax of considerable size can be obtained, the question that matters is whether the adhesion is of a type and so situated anatomically as to render impossible the result for which the artificial pneumothorax was induced. For example, in a lung with a dry apical cavity held by adhesions and a spreading toxic lesion in the mid-zone an artificial pneumothorax, complete except at the apex, might have a successful issue for the latter lesion, the adhesions at the apex in no way preventing the object for which the artificial pneumothorax was done. A clear idea, therefore, of just what is to be expected from an artificial pneumothorax will enable us to decide whether or not it has been successful. If not, we can then consider what further steps, such as adhesion cutting, should be taken. This is not a mere debating point, as every physician with experience of this work will have seen many adhesions cut unnecessarily. Speaking generally, the adhesion which calls least for cutting is the easiest to cut.

The question of the exact consequences of pleural adhesions on the results of diaphragm paralysis has not been extensively investigated, as only a pneumothorax or direct vision can demonstrate them. The only work of which I am aware which really has any bearing on this matter is the improvement in collapse brought about by paralysis of the diaphragm subsequent to the induction of a pneumothorax. It seems quite clear that in certain cases in which a pneumothorax is ineffective the phrenic evulsion allows the lung to shrink towards the adhesion, thus relaxing the tension on a cavity or diseased area. The unproved effect of adhesions largely invalidates all work in which estimates of the upward movement of the various parts of the lung are made by calculating the *x*-ray position of calcified spots before and after diaphragm paralysis. The only general conclusion that can be drawn is that the figures represent the minimum movement in a case with a non-adherent pleura and not extensively fibrosed lung. Again, in most papers the question of the length of time after the paralysis is not taken into consideration.

The part played by adhesions is possibly the cause of the dispute as to the value of diaphragm paralysis, as its detractors probably only use it when an artificial pneumothorax has failed because of adhesions, whereas its champions frequently employ it as a first choice, and thus do so in many cases in which the adhesions are absent or few in number. Taking the extreme case in which there is a complete synthesis between the parietal and visceral pleura, it is evident that no real benefit can be expected. At best, phrenic evulsion can only relieve tensions upwards and then to a slight degree at the extreme base, and can have no effect laterally, because of the thoracic adhesions. If individual adhesions only are present, the result of diaphragm paralysis will depend on their position in the pleural cavity and also on their relation to the lesion it is hoped to control. An adhesion between the lung and the side of the thoracic bony cage will exert a greater influence than a similar adhesion between the lung and the back or front of the cage at the same level, as a larger part of the lung will be free to retract upwards.

The main point, however, which will decide the issue of diaphragm paralysis is the level on the chest wall at which the adhesions are present. If there are adhesions in the diaphragmatic sulcus, the outcome will be similar to that of completely adherent pleurae. If, however, the adhesion is higher the diaphragm paralysis will have an effect on the lung at least up to the level of the adhesion, and probably higher if the adhesions are not very dense—for example, if the paralysed diaphragm is not prevented from affecting a mid-zone lesion by apical adhesions. Again, if a cavity in the upper part of the lung has an adhesion vertically to the apex, there is no reason to suppose that it will prevent a diaphragm paralysis from allowing retraction; but if the adhesion is to the lateral wall considerable interference may take place. Medial adhesions which are not much below the lesion do not seem to do a great deal of harm. The following case will illustrate the point.

A man, aged 28, had an apical cavity about the size of a golf ball, which had been present (without toxic effect) for two years following sanatorium treatment. The upper mediastinum was deflected towards the cavity. Later the cavity began to enlarge and a haemoptysis occurred. A diaphragm paralysis was performed, and within three months the cavity had closed and the scar was nearly hidden by the mediastinum. The tubercle bacilli previously constantly present in the sputum disappeared.

It would seem, then, that on theoretical grounds a diaphragm paralysis should be useful in any case, including apical lesions, in which a relaxation of tension is required, provided that adhesions do not prevent it. Unfortunately these propositions, put forward on theoretical grounds, are difficult to prove, because they require that an artificial pneumothorax should subsequently be done to show where the adhesions are; but such evidence as exists is in their favour, and my own experience has not revealed cases which disprove it.

Rib resection methods of collapse have not been dealt with: their use depends so much on the technical skill of the operator and exact amount of and position of the ribs removed and their relation to the lesions.

It seems clear, however, that knowledge of the exact effects of collapse therapy is very incomplete, and until further information is at our disposal we shall employ it, to some extent, empirically. The procedures are primarily mechanical and anatomical, and lack of accurate knowledge in these subjects will always lead to inefficiency in results. This does not mean that the knowledge at our disposal should be neglected; and treatment based on a hypothesis which fits in with known facts, even if it is incorrect, is preferable to a "hit or miss" method.