THE PLACE OF ARTIFICIAL PNEUMOTHORAX IN THE TREATMENT OF PULMONARY TUBERCULOSIS*

By Philip Ellman, M.D., F.R.C.P.

Physician to the North-East Metropolitan Hospital Board at the East Ham Chest Clinic, Hart's Sanatorium and Mile End Hospital

In view of the increasing advances in our knowledge of pulmonary tuberculosis it is indeed timely that we should, while none the less maintaining a progressive outlook, 'go wisely and slow—they stumble that run faster.' In other words, we should now pause to assess the situation in so far as the position of artificial pneumothorax in the sphere of collapse therapy is concerned, and ask ourselves whether, in the light of increasing experience, the early enthusiasm for this particular measure in the management of pulmonary tuberculosis from the active stage to complete quiescence, has been justified. At the outset we must recognize that, no matter what form of treatment is contemplated, the general constitutional nature of the disease must never be forgotten, nor the possible influence in an individual of an inherited diathesis affecting the local lesion. Special measures must be regarded primarily as adjuncts to the basic method of treatment by bed rest and the sanatorium régime, for which indeed there is no real substitute.

Artificial pneumothorax as we know it today, discriminatingly used with adequate radiological control, has certainly revolutionized the management of certain cases of pulmonary tuberculosis, particularly in regard to mechanical factors confined to the local lung lesion. While recognizing this I intend, for purposes of this discussion, to be somewhat provocative and to speak of the limitations, difficulties and disappointments in relation to pneumothorax therapy often encountered in clinical practice. More than 20 years ago when I first plunged into the maelström of this disease I felt that the subject, whilst complicated, was not incapable of solution. Today as a result of ever-growing knowledge and experience of the combined resources of collapse and other forms of therapy, over and above bed rest and the sanatorium régime, I have become ever more conscious of the greater need for clinical judgment. A critical attitude is demanded not only for the immediate but also for the end results of the more active forms of therapy available. It is, moreover, of over-riding importance that we should not divorce the social from the clinical aspects of the disease and that the recent unfortunate tendency in this direction should be checked.

I venture to say that one becomes increasingly conscious of being a victim of the old adage that one knows less and less of more and more. We now know, for example, that the ultimate fate of the tuberculous lung lesion, with or without cavitation, is closely allied to the state of the bronchi; the incidence of tracheo-bronchial disease, while perhaps not as high in this country as American literature suggests, is certainly not inconsiderable. As Rafferty, Houghton, Sellors and others have emphasized, bronchial drainage would appear to be an important factor in the healing process. Formerly we thought we could recommend with confident certainty the induction of an artificial pneumothorax, but, looking at some of the more disappointing results in the light of modern knowledge, we must confess to some disillusionment. There can be no doubt that the procedure attracts by reason of its simplicity and reversibility and in the limited number of cases where it is effective in producing a perfect concentric anatomical collapse, with or without the assistance of early pneumonolysis, in fact when it succeeds in relaxing without compressing the diseased area, then indeed the sun shines on physician and patient alike. But experience shows how much the results depend on the anatomical character of the collapse. In this connection Hjaltested and Törning's (1939) classification of collapse into four groups is appropriate and their recorded late results of 191 cases of completed treatment whose artificial pneumothorax was commenced between 1925-1931 is relevant.

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My colleagues and I at Hart’s Sanatorium, the East Ham Chest Clinic, and elsewhere have found, in corroboration of Hjaltested and Törnig’s observations, that, providing the initial indications are clearly defined, lasting results correspond to the quality of the pneumothorax; that is, the poorer the initial pneumothorax the more unfavourable the artificial pneumothorax life and the permanent result. A pneumothorax free from adhesions produces fewer complications and infinitely better end results with higher survival rates, Bentley (1936), Hjaltested and Törnig (1939), Jennings, Matill and Nemec (1940), Berg (1941), Rafferty (1944), Simmonds and Martin (1948). If adhesions are present, even though cavitation is absent and the sputum negative, they should, speaking generally, be divided if possible. An incomplete pneumothorax should be abandoned in favour of some other therapeutic measure.

The indications for artificial pneumothorax are most evident in cases of unilateral exudative disease (with or without cavitation), which is not controlled by strict bed rest, although there is a place for bilateral pneumothorax. I must here emphasis that we now hold the view that a patient with an acute febrile illness with active exudative disease, especially of the pneumonic and atelectatic type, is best treated initially by an adequate period of complete bed rest. The lapse of time thus secured presents an opportunity for the in-
flamatory process to subside, thereby reducing the possibility of pleural complications. It may, in fact, even obviate the need for collapse therapy. In this connection the use of streptomycin may in the future make pneumothorax therapy a safer procedure by assisting the active inflammatory exudative lesion to subside and thus neutralizing toxicity. I am of the opinion that pleural complications are often due to what Houghton and Holmes Sellors aptly call a ‘crash’ pneumothorax, induced while the parenchymal lesion is too active. The additional aid to bed rest afforded by another form of collapse therapy, namely pneumoperitoneum, has in our experience, much in its favour. It has the advantage over pneumothorax that it can nearly always be initiated, it is associated with strikingly few complications, it can be discontinued at any time without the occurrence of a non-expansile lung or of any pleural complications, and it can, if need arise, be readily re-introduced.

In a follow up of several hundred cases of recent tuberculosis Amberson (1937, 1942) with a very wide experience, especially among nurses, states that some 90 per cent. promptly treated with bed rest responded well and that in the remaining 10 per cent. relapse or spread of the disease was detected and usually controlled by pneumothorax. This conservative mode of treatment, controlled by serial skiagrams, is, in my view, sound and may be compared with the initial conservative treatment for peptic ulcer. The risk of adhesions during the period of observation would seem to be more theoretical than real; the ‘crash’ pneumothorax with the inevitable ‘black lobes,’ particularly characteristic of pneumonic and atelectatic lesions, induced within a few days of diagnosis is ill-advised and potentially dangerous. Moreover, in the presence of bronchial tuberculosis there is even more need for critical evaluation of the treatment if mechanical changes such as broncho-stenosis and atelectasis are to be avoided. An ineffectual pneumothorax may, by reason of the symptomatic relief afforded, give rise to a false sense of security. Patients should not be treated, as I have often told my students, as ‘petrol-filling stations.’ The physician’s needle can be responsible for complications more dangerous than the surgeon’s knife, in fact, I have sometimes felt that it would be to the advantage of the patient if an artificial pneumothorax required as much skill as a thoracoplasty. The immediate abandonment of every pneumothorax that is recognized to be ineffectual must be faced unhesitatingly and the induction of a pneumoperitoneum or a primary thoracoplasty must be considered and discussed as a preferable alternative. There are many cases in which primary thoracoplasty as an initial procedure is to be preferred to an ineffectual attempt to induce pneumothorax, for it is certainly not easy before the induction to predict whether an artificial pneumothorax is likely or not to be satisfactory. We must ask ourselves whether the complications of pneumothorax have not arisen through failure to recognize this principle. This fact cannot be over-emphasized if diseased tissue is to be controlled, cavity closure maintained, sputum conversion achieved, and such complications as contralateral spread, pleural infection, and bronchopleural fistula prevented.

Having referred to bed-rest and the sanatorium régime I must again emphasize the fact that natural resolution is well established as a form of healing. Jaquered and I, as long ago as 1927, dealt with this subject in relation to tuberculous pneumonia and broncho-pneumonia. I reiterated this plea for initial conservative treatment of exudative tuberculosis by strict bed rest in a paper read before the British Tuberculosis Association in 1939, describing how some such cases occurring in the first three or four decades of life showed this peculiar tendency to heal by natural resolution even in the presence of cavitation, and I would agree with Houghton that under present conditions it would be preferable that the waiting list of our sanatoria should continue, provided the patient adheres to strict bed rest at home, rather than that ‘crash’ pneumothorax and its dangerous sequelae should be adopted in order to relieve the pressure on beds.

Mechanics of cavity closure. On the need for closure of cavities in the successful control of pulmonary tuberculosis there is absolute unanimity, but on its mechanism there are opposing schools of thought. I do not propose to go into the mechanics of excavation but I must confess that until recently I shared the common view that bronchial occlusion was the chief factor in cavity closure. In view, however, of their bronchographic and bronchoscopic studies, as well as the effect of bronchoscopic aspirations, CO₂ inhalations, and streptomycin, Rafferty, Houghton and Sellors have convinced me that there is much to be said for the view that adequate bronchial drainage and patency of the bronchus, as in the case of pulmonary abscess, is often a likely method of healing with obliteration of the cavity. The common ‘tension’ or positive pressure cavity with its broncho-cavity check-valve allowing air to pass into the cavity during inspiration, when the bronchus becomes elongated and widened, and trapping it during expiration, when the bronchus becomes shortened and narrowed, is of considerable clinical significance. Such cavities are often resistant to treatment.

Rafferty insists on the importance of adequate bronchial drainage in relation to collapse therapy. In pneumothorax the collapse is concentric and
affects the major bronchi as well as the lung parenchyma whereas in thoracoplasty the collapse is initiated peripherally and proceeds towards the hilum. Most clinicians are, therefore, of the opinion that definite bronchial tuberculosis is a contra-indication to artificial pneumothorax, and is usually an indication for thoracoplasty (unless there is complete bronchostenosis, when the question of lung resection will have to be considered). In this connection I call to mind a patient, P.B., age 32, who three years ago had been advised an immediate thoracoplasty for a left upper lobe fibro-cavernous lesion. Unfortunately he refused this in favour of a temporising phrenic crush. Quite recently he had an haemoptysis and although he had agreed to a thoracoplasty he had by this time developed a complete stenosis of his left lower lobe bronchus, confirmed by bronchoscopy, with collapse of the lobe. He has had to undergo a pneumonectomy followed by a routine thoracoplasty. The possible adverse effects of a mechanical rise in the diaphragm, in the presence of bronchial disease, in promoting bronchial occlusion and collapse, as with artificial pneumothorax, is noteworthy. Morriston Davies says significantly, 'I am now as a routine bronchoscoping all my patients prior to a thoracoplasty and also those cases treated by pneumothorax which show localized collapse.' There can be no doubt that the wider use of the bronchoscope might avoid many complications, for few would contest the assertion that the development of bronchial occlusion with collapse is an unfavourable sequence in pulmonary tuberculosis.

Tracheo-bronchial disease contra-indicates pneumothorax therapy and favours primary thoracoplasty in so far as the latter encourages bronchial patency and drainage. It may even necessitate lung resection. Moreover, the effect of streptomycin in tracheo-bronchial disease suggests that the maintenance of good bronchial drainage with the corresponding clearing of collapsed areas and sometimes even of cavities would appear to favour the patent bronchus theory. The therapeutic trials of streptomycin by the Medical Research Council in acute bilateral exudative disease as a preliminary to collapse therapy later, would seem to support this point of view. We have already referred to the dangers of massive collapse in pneumothorax therapy. The concertina effect of which Burrell so strongly disapproved in relation to successful pneumothorax therapy may, as Houghton suggests, be justified in the case of 'black lobes' in which cavity closure has not been achieved. He believes that bronchial drainage can sometimes be obtained in such cases by successive expansions and relaxations of the lung with postural drainage with resulting re-aeration of collapsed areas and satisfactory closure of persistent cavities, although this is admittedly a hit or miss procedure. We have all of us seen a dangerous tension cavity in a perfectly collapsed lung and we are all familiar with the pneumothorax which may show gross cavitation, although prior to the collapse it had hardly been suspected. There can be no doubt that the pneumothorax life is often fraught with danger if it is used for (1) large apical tension cavities, (2) large thin-walled cavities lying at the lung periphery, (3) hilar cavities and (4) cavities situated at the apex of the lower lobe, as well as in extensive disease with multiple cavities and finally (5) in fibro-cavernous disease. These are the cases which are liable to give rise to pleural complications.

Effusions complicating artificial pneumothorax. The most serious complication of pneumothorax therapy is clearly tuberculous empyema. Transient and small effusions with a small pool of fluid sufficient to obliterate the costo-phrenic angle or even sometimes reaching the dome of the diaphragm have been a common experience in our cases. As a general rule we have found them benign and clinically insignificant. These effusions do not usually contain tubercle bacilli and almost invariably clear up spontaneously or with the aid of gas replacement with careful check on the intra-pleural pressures. In the case of an acute effusion with a high cell-content and containing tubercle bacilli, and where there is pyrexia, then the position is ominous. We regard such cases as potential empyemata. Reaction of this kind is much more likely with the unsatisfactory type of pneumothorax already mentioned. My colleagues and I are in the habit of aspirating such effusions, following up with lavages of antiseptics such as normal saline, Dakin's solution, methylene blue, and more recently the intra-pleural use of P.A.S. azochloramide, etc. This procedure has met with varying degrees of success. We have found that the early institution, if the patient's condition permits, of an air-tight drain and a thoracoplasty operation is more often than not inevitable.

Oleothorax is used by some clinicians when aspiration and irrigation have failed to clear the infected pleura, but it is apparently only valuable where the lung parenchymal disease is controlled and where thoracoplasty is contra-indicated. Morriston Davies favours oleothorax where the pneumothorax is satisfactory, and where air refills are for various reasons difficult to continue.

Mixed infection empyema and broncho-pleural fistula are intimately connected, as Coryllos has maintained. The former can, with the aid of penicillin, sometimes be converted to a pure tuberculous exudate. My own experience has
Plate 1.—From a case of active pulmonary tuberculosis with positive sputum, showing infiltration of the upper and middle zones of the right lung, with a large cavity (with a fluid level) in the upper zone.

Plate 2.—From the same case as Plate 1, after 20 months of complete bed rest, showing complete resolution of the lesion and disappearance of the cavity.

Plate 3.—From a girl aged 21, with active pulmonary tuberculosis, with positive sputum, showing extensive infiltration of the right lung with commencing excavation in the upper zone. This patient showed no response to bed rest.

Plate 4.—From the same case as Plate 3, three years later, after induction of a right artificial pneumothorax, showing perfect concentric anatomical collapse.
PLATE 5.—From a case of active pulmonary tuberculosis, with strongly positive sputum, showing extensive infiltration with excavation of the right middle zone.

PLATE 6.—From the same case as Plate 5. No response having been observed after two months of complete bed rest, a right artificial pneumothorax was induced. This shows the state of affairs a month after the initial induction; the cavity is uncollapsed and is, in fact, much more obvious than in Plate 5.

PLATE 7.—From the same case, a month later, after pneumonolysis. The cavity is now closed and concentric anatomical collapse of the right lung is shown. This was kept up for the ensuing five years.

PLATE 8.—From the same case five years later than Plate 7. The right lung has now completely re-expanded, and healing of the original lesions has been achieved.
PLATE 9.—From a case of active pulmonary tuberculosis, with positive sputum. A 'crash pneumothorax' was induced shortly after admission.

PLATE 10.—From the same case as Plate 9, after pneumothorax therapy, showing massive collapse of the right upper lobe ('black lobe').

PLATE 11.—From a case of active pulmonary tuberculosis with positive sputum, in a girl of 21. A small cavity was seen near the apex of the left lower lobe.

PLATE 12.—From the same case as Plate 11, after a left artificial pneumothorax, showing a 'black lung' with an early tuberculous empyema. The pneumothorax was eventually abandoned in favour of a complete thoracoplasty.
convinced me that these complications are less likely to occur where the ineffective pneumothorax is speedily abandoned in favour of safer forms of collapse therapy.

Obliterative pleurisy, wet or dry, has occurred in some of our cases and usually involves the abandonment of the pneumothorax.

Unexpanded lung. In certain cases associated with a thickened pleura when the pneumothorax has been discontinued lung re-expansion may prove difficult, resulting inevitably in an incompletely expanded lung. I recall two cases of this type who had their artificial pneumothorax induced 17 and 15 years ago and kept up for seven and five years respectively. Within the last 12 months, after the disease had been under control for several years, they had both suffered from a severe acute respiratory infection. The result was that the unexpanded lung lesion had broken down with cavity formation recurring in the old site necessitating in each case a thoracoplasty.

Mediastinal hernia has occurred in a few of my cases but no serious symptoms have ensued. One case showed an X-ray picture looking uncommonly like a pneumopericardium.

Gas embolism, closely allied to the so-called pleural shock, has fortunately proved a fairly rare complication.

Many complications arising in connection with pneumothorax therapy are undoubtedly due to the mechanical effects of superadded bronchial tuberculous lesions and to the maintenance of pneumothorax therapy in spite of contra-indication. In such cases the risk of pleural infection is grave. Space will not permit me to do more than refer to such rare complications as pneumopericardium associated with a congenital defect of the pericardium (Ellman and Hussain, 1948) or to the risks encountered with a self-administered pneumothorax, an example of which I have recently described (Ellman, 1948).

It is salutary to realize the complications, difficulties and disappointments that may arise in relation to pneumothorax therapy, since the history of therapeutics brings to light time and again the harm done to a valuable form of treatment by its indiscriminate use; witness the sulphonamides, penicillin and possibly even streptomycin. I feel I cannot do better than conclude by quoting a few remarks of Morriston Davies made in his address to the Thoracic Society last July:

‘By and large the results of artificial pneumothorax are disappointing but that is largely our fault. All these years we have been learning by the costly process of trial and error. Now we know that the results of a good collapse by artificial pneumothorax, combined often with an efficient division of adhesions, are mostly good. We know also that the results of contra-selective or ineffective collapse are bad, and further that artificial pneumothorax in some of these cases or when there is tracheo-bronchial tuberculosis or persistent surface of tension cavities is not only bad but often dangerous. One great difficulty is that there are still too many sanatoria or centres for which the facilities for major surgery are not yet available. In consequence there is in these places a reluctance to abandon an active procedure since no alternative is available. Let us hope that within another five years this unfortunate state of affairs will be remedied and that the necessity of the immediate abandonment of an unsatisfactory pneumothorax in favour of major surgery will be recognized and the means for this made available.’

It is pleasing to note this pioneer surgeon’s appreciation of the physician who ‘while wise in his knowledge of surgery, puts his patient to bed, studies his case from all angles, watches his response to a sanatorium régime and balances the pros and cons before putting up the case for ancillary treatment.’ While deploing the rush to ancillary treatment Morriston Davies finds his most pleasing successes in those patients whom he can send out healed by sanatorium treatment alone. I need hardly add that I most heartily endorse these sentiments.

Summary

1. Pulmonary tuberculosis is primarily a general constitutional disease, with an individual inherited diathesis which, together with the mechanical state of the bronchi, influences the response of the local lung lesion to treatment. Moreover, the clinical condition is closely linked to the social factor throughout the patient’s life.

2. Initially in the treatment of active pulmonary tuberculosis the effect of adequate and complete bed rest, with X-ray control, should always be given a trial. In some selected cases this can be assisted by a pneumoperitoneum or by streptomycin.

3. The value of pneumothorax therapy under controlled conditions is recognized; and effective pneumothorax, with or without aid of pneumonolysis, producing sputum conversion and cavity closure, achieves as a general rule satisfactory end results and survival rates. An ineffective pneumothorax should unhesitatingly be abandoned to avoid undesirable complications.

4. A ‘crash pneumothorax’ induced while the lung parenchymal lesion is too active is, speaking generally, to be condemned. Complications are especially likely to arise in the presence of acute pneumatic phthisis, bronchial tuberculosis and
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W.N.L.H.

VIRAL AND RICKETTSIAL INFECTIONS OF MAN


Written by a group of American experts under the editorship of the Director of the Rockefeller Institute, this excellently produced book is published for 'interested people not actively engaged in the study of viral and rickettsial infections.' It should be said at once that this is an outstandingly useful addition to medical literature and may well become a standard textbook. It deals with all aspects of these diseases but emphasis is placed throughout on describing the data, gained either clinically or by research, of the aetiological agent itself. The clinical side is by no means neglected but has been a secondary consideration.

There are two main divisions, The first, of some 150 pages, deals with general considerations; the second deals sectionally with each disease or disease-group. In the first part are found detailed descriptions of physical and chemical procedures and principles involved in virus laboratory work, electron microscopy (with many fine micro-photographs), serology, chick-embryo techniques and tissue-culture propagation of viruses. The many to whom much of this material is new will find this section thoroughly readable and full of instruction. The few who may be considered experts will find here an up-to-date precis of all modern virus work with ample references to original sources. It might be said that this section resorts to too exact a description of details in techniques which is unnecessary and fatigueing for those for whom the book is intended. This may occasionally be so as in the precise description of chick-embryo techniques, but it must be remembered that the essence of this portion is to give an
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