TREATMENT OF TUBERCULOUS PLEURAL EFFUSIONS AND EMPYEMATA*

BY

R. R. TRAIL, M.D., F.R.C.P.

PHYSICIAN TO THE ROYAL CHEST HOSPITAL

The adequate and justifiable treatment of tuberculous pleural effusions and empyemata can be undertaken only with a proper understanding of their pathogenesis, and of the changes in normal structure postulated by their presence and possible as their end-results.

Pathogenesis

Inflammation of the pleura, apart from the so-called idiopathic type, may occur in any stage of phthisis, and may be acute or chronic, with exudation either on to its surface only or into its cavity. In acute cases the membrane becomes soft and reddened, and a grey lymph exudes; should the process stop at this point fibrinous adhesion through the action of pleural cells takes place between the two surfaces, and a repetition of this process produces plastic pleurisy. Such a condition is a frequent post-mortem finding in cases with as well as without known clinical phthisis during life, and is commonest and most dense at the apices, just as in recent cases the most usual point for inflammatory changes on the membrane is posterior to the apices. Should the inflammation continue beyond such exudation of lymph a second stage supervenes, with outpouring of serum from the capillaries, leading to pleurisy with effusion, the fluid being yellow with a greenish tint, more or less pronounced, and with flakes or even coagulated masses of fibrin.

That a pleural effusion may be, therefore, the result of an infection of the lung periphery by contiguity and extension will be readily admitted, but battles are still raging in the medical publications of every country over so-called "idiopathic" pleurisy, some writers wishing to find every possible excuse for avoiding sanatorium treatment, others boldly advocating gas replacements and even surgical collapse in every case. Norris and Landis state that 80 per cent. are tuberculous; Fishberg says that in many cases it would appear to be tuberculous but many are rheumatic. Bowditch, in a study of ninety cases, found that thirty-two died of phthisis. Allard and Koster state that of 2,123 cases of phthisis they found a history of previous pleurisy in 30 per cent.; of all cases I admitted to Midhurst Sanatorium with definite pulmonary disease between 1924 and 1933, 26.6 per cent. had a history of pleurisy three to four years before admission.

Diagnostic Criteria

Such investigations show that the true percentage of tuberculous origin must be very high, for apart from the fact that many cases are never investigated or followed up, we recognize the curative value of a pleurisy on an underlying lung lesion. It can be taken as a safe rule that a pale yellow fluid which has a specific gravity over 1018, is alkaline, clots on standing, and has an excess of small lymphocytes is tuberculous. It is true that tubercle bacilli will seldom be found by the methods now in common use, in the early stages of the effusion, and that even massive injection into a guinea-pig may give negative results, but it has been shown that positive results can be got in far greater numbers than formerly by the culture method of Dr. Jensen of the Serum Institute of Copenhagen, which he developed on the techniques of Griffiths and Lowenstein, and which has been used and further improved in this country by the work of Dr. McCartney and Dr. Holmes.

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An interesting piece of research has been done by Tobiesen; he examined the stomach washings of 162 cases where no signs of an underlying lung lesion could be found at the time of the effusion, and compared his findings later with the x-ray signs after absorption. Of the 162, 45 per cent. had been positive; of forty-seven afterwards shown to have an x-ray lesion 47 per cent. had been positive; while of 115 without x-ray signs 39 per cent. had been positive. It seems only reasonable to conclude that idiopathic pleurisy means previously or presently active tuberculosis of the lung even though this is not discovered clinically, and that all cases should be treated as such at least until they are proved to be otherwise.

This belief need not lead us, however, to employ the drastic immediate treatment advocated by J. L. Lewis in a recent paper. He advises in all cases gas replacements, with enough air to separate the pleural surfaces but not materially collapse the lung, and frequent replacements until the fluid disappears. E. P. Smart claims that mortality has been reduced since he practised such treatment, but surely experience has proved such interference to be wholly unnecessary.

Fluid is Nature's splint, to be removed only where the patient is in cardiac or respiratory distress; where it is not absorbing in reasonable time, when often even a slight amount withdrawn will start the process; where there is active lung disease as shown by long-continued temperature, in which event air replacement for therapeutic pneumothorax is indicated; and where it is of such long standing that temporary replacement is necessary in order to watch the lung for active disease during re-expansion.

Value of Sanatorium Treatment

If the object of such treatment is to keep the patient until he has had sufficient rest absolutely quiet, and during that time to instil the principles of sanatorium regime, it might have some excuse, but even this is not sufficient reason for exposing him to the risk of superimposed infection. Rest till absorption, followed by sanatorium treatment, is undoubtedly the best treatment. A pleuro-pulmonary fibrosis is the excellent result in many with underlying lung lesions, and I believe such a secondary type of pleurisy can be diagnosed from the temperature reaction, for the fall is not by lysis, but by a series of peaks of temperature, each succeeding peak being lower than its predecessor. The value of sanatorium treatment has been proved by many observers. Helms followed the after-history of 150 cases: in two years the combined morbidity and mortality was only 7 per cent.; in eighty-four, followed for more than five years, it was 5 per cent. J. W. Gaarde reports that of sixty-eight who had at least four months' rest forty-three recovered completely, while of seventeen with at most two months' rest eleven had died. Between 1926 and 1933 I admitted sixty-one cases at Midhurst; of these to date only two have developed any further signs or symptoms of tuberculosis: both have died of tuberculous meningitis, without intermediate lung involvement, so that it is probable that both had actual pleural involvement from the time of onset or developed it later as a result of the effusion.

Such tuberculous infiltration of the membrane may occur in two ways: (a) by the blood or lymph stream—a primary infection; (b) by absorption of a pleural exudate containing tubercle bacilli from an infected lung—a secondary infection.

Bacilli transmitted through the blood or lymph stream may lodge in any part of the membrane, internal or external to its elastic layer, to form true tubercles, which may go through all the known stages of evolution up to calcification. The resulting exudate seldom becomes pro-

fuse, but a steadily extending plastic pleurisy may develop or a true tuberculous empyema supervene. In the former result I would place such cases as never show a demonstrable lung lesion by sputum test or *x*-ray findings, but who either go slowly downhill by progressive pleuropulmonary fibrosis or suddenly develop meningitis after apparently reaching a dormant condition following the pleurisy.

Secondary Tuberculous Empyema

Tuberculous infection of the pleura has long been recognized as a cause of chronicity in empyema, and while it has never been rare as a primary infection, as a secondary manifestation of phthisis it is much more common, especially since the introduction of artificial pneumothorax. In such an event there has been absorption of an exudate containing tubercle. Here the pathology is quite different: a tuberculous pleuritis develops, producing granulation tissue without true tubercles, limited to the pleura internal to the elastic layer. Profuse, clear, or blood-stained exudate is the usual result, and while withdrawal may lead to adhesion of the surfaces and a consequent dormant condition, it is very liable to recur, and to change in character until it becomes a true tuberculous empyema. Such a secondary empyema has a profound effect on the pleura, especially the parietal layer, which now develops true tubercles internal to the elastic layer and non-tuberculous fibrous tissue external to this layer up to one or more centimetres thick, or even calcareous plaques.

The possible danger of fluid complicating artificial pneumothorax is therefore evident. Its effect on the underlying lung in long-standing cases must also be taken into account, for by the formation of adhesions it may cause obliteration of the pleural space and consequent reactivity of the lesion. J. B. Nablant therefore advises that all effusions should be frequently aspirated, a rule of thumb which ignores the individual, who must be considered on (1) the character of his effusion; (2) the activity in the underlying lung; (3) his possible complications—for example, disease in the other lung.

The majority of effusions which occur where pneumothorax was induced prior to the stage of cavitation are tubercle-free. The thoracoscope has demonstrated that adhesions are present in far greater numbers than can be shown by the most careful *x*-ray control; and, while many of these strands give way under continued treatment, even with consistently low intrapleural pressures, without any but good effect, their rupture is the usual cause of fluid, as their injured surfaces are exposed to contaminated atmospheric air. Actual tuberculous infiltration of these strand-like adhesions is rare.

Many observers have noted the distinct benefit of fluid in cases previously not doing well; again, many have seen how, just as in idiopathic pleurisy, a small amount withdrawn for examination has hastened absorption. The danger of resulting adhesions in sufficient amount to interfere with the efficiency of the pneumothorax is often grossly exaggerated, and even when they do form they are generally basal and so over a part of the lung which is healthy; moreover, the re-expansion following slow obliteration may give an excellent result, while if it is desirable to continue the refills and the conditions are otherwise suitable, phrenic evulsion can relax these basal adhesions. It may be as wrong to continue replacements in a sero-purulent effusion as it would be to do a phrenic evulsion in a case with apical cavitation, and therefore very likely to absorb the effusion and attain arrest of the diseased area by an upper thoracoplasty.

Of my own pneumothorax results I have compared forty with fluid and forty without fluid; they are all end-results in so far as all had ceased refills and had been followed for from two to nine years from the date of induction. Of the forty without fluid thirty-three

were alive as against thirty-one with fluid; the corresponding numbers with negative or no sputum were twenty-six and twenty-five; the numbers fit for work, twenty-seven and twenty-four; and the numbers dead, seven and nine—a remarkably parallel result. In thirty-five of forty cases fluid had no apparent immediate ill effect; twenty of these carried on refills as usual after absorption with no evidence of interference by adhesions, and while six showed fresh disease later, in five it was in the opposite lung and in one in the larynx. This may be compared with the result in the fifteen who obliterated, of whom three showed rapid spread in the other lung—but two were already bilateral at the time of induction and the third had tuberculous laryngitis. Again, of the twenty who continued refills only two had replacements, while of the fifteen who obliterated six had this treatment.

I am not, therefore, convinced of the great dangers of an effusion which is tubercle-free and does not become sero-purulent; of the benefits of continued gas replacements, or of the invariable disadvantages of obliterative pneumothorax; and I would apply the same rules on observation and treatment to cases of spontaneous hydro-pneumothorax.

Primary Tuberculous Empyema

The same guiding principles outlined above ought to apply to the treatment of primary tuberculous empyema; there is no general rule. The patient's reaction may be so profound that rib resection is a necessity, and the resulting sinus only a secondary consideration. If such operation is imperative too low an incision is a mistake, as there is the possibility of a tract forming between the diaphragm and the chest wall, very difficult to drain, and therefore leaving the patient open to the danger of a residual active focus. Where it is intended to go on to thoracoplasty later it may be better to do a phrenic last, as contraction of the diaphragm may aid approximation of the cut ends of the seventh to tenth ribs as against the tendency of the rising abdominal viscera to push them apart.

In less severe conditions Lambert of New York claims good results from irrigation with 1 per cent. watery solution of gentian or crystalline violet. Simple aspiration may occasionally be quite successful, as it may lead to adhesion of the pleural surfaces, but on the whole the literature on end-results seems to support aspiration and air replacement—that is, the production of a pyo-pneumothorax, and then extrapleural thoracoplasty, as adhesion takes place provided accumulation is not allowed, and the operation wounds heal as they are in normal healthy tissue. Intermediately in cases with considerable pleural thickening it may be advisable to irrigate with Dakin's solution to attempt decortication, although it is usually impossible to forecast which lung will re-expand, as some show more tendency than others to a gross pleuropulmonary fibrosis. Operations of the Schede and Estlander type, which aim at obliteration by removing the pleura and intercostal muscles first, are usually unsuccessful. Further experience in thoracoplasty seems to offer the best chance of improving the prognosis in this grave condition, for whereas Archibald in an earlier series claims only five recoveries in nineteen cases, Hedblom, in a more recent series, can claim seventeen of twenty-three cases.

Secondary Pyogenic Infection

If pyogenic organisms infect such a tuberculous empyema the clinical picture is that of acute pyogenic empyema, masking in most the true tuberculous nature. Theoretically such infection could occur by the blood or lymph supplies, but in practice the organisms enter from without through some fault of technique in gas replace-

ment, or from within by rupture of the lung, which again is very liable to result from this treatment, when recurring fluid causes a high intrapleural pressure, and a pleuro-pulmonary fissure may be established. Streptococci, staphylococci, colon bacilli, and many forms of anaerobic organisms have been found. The greenish tint of tuberculous pus now disappears; the effusion is of a dirty yellow colour, is definitely thicker, and has an excess of polymorphs. The effect on the pleura is thickening, by increasing fibrosis of its external layer.

There is no evidence that the prognosis is any worse than that of uncomplicated tuberculous empyema: some observers say it is better; but the literature on treatment is full of contradictory advice, and leaves one with the impression that unlike conditions are being compared, and the individual patient lost sight of in the battle over the comparative advantages of conservative and radical measures.

Conservative Treatments

Conservative treatments range from aspiration, aspiration and air replacement, aspiration and irrigation with so-called antiseptics as saline, gentian violet, acriflavine, and Dakin's solution, up to replacements with gelatin and the various forms of oleothorax. Widely varying results have been reported, each method having its champions prepared with irrefutable arguments for its mechanical and disinfecting properties. Thus Rosenblatt quotes twenty-one cases treated by aspiration and replacement with a saturated alcoholic solution of methylene-blue, and all followed up for from one and a half to eleven years. Eleven survived, but the period necessary for them to become fever-free was five months to two years; in only four did he continue pneumothorax when the cavity was entirely free from effusion, and then re-expansion produced more retraction and mediastinal displacement than ordinary pneumothorax, which may be a not altogether unmixed blessing to the patient.

The same applies to oleothorax. Some writers are still claiming disinfecting properties for gomenol, although Gloyne showed in 1927 that it has no such value, and can act for good only by producing a thickened and therefore less permeable pleura, thus inhibiting or delaying the absorption of bacillary toxins. Bufalini quotes four cases to show its special value; Gullbring, fifteen which were not favourably influenced. Recently, Josevitch has made claims for the oil of cajuput, saying it is less irritant and toxic, while it dilutes the substances affecting the pleural surfaces, but he advocates it as a permanence only where radical treatment is impossible. This is in keeping with the conclusions to be gathered from published results as a whole, where major surgery shows up as far superior to all conservative treatments, and thoracoplasty as the method of choice. Intercostal tube drainage by open or air-tight methods may, of course, be indicated as preliminary by toxicity.

As in pure tuberculous empyema the prognosis is improving with advances in technique. Thus Berry reported of seventy-five cases only fifteen recoveries, but Hedblom has recently obtained obliteration in thirty-seven of fifty-nine, and the Tuberculous Empyema Commission states that of 134 with virulent infection sixty-five were cured and twenty-eight clinically well. These figures are a great advance on Hedblom's result of only six recoveries in sixty-one treated by conservative and minor surgical measures.

Conclusion

We must therefore agree with the finding of the last report of the Committee of the National Tuberculosis Association of America that while no one seems to have found a specific for this distressing complication, we can only hope to follow the method productive of the greatest good, and conclude that while a predominantly tuber-

culous infection may warrant conservative treatment, a predominantly secondary infection or an underlying lesion which renders the empyema a secondary consideration demands major surgery.

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MAJOR HYSTERIA*

BY

W. RITCHIE RUSSELL, M.D., F.R.C.P.Ed.

ASSISTANT PHYSICIAN TO EDINBURGH ROYAL INFIRMARY AND TO THE DEACONESS HOSPITAL, EDINBURGH

To those who are unfamiliar with the endless variety of physical disturbances which may occur in hysteria the disease presents many diagnostic pitfalls. It is my intention in this paper to give some illustrations of these and to attempt to show how the disease may be diagnosed and how it may be treated. I shall endeavour to use simple terms to explain what seem to me to be the mental mechanisms through which hysterical states are liable to develop.

Evasion and Dissociation

Most of us have the germ of hysterical behaviour in ourselves. Our ability to forget unattractive appointments, and our desire to be deaf to unpleasant facts show a mental process not unlike that seen in major hysteria. In these examples our forgetfulness or deafness relieves us of some mental disturbance or bother. We do not really fail to remember or to hear, but we are able to push unpleasant facts into a corner of our brain which we are able to forget about in everyday life. In achieving this we demonstrate our ability to split our mind into compartments which are almost "watertight." In hysteria this faculty is developed to a remarkable degree, and is described under the term—dissociation of the personality. Both in our own case and in the case of the hysteric, this juggling trick which we play with our minds enables us to avoid some difficulty, and there can be no doubt that the individual who makes a habit of evading awkward situations is thereby cultivating a series of mental processes which, under certain circumstances, may lead to the development of some manifestation of major hysteria.

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