THE DIAGNOSIS AND TREATMENT OF EMPYEMA.

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

(Physician, Royal Chest Hospital and Papworth Village Settlement.)

Few diseases conform so seldom to their classical book diagnosis as empyema or purulent pleurisy in its various types. If even the majority of cases showed consecutive layers from below upwards on one side of the chest, of dullness with absent breath sounds and increased vocal resonance and fremitus, ægophony, and then tubular breathing, with displacement of the heart to the opposite side, there would be little difficulty in differential diagnosis, nor should we see the tragedies of long-standing chronic empyema which are still admitted to our hospitals. The reason is that it is very unusual for empyema to be anatomically complete. It may, of course, be so found in infections with malignant haemolytic streptococci where the onset is acute and limiting adhesions are not present. In most cases, however, the effusion is so tied down by adhesions that division can be made into types according to the position of such restricting bands; for example, interlobar, mediastinal or diaphragmatic empyema.

It follows that the basic infective pneumonitis which is the true primary cause of both conditions will often make the differential diagnosis as between interlobar empyema and lung abscess exceedingly difficult; every physician will agree that the patient's future depends so much on accurate definition and localisation that every diagnostic means at our disposal must be employed to this end. The past ten years has accumulated vast evidence of the usefulness of X-rays and in especial lipiodol X-rays in this difficult subject, and explained why previous errors were so prevalent. It has often been said of late that carcinoma of the lung is not so much more frequent as so much more frequently diagnosed; the same may be said of lung abscess in many cases that would formerly have been considered in all good faith as due to localised empyema and so treated with disastrous consequences; indeed the comparative frequency of abscess within new growth of the lung is now recognised as a still further entity that must be considered in the differential diagnosis of chronic empyema.

Clinically the condition falls into acute and chronic types:

Acute Empyema.

The primary cause of acute empyema is seldom from without; it is almost always from within the lung, and secondary to such conditions as pneumonia, abscess, gangrene and suppurative pneumonitis. It is just as seldom below the diaphragm in its origin from such cause as a sub-phrenic abscess, chronic suppurative appendicitis and gastric ulcer.

The commonest cause is pneumonia and the empyema may be coincident with the disease or follow it very closely. While the pneumococcus is the most usual one in post-pneumonic cases, the streptococcus, particularly the haemolytic variety, is the most serious of all causative organisms, and is often found in the coincident type. In rare cases the staphylococcus or bacillus coli may be demonstrated.

While the disease may set in most acutely, simulating an acute pleurisy, the onset is in a large number of cases very insidious, and there may be no characteristic symptoms. All the doctor may be aware of from his examination is that there is pus somewhere in the body, as the patient complains of chills and has a
swinging temperature more erratic than that of a serous effusion. He may be aided by finding a higher leucocyte count than is expected in a simple serous exudate, but often diagnosis may be a matter of chance rather than skill. The patient, especially if a child, may look very ill but, as in miliary tubercle, may have no particular symptom such as cough or dyspnoea to call attention to the chest. This is often the case where a previous broncho-pneumonia has been missed as having caused so little upset that the doctor has never been called in, for example following one of the infectious fevers.

On the other hand, there may be the most evident signs of pus in the chest: oedema of the chest wall, bulging of intercostal spaces, and more marked displacement of the mediastinum than would be expected in a serous effusion; there may even be evident pus in the sputum by rupture into the lung.

The most useful single aid in diagnosis is, however, the temperature chart. The fact that no true crisis occurs, or that higher levels of temperature recur within a few days of the apparent crisis should make the doctor suspicious of the presence of a purulent effusion. Unfortunately little aid can be obtained in the coincident type, and only clinical experience in noting the increased pallor of the patient and the marked toxæmia out of proportion to the underlying known lung disease will bring the true diagnosis. In some cases increased cough, more profuse sweating, or an increasing leucocyte count may help, but there is no doubt that needling should be resorted to in all cases where there is any suspicion in the mind of the medical attendant. This is particularly true in the case of children.

The evidence of toxæmia is always marked in the acute hemolytic streptococcal type, and needling usually shows the effusion to be thin, but almost always turbid, from the onset, and producing a pure growth of the organism.

**Differential Diagnosis.** Naturally when on needling the chest pus is found in the exploring syringe it is more than probable that the patient has an empyema. We must remember, however, that other diseases may simulate this condition; we must not let the diagnosis rest at this point, but rather keep an open mind and believe this finding to be a symptom only, until we have definitely diagnosed its underlying cause. On the other hand, we must not conclude that because we do not find pus there is no empyema. Mention has been made of the liability of adhesions to tie down the collection of pus between the diaphragm and the lower lobe, or again in front of the lung, or between the lobes in such a position that it may be very difficult to strike it with the exploring needle. Further, it is very often much more difficult to decide on the real primary cause when a small amount of pus only is discovered, as then we shall be in doubt whether we are dealing with an empyema from outside the lung, with pus from a bronchus, or with an abscess cavity. The possibility of an abscess within the liver or of a sub-diaphragmatic abscess must always be kept in mind; indeed a final diagnosis may not be made until the surgeon has explored the wound with his finger to find whether the collection lies above or below the diaphragm; it may even be in both positions; sometimes the history of an attack of appendicitis or the possibility of a gastric ulcer may aid in such a differential diagnosis.

Clinicians nowadays diagnose interlobar empyema much less frequently than formerly; increasing knowledge of intrapulmonary suppuration has shown that many more cases are really lung abscesses. Urgent diagnosis is a necessity, as there is always a danger of rupture into the lung and subsequent extensive supplicative pneumonitis, as well as the possibility of rupture into the main pleural cavity. Both lung abscess and interlobar empyema may produce exaggerated
swinging of the temperature, and gross osteoarthropathy; some cases are seen where other joints are affected, particularly the elbows and knees.

The most confusing physical signs often appear when an underlying lung abscess bursts into the main pleural cavity. Examples can be quoted where neither physical signs nor X-rays were of any real assistance in reaching the true summation of the condition; only clinical experience, or a so-called clinical "hunch" has compelled the physician to call in a surgeon who has been as surprised as he to find a larger accumulation of pus in the pleural cavity.

**X-rays.** The best diagnostic view is got in a hard lateral film, especially if a grid has been used, as this will cut out some of the accompanying radiation from pneumonic lung tissue or from a coincident sympathetic simple effusion, often found in the main cavity of the pleura. Thus a spindle-shaped or round opacity in the region of a fissure clinches the diagnosis of interlobar empyema. Unfortunately the picture is not always so clear, as very often there is a merging of the shadows of the pneumonic lung and of the empyema, but often the diagnosis can be made by the fact that the upper border of the pneumonic lung is convex and has a still greater density than its lower edge. Screening is always of great aid and often by this means alone will it be possible to find an effusion between the upper and middle lobes, as the shadow it casts has two sharp borders and is of similar density throughout, while the lung above and below this dense area is quite clear.

Of all cases, the most difficult in diagnosis are those where there is a small effusion localised to the lower part of the greater fissure. Here X-rays may fail, and it may be necessary to resort to puncture or to an injection of lipiodol to make the differential diagnosis as between unresolved pneumonia or new growth.

Enough has been said to warrant a condemnation of the pernicious habit of "spot diagnosis" that has been employed all too frequently with disastrous consequences in the various forms of empyema. In children miliary tuberculosis is often just as difficult to diagnose without X-rays, but such a label means, and will inevitably ensure, the death of the patient. Contentment with negative results of needling in older subjects has condemned them to the useless delays of sanatorium treatment, and insufficient investigation or localisation has produced such sad results as suppurative pneumonitis and gangrene. The most rigorous diagnosis and differential diagnosis must precede treatment if we are to justify ourselves in the light of modern knowledge.

**Treatment.** The choice of the right moment for open operation is always a problem. Intervention before adhesions have formed is a most dangerous procedure, and it may be necessary for days or even for weeks to be content with aspiration or closed drainage only. Until the mediastinum is fixed by adhesion, until the doctor is certain that the underlying pneumonitis has been arrested, and that his patient's general condition warrants intervention he must not resort to thoracotomy. Two immediate pictures in all acute cases must ever be in the mind of the physician, the possibility of sudden death by an abrupt shifting of a mediastinum which has not been fixed by limiting adhesion, and the possible serious consequence of increased toxaemia by absorption from cut surfaces. In all cases, however, the surgeon should see the patient earlier rather than later, and this for two main reasons: first, that while aspiration by the physician may be indicated more than once, closed intercostal drainage is usually preferable to a second aspiration; and secondly because the physician must never forget the dangers of a permanent lung collapse and a consequent persistent open empyema cavity, where layers of fibrin have been allowed to collect on the visceral pleura.
No patient is safe by any method of treatment until his lung is completely re-expanded. Far too often, as we still see from cases re-admitted to hospitals, drainage has been given up too soon; the physician must always be certain that the sinus has done all it was intended to do and is then absolutely closed before his patient is discharged, and if he is in the slightest doubt of its possible connection with the underlying cavity it is always better to introduce lipiodol and have a further X-ray before deciding to terminate treatment. In addition the patient must be taught how to aid and maintain re-expansion by properly regulated breathing exercises. The little time necessary to obtain the co-operation of the patient by explaining matters to him in non-technical language is always time well spent, and this applies equally to hospital and private practice.

**Chronic Empyema.**

A chronic empyema may be present for months or years, sometimes because no diagnosis has been made, but more often because none has been sought. The patient has become ill so insidiously, his loss of weight, finally alarming, has been so gradual, his cough and his fleeting chest pains so occasional, and his fever so slight, that it has usually been because his friends have compelled him to seek advice for his evident deterioration in general condition that he has finally consulted his doctor.

It may be disclosed in the history that there have been occasional severe sweats or swellings of the joints, but by the time that the majority of sufferers have come for their first examination marked clubbing and osteoarthropathy are present and there may be definite signs of cardiac and kidney damage.

As already noted in the treatment of acute empyema, the chronic condition is often due to an imperfectly drained sinus with continuance of the original empyema cavity, or to a sequestrum of bone left at the time of open operation. Other possible causes are pulmonary fistula, which fortunately often heals itself, and bronchial fistula, this latter being one of the most difficult causes to deal with.

A further cause, which will be dealt with more fully later, is tuberculosis, which may produce a primary or a secondary empyema.

**Treatment.** In non-tuberculous cases continuous systematic washing by Carrel-Dakin solution may be enough to bring the necessary peripheral adhesion and final closure of the residual cavity. Treatment is always very slow and consequently very trying to the patient. His initial improvement may make him restive as he cannot understand why something cannot be done quickly to arrest his disease, but the physician must be certain of efficient adhesion, and in long-standing cases, of recovery in the cardiac and kidney condition before he submits his patient to what will be necessary in most of them, i.e., one of the various types of plastic operations.

Naturally the most difficult in treatment from every aspect is the residual case, re-admitted after previous operation. Only the intelligent patient will understand that his illness is trying to his doctor and his surgeon as well as to himself; to him it is essential to explain that chronic irritation has so thickened his visceral pleura that continuous washing with a decorticating solution must be tried until he is in fit condition to have such surgical treatment as a Schede's operation. Even this is often disappointing in results, and thoracoplasty to such extent as the indications call for is probably best in the majority.
**Tuberculous Empyema.**

Tuberculous infection of the pleura has long been recognised 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.

**Primary Tuberculous Empyema.** As a primary infection tuberculous infiltration of the membrane may occur through the blood or lymph streams. The bacilli may lodge in any part of the membrane, internal or external to the elastic layer, to form true tubercles which may go through all the known stages of evolution up to calcification. The exudate is seldom profuse so that one case may show a steadily extending plastic pleurisy and another a true tuberculous empyema. The latter is usually diagnosed by (1) constitutional debility out of keeping with the minimal extent of physical signs, (2) X-ray examination, (3) the bacteriology of the effusion, or (4) the persistence of a sinus in cases operated on as of pyogenic origin. It accounts for some 10% of all cases of chronic empyema.

The outlook without operation is exceedingly serious, and even where there is no appreciable exudate the disease usually produces a steadily extending plastic pleurisy. A pleuro-pulmonary fibrosis is a common result, and this is often the cause of so-called "pulmonary fibrosis of unknown origin." The patient produces little if any sputum, usually continuously negative for tubercle bacilli, and dies of right heart failure.

**Treatment.** The treatment of such a condition can follow no general rule. In some the reaction may be so profound that rib resection is a necessity, and the resulting sinus of only secondary consideration. Too low an incision is always a mistake owing to the danger of a tract forming between the diaphragm and the chest wall which is very difficult to drain and therefore leaves the patient open to the danger of a residual active focus. If it is intended to go on to thoracoplasty later it may be better in most cases to do the phrenic evulsion first, so that contraction of the diaphragm may aid approximation of the cut ends of the 7th to 10th ribs and so defeat the tendency of the rising abdominal viscera to push them apart.

Several writers have claimed good results from irrigation with a solution of gentian or crystaline violet. Even simple aspiration may be quite successful as leading to adhesion of pleural surfaces, but the literature of end results would seem to favour aspiration and air replacement—i.e. the production of a pyopneumothorax—then later extra-pleural thoracoplasty, as adhesion will take place provided accumulation is not allowed, and the operation wounds will heal as they are made in normal healthy tissue.

There are intermediate cases with considerable pleural thickening in which it may be advisable to irrigate with Dakin's solution to aid decorticatation; it is, however, impossible to forecast which lung will re-expand. Operations of the Schede and Estlander type which aim at obliteration by removing the pleura and intercostal muscles first are usually unsuccessful, and there seems no doubt that further experience in thoracoplasty offers the best chance of improving the prognosis in this grave condition.

**Secondary Tuberculous Empyema.** The second method by which tuberculous infiltration of the pleura can occur is by absorption of a pleural exudate containing tubercle bacilli from an infected lung, i.e., a secondary infection. In some the primary illness has appeared to be a simple pleurisy with effusion, and has been
treated as such with evidently good results for some months; later there has been a sudden onset of tuberculous meningitis by spread through the lymphatics.

This condition is especially common since the introduction of artificial pneumothorax. In such an event there has been an absorption of the tubercle-containing exudate. First a tuberculous pleuritis develops, producing granulation tissue without true tubercles, and limited to the membrane internal to the elastic layer. A profuse clear or blood-stained exudate is the usual result.

While withdrawal may lead to adhesion of the surfaces and a consequent dormant condition, the effusion is very liable to recur and steadily change in character until it becomes a true tuberculous empyema. This secondary type now has a profound effect on the pleura, especially the parietal layer, which develops true tubercles internal to the elastic layer, and non-tuberculous fibrous tissue external to it, perhaps one or more centimetres thick. The possible dangers of fluid complicating artificial pneumothorax are therefore evident.

Secondary Pyogenic Infection. If pyogenic organisms infect such a tuberculous empyema the clinical picture becomes that of acute pyogenic empyema which masks its true tuberculous nature. Theoretically such an infection could occur by the blood or lymph, but in practice the organisms enter from without through some fault of technique in gas replacement, or from within by rupture of the lung and the production of a pleuro-pulmonary fistula.

Streptococci, staphylococci, colon bacilli and many forms of anaerobic organisms have been found. The greenish tint of true tuberculous pus disappears and the effusion becomes a dirty yellow colour, definitely thicker and with an excess of polymorphs. The effect on the pleura is thickening, by increasing fibrosis, of its external layer.

Treatment. There is no evidence that the prognosis is any worse than that of uncomplicated tuberculous empyema. The literature, however, is full of the most contradictory advice on treatment although it leaves one with the impression that totally unlike conditions are being compared. Conservative treatments range from aspiration, either with air replacement or with irrigation, up to the insertion of gelatine and the various forms of oleothorax. No consistent results of such treatments have been reported. Major surgery shows up as far superior to all conservative treatments, and undoubtedly thoracoplasty is the method of choice. Naturally tube drainage by open or closed methods may be indicated as a preliminary by toxicity, in which case the prognosis is, of course, definitely grave. On the other hand, prognosis is definitely improving with advances in technique of operation, so that even six years ago the Tuberculous Empyema Commission could claim 65 cured and 28 clinically well out of 134 cases with virulent infection.
The Diagnosis and Treatment of Empyema

R. R. Trail

Postgrad Med J 1938 14: 251-256
doi: 10.1136/pgmj.14.154.251

Updated information and services can be found at:
http://pmj.bmj.com/content/14/154/251.citation

These include:

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/