THE DIAGNOSIS AND TREATMENT OF TUBERCULOUS PLEURISY.[1]

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TUBERCULOSIS, as a disease, has certain features which it is very necessary to bear in mind in discussing the subject of tuberculous pleurisy. Thus it may be acute in its onset, or slow and insidious. It tends to recovery and the lesion during the process leads to thickening of the part or organ affected and to contraction or scarring. The healing process is a slow one and is essentially associated with fibrosis of the part. When the seat of the lesion is completely fibroid it is not infective—i.e., it contains no active tubercle bacilli. But before this stage is reached there are still foci in the fibrous tissue which contain living bacilli and so may be a source of a fresh outbreak of the disease. In the consideration of tuberculous pleurisy this fact must be borne in mind as well as the final effect of a severe pleurisy on the lung in producing adhesion and thickening of the pleura, in producing contraction of the lung tissue, and in causing a local bronchiectasis at the seat of the adherent and thickened pleura. Acute tuberculous pleurisy is not infrequently associated with a simultaneous outbreak of tuberculosis of the lung tissue and, as is well known, is a common occurrence in cases of progressing pulmonary tuberculosis. The cases, however, which will be more particularly discussed are those which clinically are considered as primary and acute tuberculous pleurisy, with or without effusion. Primary pleurisy may be caused by many different kinds of infective agents—by the pneumococcus, streptococcus, staphylococcus, and tubercle bacillus. These are the commonest causes. Secondary pleurisy may arise by direct extension from an infective focus in or near the chest wall or by metastasis, as in septicæmia, pyæmia, and typhoid fever. These secondary cases are excluded from the present discussion.

A case of primary pleurisy which comes under observation may in the large majority of cases be due either to the pneumococcus, streptococcus, or staphylococcus on the one hand, or, on the other hand, to the tubercle bacillus—i.e., it is *tuberculous* or *non-tuberculous*. This division of acute primary pleurisy is of the highest importance clinically and to be constantly borne in mind during the watching and treatment of an individual case. The diagnosis as to whether the disease is tuberculous or not is of great importance not only in estimating the prognosis but in the treatment of the case. Acute non-tuberculous pleurisy, with or without effusion, almost invariably gets well unless it leads to the formation of empyema. In tuberculous pleurisy the course of the disease is very varied and an accurate prognosis in the acute stage of the disease is impossible. The difficulty of diagnosis between the two kinds of cases may be very great; both tuberculous and non-tuberculous pleurisy may in the initial stages be shown by the same general symptoms of pyrexia, dyspnœa, and dry cough, with stitch in the side, and the same physical signs of a friction rub at the base of the lung, or of the presence of a large amount of fluid in the pleura. In many cases in the early acute stages no diagnosis as to the nature of pleurisy is possible by a careful examination of the history of the illness, of the symptoms present, or of the physical signs in the chest. The diagnosis has to be made by a study (1) of the mode of onset of the disease; (2) of the course of the disease, including an examination of the fluid poured out; and (3) of the after-effects of the disease on the chest wall and lung.

1. *Mode of onset of the disease.*—The acute onset of pleurisy, as shown by more or less sudden illness and pain in the side, with dry cough and pyrexia and with dyspnœa, if the effusion into the pleura is rapidly poured out, may be observed in both tuberculous and non-tuberculous pleurisy. But in certain cases the onset of the disease indicates its tuberculous origin. (*a*) In one class the disease begins insidiously with pyrexia as a rule but with no symptoms or even physical signs referable to the chest and lungs; after a varying period—it may be some week or two or even longer—there are found well-marked signs of pleural effusion. These cases are called "latent" pleural effusion. They are almost invariably tuberculous. In many cases before the effusion of liquid, at the time when the patient shows pyrexia but no symptoms referable to the lungs, the site of disease may be indicated by deficiency in movement on one side of the chest but not usually by any deficiency of resonance or by any marked alteration of the breath sounds or by crepitations. I regard this unilateral deficiency of movement as a valuable sign in the early diagnosis of the site of disease in these cases. (*b*) In another class of cases the tuberculous nature of the disease is indicated by the history (or the observation) of a similar attack of pleurisy which has occurred a few weeks or even months before, either on the same side (as is usual) or on the other. This "recurrent" pleurisy is almost invariably a sign of the tuberculous nature of the disease.

2 *and* 3. *Course of the disease and its after-effects on the chest wall and lung.*—Although the mode of onset may, in the directions indicated, be a valuable help in the diagnosis of the cause of the pleurisy, it is mainly by observing the course of the disease and its after-effects that an accurate knowledge may be obtained of its tuberculous or non-tuberculous nature. Excluding from the discussion cases of empyema, the course of non-tuberculous, non-purulent pleurisy with or without effusion is towards getting well. The disease lasts a varying time. Dry pleurisy may run only a short course with but slight pyrexia and disappears without leaving any appreciable deformity of the chest wall. When effusion occurs in non-tuberculous cases the duration of the disease is longer; the pyrexia lasts for 10, 14, or even 28 days. The fluid is either absorbed or if removed by paracentesis does not as a rule accumulate to any great extent again. Two tappings are rarely if ever necessary in non-tuberculous pleural effusion. The deformity left by even a large effusion is not great; in children it may be very slight and, indeed, unobservable after a few weeks convalescence. In adults, although there may be for some time a deficiency of movement of the affected side and some slight diminution in intensity of the breath sounds, there is no permanent and marked retraction of the side or lung, and recovery of the side may be to all intents and purposes complete. The fluid removed in non-tuberculous pleural effusion, as a rule, coagulates spontaneously and by centrifugalising about 100 cubic centimetres of it and utilising the deposit for examination, either microscopically or by culture, evidence of the presence of the pneumococcus, staphylococcus, or streptococcus may be obtained. Such fluids are, in my experience, never sterile if the liquid is removed in the acute stage of the disease, and the discovery of one or other of these bacteria is an important element in the diagnosis of the disease.

These results are in contrast in many respects to those observed in tuberculous pleurisy with or without effusion, as in this condition the disease is longer in duration in its acute stage, slower in recovery, and during the process of healing more frequently than not tends to well-marked deformity of the chest with contraction of the lung—i.e., to "pleurisy with retraction." Cases of tuberculous pleurisy do not always lead to effusion into the pleura. Two classes belonging to this category may be distinguished, according to the extent and duration of the disease. In the slighter cases the disease commences with pyrexia and friction sound, usually in the lower part of the axilla. The pyrexia may not last long but in some cases it is observed for a month or more, the type of pyrexia being that of an evening rise and a morning fall. It is during this long pyrexia that, without any signs of affection of the lung tissue, changes are observed taking place in the pleura; there is increasing dulness with deficiency of movement and retraction of the chest wall; weak breath sounds are heard but no crepitations; the friction sound has disappeared. The insertion of a needle into the diseased area shows that no fluid is present; the dulness is due to an increasing thickening or fibrosis of the pleura caused by the tuberculous process. More severe cases of this kind are seen in which with prolonged pyrexia for weeks or months there is a persistence of the serious signs of disease of the pleura; but there is no effusion, or only to a slight extent. Recovery takes place in such cases; or subacute general tuberculosis may occur or the signs of a chronic pulmonary tuberculosis slowly make their appearance. In nearly all such cases for the purposes of diagnosis it is necessary to puncture the pleura; not only to exclude

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the presence of pus but to determine whether an effusion is present or not.

Cases of tuberculous pleurisy with effusion frequently give rise to much difficulty in the diagnosis. The general symptoms may be the same as in non tuberculous cases, but the diagnosis is to be made by attention to several points: 1. The pyrexia in tuberculous cases is frequently much more prolonged and, even in the absence of empyema, will last weeks or even months. This persistence of the pyrexia in the absence of suppuration or other complication is a sign of the tuberculous nature of the case. 2. The effusion into the pleura, when once removed by paracentesis, tends in many cases to accumulate again, requiring a second or third paracentesis, or even more, for the relief of symptoms. If repeated tapping of the pleura is required in the class of case under consideration the disease is in the great majority of cases tuberculous. Its necessity is explained by the firm adhesions formed round the edge of the effusion during the tuberculous process, so that the area of pleura affected becomes a sac which cannot disappear because the lung is prevented from expanding by adhesions. 3. The fluid removed in cases of tuberculous pleural effusion sometimes coagulates spontaneously; if, however, the disease has lasted some time or has become chronic coagulation may be slight or absent. In some cases much may be learnt by an examination of the fluid. I will not say that the effusion in a tuberculous case never contains the bacteria which are the cause of non-tuberculous primary pleurisy, but if these and other bacteria are shown to be absent by cultivating from the deposit of the centrifugalised deposit (100 cubic centimetres)—i.e., if the fluid is found to be sterile by cultivation on ordinary media—the disease is tuberculous. In some cases microscopical examination and inoculation of the deposit of the centrifugalised fluid are important. For this purpose it is best to use from 100 to 200 cubic centimetres of the fluid and to centrifugalise till all the solid matter is at the bottom of the tube. In the lowest layer is seen the blood which has got into the fluid during paracentesis, above this is a layer which is in some cases creamy white and is composed of pus cells, while in other cases no white layer is seen but a flocculent layer consisting of a few pus cells and cells of the pleura. The white layer (composed of pus) not infrequently shows the presence of tubercle bacilli on microscopical examination. If such an examination fails to reveal the presence of the bacilli the deposit is to be inoculated either intraperitoneally or subcutaneously into a guinea-pig. The result is often positive, even when no tubercle bacilli are discovered in two or three preparations of the deposit. This examination of the fluid is not solely of pathological interest. It may be the only way in which at that period of the disease the diagnosis may be made as to whether the process is a tuberculous one or not. In some cases, few in number, the tuberculous nature of the pleurisy is recognised by the appearance of signs of peritonitis. It is not common for a primary tuberculous pleurisy to spread to the peritoneum; the occurrence during life may be recognised by the gradual appearance of tumidity of the abdomen or by the presence of a small amount of fluid in the peritoneum. It is much more usual for the tuberculous disease either to spread from the pleura to the lungs or to be associated with disease of the lungs. A history of pleurisy, with or without effusion, is common in cases presenting themselves with signs of tuberculosis at one or other apex, usually on the same side as the antecedent pleurisy. But the cases I want more particularly to refer to are those which come under observation with very evident signs and prominent symptoms of acute pleural effusion which have expectoration containing tubercle bacilli and in which, as may be gathered from the history, the period of commencement of disease of the pleura and of the lung was identical. Clinically such cases may be considered as cases of pleural effusion with expectoration. They are not all tuberculous, as such a condition may be caused both by the pneumococcus and by the streptococcus. The diagnosis is readily made by an examination of the sputum, as, if tuberculous, the sputum invariably contains tubercle bacilli. The after effects of the tuberculous pleurisy on the chest wall and the lung are important points to observe for the diagnosis, as well as prognosis, of the case.

One class of case need not be discussed at length—viz., those cases which, commencing with a dry pleurisy or pleural effusion, terminate after a period of pyrexia of varying duration in subacute or chronic pulmonary tuberculosis. More important from the present point of view are those cases which get well but leave their mark on the chest wall and on the air-containing capacity of the lung. No doubt there are cases of slight tuberculous pleurisy which heal without any serious damage to the chest wall or lung. But other cases, in which there is either that great thickening of the pleura to which reference has already been made or in which there has been a large effusion, show for months, and, indeed, for years, a damage to the part affected. The chief physical signs are well-marked retraction of the side (chiefly the base) and a deficiency in the entrance of air, chiefly into the lower lobe of the lung. Deficiency of resonance also persists over this area, but is not great, owing no doubt to the surrounding compensatory emphysema. In some cases the signs of damage are most severe; inasmuch as after a time, signs of dilatation of bronchi may be evidenced in the appearance of bronchial or cavernous breathing and of dry crepitation. These signs may persist, even though there is every evidence of physical well-being and there is no expectoration.

Treatment.—In the acute stage, beyond the ordinary measures of rest in bed and good nursing, the main point that will arise for decision is the question of removing the fluid from the chest. There does not appear to be any greater danger in removing the fluid in tuberculous cases than in the non-tuberculous and, indeed, there may be a definite reason in advising the removal of the fluid owing to the permanent adhesions that are liable to be formed in tuberculous cases. Apart, therefore, from any urgency for paracentesis such as may occur when there is respiratory distress it is not wise to allow a large quantity of fluid to remain in the chest for too long a time. If the fluid, therefore, is large in quantity and shows no sign of absorption it had better be removed. Cases which run a prolonged course with pyrexia and perhaps no urgent symptoms are best treated by lying in the open air, properly wrapped up and well fed. After the pyrexia has ceased all such patients require a prolonged course of treatment in the open air in order to help the healing of the tuberculous lesion. If this were done more systematically it would perhaps to a great extent prevent a second attack of tuberculosis which so frequently affects the lung tissue and leads to either a very chronic illness or more immediately to death.

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A NEW METHOD FOR THE PRODUCTION OF ULTRA-VIOLET RAYS AND OTHER RAYS BY LOW-TENSION HIGH-FREQUENCY CURRENTS.

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ULTRA-VIOLET rays, actinic rays, Finsen rays, chemical active, or purifying rays of the sun,—under these various terms these rays are at present known and although they have many characteristics similar to their nearest affinities—namely, x rays and Becquerel rays—they are distinct from, and in several ways may be regarded as superior to, them. Hitherto the great difficulties experienced in the production and handling of them have greatly hindered their application to practical therapeutics and although it is not my intention in this communication to deal largely with this aspect of the subject it may be said as far as ultra-violet rays have been tried in therapeutics it has been found that they possess qualities of a curative power in certain diseases much superior to the better known x rays. Although the subject will be gone into more in detail further on, it may be sufficient at this stage to say in a word that the difficulties referred to consist chiefly of the impossibility hitherto of getting these invisible rays to penetrate glass, thus making it impracticable to liberate them in any form of glass tube or lamps, such as is the case with x rays; also the difficulty of freeing them from the infra-red or heat rays. These and other hindrances I venture to claim to have removed and thus effected what may be deemed practically a cold tractable ray. To produce these ultra-violet rays in the manner which will be described below it is essential, in order to secure the volume and purity