fibrillation, heart-block, and branch bundle lesion are all common in syphilis, while the "large silent heart" is sometimes caused by it, and I have known instances of syphilitic pericarditis. Infective endocarditis sometimes gives a positive Wassermann reaction, and it is assumed in this case that such a reaction does not mean syphilis. It is, however, interesting that Byrom Bramwell,³ in a book published before the Wassermann test came into vogue, gives syphilis as one of the causes of infective endocarditis; so acute an observer must have had good reason for his belief.

Symptoms.

Broadly speaking, symptoms of heart failure are the same, whether the underlying cause is syphilis or something else. It has been asserted that nocturnal dyspnoea is characteristic of cardiac syphilis. This has not been my experience, but as many of my cases are early cases of syphilis it is possible that if we take only advanced cases nocturnal dyspnoea may be more frequent. The one characteristic symptom of syphilis in my cases was cardiac pain, which was present in 70 per cent., but this pain has not usually the characteristic features of a classical angina pectoris. In my experience the great majority of typical cases of angina are not due to syphilis. The pain in the syphilitic variety is not so intense, does not appear so suddenly, and differs often in distribution from the pain met with in true angina; occasionally, however, the pain is similar.

The next important symptom is the gastric pain; pain is frequently referred to the stomach, simulating gastric ulcer, but the gastric symptoms are easily distinguishable from those which may be due to heart failure.

Electro-cardiographic Evidence.

The electro-cardiogram gives information about the condition of the myocardium and also the nature of different irregularities. The great majority of electro-cardiographic abnormalities, however, are not characteristic of syphilis, and only denote a damaged heart muscle. They are, however, of the utmost importance in the diagnosis of cardiac syphilis, because in many instances it is by means of the electro-cardiograph that we are able to demonstrate conclusively that there is something wrong with the heart. In my experience, however, there is one type of electro-cardiogram characteristic of syphilis—at least, I have so far not seen it in any other disease. It consists of certain wavelets all over the electric line.

Blood pressure reading yields nothing characteristic of syphilis of the heart; the majority of cases do not show abnormal blood pressure reading.

The result of treatment is often a valuable aid in diagnosis. A patient was admitted into hospital suffering from gastric symptoms, but obviously very ill. The electro-cardiograph revealed branch bundle lesion, a condition invariably fatal if not due to syphilis. In this case syphilis was subsequently admitted by the patient. Antispecific treatment of a few weeks' duration was sufficient to restore the patient to practically normal health, and the electro-cardiogram became normal. Another patient developed a heart affection which was diagnosed as tired heart; he got worse, was able to walk only a yard or two, and became almost blind. He denied syphilis. After admission to the Heart Hospital the Wassermann reaction was found to be double *plus*, and antisyphilitic treatment was so effective that this man is now able to follow his employment. In this case, no doubt, the diagnosis of syphilitic myocarditis was correct. All cases of cardiac syphilis do not show such good results under treatment, but in my experience the great majority improve definitely.

I have said sufficient to make it clear that from clinical evidence alone it is quite impossible in every case to diagnose syphilis of the heart with absolute certainty. The disease is very common. Those who concentrate on the subject will probably make fewer errors in diagnosis than others, but all sometimes overlook a case of cardiac syphilis. It is obviously a very serious matter to leave a case of syphilis without treatment. In my opinion we have no alternative but to adopt the Wassermann test as a routine procedure for all cases suffering from heart

disease. A positive reaction, particularly in a patient with some cardiac trouble, ought to be sufficient indication for a prolonged course of antisyphilitic treatment. We are making this test part of the routine examination at the Heart Hospital.

I am indebted to my house-physicians, Dr. Wilson and Dr. Waters, for their valuable help in connexion with the case-taking.

REFERENCES.

¹ Mackenzie: *Text Book of Heart Disease*. ² Romberg: *Krankheiten des Herzens*. ³ Byrom Bramwell: *Text Book of Heart Disease*

SOME FUNDAMENTAL FACTORS IN THE TREATMENT OF PULMONARY TUBERCULOSIS.

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IN the search for guiding principles in dealing with cases of pulmonary tuberculosis it is more instructive to approach the subject from the point of view of objective in treatment than to attempt a classification of methods.

The discovery of the tubercle bacillus by Koch led to the rational adoption of all those measures calculated to stimulate the normal resources of the body so that the patient might overcome the infecting micro-organism—indirect therapy. It was also to be expected that direct therapy, in the form of vaccines and serums, would emerge from the discovery. The aim of the direct and indirect therapies is, therefore, similar, for the objective in both instances is to overcome the tubercle bacillus on the lines of immunization, producing a serological immunity.

The issue is thus simplified, for, whether the patient is aided by such traditional methods as good food and fresh air in overcoming the infection, or whether vaccines and serums are administered, it is assumed that the toxin-antitoxin balance is involved, and that increase of the antitoxic content will lead to healing. The terms "toxin" and "antitoxin" are used here in the widest sense.

Now the existence of a toxin—probably entirely endotoxin—is established, but what evidence is available in favour of the existence of a specific antitoxin in the serological sense? That the human body must have some method of dealing with tubercle toxin is beyond question. Sanatorium principles and probably tuberculin therapy heighten the capacity to withstand tuberculous toxæmia, but the vital point remains: Does a highly positive resistance to toxæmia on the part of the patient necessarily overcome the tuberculous infection or lead to healing of the diseased focus? In other words, is the defence against the disease purely a local histological problem independent of systemic resistance beyond that required to maintain life?

In dealing with tuberculosis we have become so accustomed to accepting direct and indirect therapy as resting on an established basis that it may seem absurd even to open the question. Still, the answer to the previous query involves the validity of the present attitude to the treatment of this disease.

My survey covers some clinical and experimental aspects of tuberculin therapy, clinical observations, and pathological findings. I wish to emphasize that this survey is only an expression of my attitude to the question raised.

The presence of an antitoxin in the blood has not been proved by direct tests; besides, if such an antitoxin exists, its presence should be manifest in clinical experience.

TUBERCULIN.

To obtain evidence of this antitoxin as contrasted with detoxication tuberculin was studied. The first point to become prominent was that relatively enormous doses could be administered at the end of a gradually increasing course without any apparent improvement in the pulmonary lesion. In such a case the patient has obviously increased detoxicating power, the general condition may also be good, and yet such a favourable situation is not coincident with healing power.

Of course, it may be argued that the above citation is only an example of tolerance to injected toxin if focal reactions are ignored. At least one may accept increased tolerance to injected toxin and proceed to test the position as follows. It is recognized that a skin reaction to tuberculin is a miniature tuberculous lesion. Now lesions established cutaneously are capable of observation. As skin tests are more informative in children than in adults I selected altogether forty tuberculous children suitable for tuberculin therapy, and performed a quantitative skin test. A note was made of the sensitiveness and the time required by the papule to disappear. These children were treated with the same brand of tuberculin over a period of three months, the ultimate dosage varying with the capacity of the child. Within the period when I could still have injected the last dose of tuberculin—namely, on the eighth day—I repeated the quantitative test and made notes as before.

Comparison of the results revealed no relation in regard to either sensitiveness or duration of disappearance of the papule and the artificially produced systemic tolerance. One rather expected to find on the whole an increased sensitiveness.

Cutaneous reactions are too imperfectly understood to make any rash claims on the basis of these experiments; still, the trend of the results is again unfavourable to the standpoint that healing is necessarily dependent on systemic tolerance beyond that required to maintain life. I have simply recorded the results of these tests on a comparative basis in the same case without asserting a defined relationship between tissue and skin sensitiveness ordinarily.

These observations as to the effects of tuberculin are in accord with the attitude of the majority of tuberculosis workers to tuberculin therapy.

CLINICAL AND PATHOLOGICAL EVIDENCE.

There is an inevitable association in the information afforded by these two sources, consequently the facts are recorded under one heading.

Post-mortem records definitely indicate the power of the human body to cure tuberculous lesions in its own way; yet the exercise of this power fails to confer immunity to further disease. When disease occurs it is usually referred to as the result of broken-down immunity. Is this justifiable? If the factor determining activity of disease is failure of immunity, why should a patient suffering from prolonged pulmonary tuberculosis not produce disease in all parts of the body susceptible to the infection? "Lytic hypersensitiveness" is more a defence of a theory than an explanation of this fact. I think the infrequency of generalized tuberculosis in progressive phthisis is against reduced immunity as being the determining factor in resistance.

Again, it is a comparatively common experience to find latent disease becoming active as the result of a fall, a blow on the chest, the occurrence of an acute illness damaging the lung, or the resumption of a life where lung function is placed at a disadvantage. It is difficult to see how the humoral aspects of immunity are involved where the exciting cause is a fall or a blow. It seems to me that trauma is a more vital factor, and the same factor is at work where activity results from illness and bad environment.

These observations amount to a claim that trauma, or strain, is an important factor in the causation of tuberculosis, a factor, too, operating locally, and that general immunity as such is negligible. They do not, however, exclude a local cellular defence, a histogenous immunity, which in a sense is an enlightened way of referring to the "soil." It is not sufficient to explain away reactivation on the basis of a short-lived general immunity, because the longer a case has remained healed the less are the chances of renewed disease.

Now the same idea may be approached from a different clinical angle—namely, the progress of multiple lesions in a patient dying from tuberculosis. I had under observation for over two years a patient dying slowly from pulmonary tuberculosis, and during that period he effected apparent healing of an osseous lesion. In patients with

multiple lesions it is not uncommon to see some healing and others advancing, while the case as a whole is going downhill.

At this point it is interesting to survey the question of healing. It is recognized that healing in tuberculosis, as met clinically, is not by resolution, neither is it dependent on the extinction of the infection. The term "healing" in this disease is really more accurately regarded as a replacement of the primary physiologically active tissue by a practically inert substance which acts as a barrier to further damage by the bacillus. Such is the pathology of tuberculosis that any agent acting through the circulation must fall short of complete extirpation of the infection, and consequently tissue replacement will remain a vital factor.

On the above reasoning the classification of objectives in treatment as direct and indirect therapy does not seem to me well founded. With our present knowledge of immunity reactions, including the reaction of tuberculous and non-tuberculous subjects to infection and toxin, I cannot see that a case is made out for the humoral factors of immunity. The introduction of an intermediate factor, such as lysin, may explain the difference between immunity in tuberculosis and such a disease as diphtheria, but we have then only a hypothetical presentation of the subject, which does not fit in with clinical experience.

The specific reaction to infection and toxin when inoculated suggests a specific histogenous defence; it is a local phenomenon, and would not seem dependent on high general resistance to toxæmia on the part of the patient. In these days of much advertised serums it would be informing to have the basis of their efficiency.

Again, if the general resistance be not a vital factor in local defence, the basis for treatment by sanatorium and allied methods is still to be established as a stimulant to healing. That the sanatorium, being the embodiment of all those factors which make for health, is a sound treatment of the patient is beyond question. It is also obvious that the restoration of systemic resistance is desirable in the interests of living, but the elaborate building up of systemic resistance with the object of producing healing is very questionable. The sanatorium does, however, afford the patient an opportunity to effect healing which might be withheld in the less satisfactory environment of many homes.

For the sake of lucidity I might recapitulate that, first, treatment of the patient is not necessarily treatment of the disease, and secondly, the patient must be treated to allow time for the local cellular defence to effect restraint of the disease.

The treatment of the patient is that of a bacterial toxæmia; the treatment of the diseased lung is rather that of a damaged than an infected organ. I take this standpoint on the basis of the nature of healing as at present known; healing is mainly a circumscribing tissue-replacement process, not even demanding extinction of the infection. The relative indifference to systemic tolerance and the after-history also seem to me to uphold such a standpoint. Even if a safe bactericide were found, such an attitude would still be indicated.

This presentation of the subject, in my opinion, demands rest of the damaged organ as a first step in treatment. Support for this demand is got from observation of cases—thus an attack of pleurisy often heralds an improvement in the disease; a serous effusion is also beneficial. The mechanical support arising from these conditions is apparently contributory. Again, a general review of tuberculosis reveals the great predominance of clinical disease in organs the function of which involves movement; mortality rates from tuberculous infection show the same association with mobile organs. Add to these observations the fact that where mechanical immobilization can be effected—as, for instance, in joints—the results of treatment are far superior to those obtaining in the case of the lungs. Surely, then, a *prima facie* case is made for rest in treatment.

Again, in the treatment of a pyrexial patient absolute rest is the only therapeutic measure available. The toxæmia is lessened because the lung is being less disturbed, and consequently the metabolic and excretory

functions are better controlled. It is agreed that toxæmia is much more easily overcome than the disease itself. Rest in bed brings about the recovery of the patient, but the disease remains a problem. To effect the local healing which I have previously emphasized I wish to suggest that local rest of the lung should be the aim at this stage, rather than graduated exercise. In short, the plan is local and general rest until toxæmia is controlled, followed, if possible, by continuous lung rest until repair has been effected.

SOME POINTS CONCERNING LOCAL REST.

Some hint as to the application of local rest is given by the observation of healing or healed cases. In such patients, apart from the attempt at muscular restraint, there is retraction of the chest wall; fuller investigation usually reveals displacement of the heart and mediastinum towards the diseased side. In other words, the diseased lung is permanently protected, in that its field of operation is finally restricted. This is usually referred to as a result of the pull of the fibrosing lung; I regard it rather as a compensatory process brought about by the healthier lung increasing its field of operation, and at the same time ensuring restraint of function on the part of the diseased organ.

It may reasonably be argued that there is no good outlook for bilateral cases. As a fact there is not much for the moderately advanced bilateral case; improvement, if any, is usually temporary. We all have seen rare cases of extensive tubercle clear up in an apparently miraculous manner. I have felt that most of such cases are examples of superficial lesions where the pleura contributes to the result by promoting fibrotic change, and an enhanced cellular defence may be in operation; but the miraculous case is not our problem.

To return to the question of retraction, which I have described as compensation, I wish to emphasize its vital importance. A tuberculous lung is an organ as permanently damaged as a heart with diseased valves. In the latter case every effort is made to establish compensation, whereas in the former I am not aware of any real recognition of such a process, which is probably more protective than functional.

To investigate the possibility of inducing compensation, and also the results accruing from it, I devised an appliance controlling the ribs on one side and leaving the opposite side comparatively free. In a suitable case it was found that rib restriction brought about some displacement of the mediastinum in a few weeks; this must be a compensatory process, as fibrous retraction could not be operative. It may be that the obvious is being laboured in this connexion, but its recognition in treatment is not obvious. As to results, I have no hesitation in asserting that, acting on the above basis, healing was effected in cases where other methods, including the sanatorium, had failed, and these patients have remained well over four years. These results are claimed for the principle; no claim of universal cures is intended.

In the production of rest certain factors have to be kept in mind. After considerable work on the question of rest in treatment I defined certain postulates, which are as follows:

1. The method must be capable of comparatively wide application.
2. The method must not restrict unduly those conditions which promote a healthy state of the body and mind.
3. It must promote compensation, and not impair to any considerable extent the normal function of the opposite lung.
4. In the event of healing being effected the patient's physique must be unimpaired.
5. It must recognize the mechanism of respiration.
6. The usual distribution of the disease must be noted and efforts made to promote rest at the zone of extension of the disease, and thus facilitate the formation of "barrier" tissue.

The postulates 1 to 4 require no further elaboration; a note on the last two, however, is indicated.

Enlargement of the thoracic cavity is brought about mainly by movement of the ribs below the second, and by diaphragmatic movement. Lung movements are subservient to these two factors. Thoracic enlargement in any direction causes general enlargement of the lung; thus Keith

has shown that movements of the diaphragm are reflected even at the lung apex. Again, tuberculous disease in the lung more frequently starts in the upper part and spreads downwards.

With these points in mind, it may be permissible for the sake of explanation to suggest that we have a lung fractured at the zone of extension of the disease, with an upper diseased and relatively immobile fragment, and a lower more actively movable fragment. With this conception it will be seen that rest can only be attained if restraint be applied to the more freely moving fragment.

CONCLUSION.

This paper is concerned with principles rather than methods. Further elaboration may be desirable in certain parts, but I think I have indicated sufficient grounds to justify a trial of more widespread surgical measures in treating pulmonary tuberculosis.

Condensation rather tends to give the impression of lack of balance in the statement of a case. Still, if I have made my objective clear, and given some indication of the lines along which it should be sought, my purpose in writing this article is attained.

BRONCHO-PNEUMONIA IN CHILDREN TREATED BY INJECTIONS OF EMETINE HYDROCHLORIDE.

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THE alkaloid emetine, chiefly known for its specific action in amoebic dysentery, has in the past been vaunted as a remedy for other conditions, notably the arrest of hæmorrhage (especially hæmoptysis) and respiratory diseases. Within this latter group falls broncho-pneumonia, to which disease alone the ensuing observations refer. Burdick¹ says, "There is now accumulating evidence that the remedy has some beneficent action in this class of diseases aside from that due to its expectorant properties." He goes on to state that Raeburn, Flandin, and others believe that this action is of a decongestant nature. Little seems to have been written concerning this method of treatment during the past ten years, so some further notes on the matter may perhaps prove of general interest.

The Booth Hall Observations.

The cases treated were not picked ones, but 50 consecutive cases of definite broncho-pneumonia, primary or secondary, admitted to the hospital, which takes patients from birth to 16 years old. As a control, 50 further cases treated by any other method were observed, these only being selected in so far that just the same number of secondary cases were taken as had occurred in the emetine series.

Dosage.

Bertrand² states that for pulmonary conditions he used a smaller dose than that normally given in dysenteric cases. The daily dosage used in this series was roughly in accordance with the following table:

Age up to 4 years ...	...	...	...	1/9 grain
4 to 10 years ...	...	...	...	1/6 "
10 to 15 years ...	...	...	...	1/3 "

These were used as commencing doses, but each case had to be judged by its reaction. In the majority of cases these amounts were found quite adequate to produce effects. The salt of emetine used was the hydrochloride, this being the most readily soluble, and the suitable quantity was administered daily by hypodermic injection.

The number of doses administered to any one case in the series varied from two to nine. In the main it was found that if at the end of six daily injections of the drug the case had not shown definite signs of settling, as regards temperature, pulse, respiration, and clinical picture, little further benefit was to be expected from its use. In a number of cases the doses were discontinued short of six.