A Lecture

ON THE

EARLY DIAGNOSIS OF PULMONARY TUBERCULOSIS.

GIVEN AT THE LONDON MEDICAL GRADUATES' COLLEGE AND POLYCLINIC.

BY

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THE early diagnosis of pulmonary tuberculosis is one of the most important subjects in the whole realm of the practice of medicine. As illustrating this I may mention three facts. First, the great prevalence of the disease in this country. Secondly, whatever form of treatment is to be adopted, it is almost universally admitted that to obtain any substantial good, treatment must be begun in the early stages of the disease. And, lastly, the early diagnosis of this disease is very frequently attended with extreme difficulty. Broadly speaking, there are three classes of patients who present themselves for examination at a chest hospital: (1) Those in whom a diagnosis of pulmonary tuberculosis can be made with certainty; (2) those in regard to whom it can fairly readily be said that they are free from the disease, and (3) a considerable proportion in whom a diagnosis is extremely difficult, where the most thorough inquiry into the symptomatology, the most careful examination for physical signs, and sound judgement are required before we can with certainty place them into either of the two classes already named. It is with this third class that I propose mainly to deal, for it is among these questionable cases that the earliest stages of the disease are found, and we will further limit our consideration to the common variety of the disease—that is, the fibro-caseous. I shall not deal with the acute (whether miliary, bronchopneumonic or pneumonic) or fibroid varieties.

It may be said that the examination of the sputum should settle the question. That is not so; because either there may be no sputum to examine, or, if there is, there may be an absence of tubercle bacilli on repeated examination when undoubtedly the patient is suffering from tuberculosis. There is a tendency on the part of many practitioners to place too great reliance upon the result of the examination of the sputum, and correspondingly to neglect a close inquiry into the symptomatology, and, what is of far greater importance, a thorough examination for early physical signs of the disease. During a period of three and a half years it was my lot to examine the sputa of some thousands of patients treated at the Brompton and Victoria Park Chest Hospitals. During this time it was proved beyond all shadow of doubt that a due consideration of the symptoms and a very careful examination for physical signs were sufficient to establish a positive diagnosis in by no means a small proportion of cases several weeks, and occasionally even months, before tubercle bacilli were found in the sputum. This is really not surprising, because it is necessary to have some breaking down of a tuberculous focus and a communication between such a focus and a bronchus in order to give rise to bacillary sputum, and this may occur in only a comparatively late stage of the disease. The examination of the sputum is of very great service in those patients who suffer from bronchitis and emphysema. Indeed, as a means of diagnosis of early pulmonary tuberculosis it is of far greater value in this class of patients than in younger people, for the physical signs of the primary disease frequently mask the presence of a tuberculous lesion, except in its later stages. Consequently the sputum should be examined periodically and as a matter of routine in all cases of emphysema and associated bronchial catarrh. It should be remembered that pulmonary tuberculosis in elderly people is fairly common. When tubercle bacilli are found in the expectoration, and we are able with certainty to exclude tuberculosis of the mouth, pharynx, and larynx, a diagnosis of pulmonary tuberculosis should be made, even in the absence of

symptoms and of physical signs. It is worth while making inquiries in regard to the family and past history of the patient, and whether the patient's general health has been lowered by such causes as overwork, mental worry, excess in alcohol, or over-lactation, and whether at the time there is any one suffering from pulmonary tuberculosis in the house or at the place of work.

Onset and Symptomatology.

The clinical picture is very varied, and the symptoms often suggest some other system than the respiratory, and for these reasons mistakes are made, the symptoms being attributed to some trivial and temporary cause instead of pulmonary tuberculosis.

The commonest modes of onset are: (1) An attack of common bronchial catarrh in which the cough continues; (2) repeated attacks of bronchial catarrh and the super-vention of chronic bronchitis and emphysema (as already stated, it is here that the examination of the sputum for tubercle bacilli is of such value); (3) influenza; (4) haemoptysis; (5) the insidious form (such as loss of strength and anaemia); (6) pleurisy.

The influenzal mode of onset is now well recognized. Buchanan has recorded 12 cases. But a word of warning is necessary; when a patient says he has had "influenza," it is always well to inquire whether any of the characteristic symptoms of that malady have been present, because the general public often apply this term to any attack of fever, whatever its cause may be, and in this way the initial symptoms of pulmonary tuberculosis have frequently been diagnosed as influenza. But the attack may be one of true influenza, which has so lowered the patient's vitality that he has become infected with tuberculosis. On the other hand, influenza may occur during the insidious course of pulmonary tuberculosis, and cause the latter to take on a much more acute phase; or it may light up a quiescent lesion. Another warning is necessary. It must not be forgotten that haemoptysis is by no means rare in the pneumonia of influenza, and furthermore a subacute relapsing bronchopneumonia, with loss of flesh and even slight blood-spitting, may occur.

In the insidious mode of onset, the symptoms may be general ill-health, languor, debility, loss of strength, an irritable or depressed disposition, breathlessness, palpitation, night sweats, functional derangement of the digestive system, anaemia, or tachycardia—either persistent or on slight excitement. In all cases in which a patient complains of these symptoms there are two points which should invariably be inquired into—namely: (1) Is there a steady loss of weight? and (2) Is there any pyrexia? Either of these, in the absence of other discoverable causes, such as tuberculosis of the lymphatic glands or some joint disease, should arouse suspicion. Of course, loss of weight, as well as night sweats, may occur in general debility having such a cause as overwork or over-suckling. On the other hand, loss of weight is not a necessary accompaniment of early pulmonary tuberculosis, especially in anaemic girls. The temperature should be taken four times a day. This presents the possibility of an inverse type, which exceptionally occurs, being overlooked. As a rule, the maximum temperature is found from 4 to 8 p.m., and the minimum from 2 to 8 a.m. One of the characteristics of the fever in pulmonary tuberculosis is its fluctuating and irregular course. The temperature should also be taken after exercise, not because a mere elevation is necessarily indicative of tuberculosis, since this may occur in healthy people after exercise, but because it is characteristic of tuberculosis that the temperature sometimes does not return to the normal within an hour, which it invariably does in a healthy person.

Haemoptysis.

Of all the symptoms of pulmonary tuberculosis spitting of blood is the most important from the point of view of diagnosis. Haemoptysis is a common mode of onset, but, as far as my observations go, it is not such a common mode as is frequently supposed. It is quite true that a large percentage of patients complain of blood-spitting as a first symptom, but on careful inquiry it will be found that a considerable proportion of them have suffered from a cough or other symptoms for some time previously. The value of blood-spitting in diagnosis rests upon the thoroughness

and success of an inquiry in regard to two points, namely: (1) Does the blood come from the respiratory organs—in other words, is it a case of true haemoptysis? and (2) if it is a case of genuine haemoptysis, is there any cause other than pulmonary tuberculosis to account for it? The rules I follow are these: If it is a case of genuine haemoptysis, and if all other causes than pulmonary tuberculosis can be excluded, and there are suspicious symptoms or physical signs or both, a positive diagnosis of early pulmonary tuberculosis should certainly be given; and even in the absence of suspicious symptoms and signs, since in a very large majority of cases there is a small or deep-seated tuberculous lesion in the lung, though the lesion may heal completely without treatment, there are grave risks, and the only safe course is to promptly put the patient under proper treatment. It should be remembered that there is no such thing as idiopathic haemoptysis, and “phthisis ab haemoptoe” does not exist.

It is not always easy to exclude haematemesis. In haemoptysis the blood is usually coughed up, bright red in colour, frothy, mixed with sputum, alkaline, and as a rule does not clot; there is frequently a history of pulmonary symptoms, and there may be physical signs in the lungs. On the other hand, a possible pulmonary origin is not put out of court by the mere facts that the blood is vomited, is mixed with food, and is acid, for sometimes the blood is first swallowed and then vomited. In haematemesis the blood is usually dark red, non-frothy, and generally clotted; there is often a history of gastric symptoms and abdominal physical signs may be present. If the blood is brought up without the act of coughing or vomiting, it is probably not a case of haematemesis. In true haemoptysis, if the cough persists, the sputum is frequently stained with altered blood, or contains small clots for some days afterwards; this is the most reliable sign of pulmonary haemorrhage. When a large quantity of blood is brought up the differential characteristics already named are of no value, since the blood, whatever its source, is usually bright red in colour, alkaline, non-frothy, and not mixed with food or sputum. But a lesion of the lung sufficient to give rise to haemorrhage of this magnitude is revealed by well marked physical signs, so that for purposes of diagnosis of pulmonary tuberculosis it is not necessary to determine the source of the bleeding.

Next the nose, mouth, pharynx, trachea, and larger bronchial tubes must be excluded as the source of bleeding. A uniformly bright red watery fluid often means that the point of origin is in the mouth. Mere streaks of blood may be present in the expectoration from many causes, and therefore cannot be relied upon as necessarily indicative of pulmonary tuberculosis. In bronchitis and emphysema there may be a rupture of capillaries during a paroxysm of severe coughing. A few cases have been recorded in which malignant disease of the thyroid, pressing on the trachea, has given rise to local congestion and catarrh, with accompanying cough, and the expectoration of scanty mucus tinged with blood; if the enlargement has lasted for some time, emphysema of the lungs may be set up. Aneurysm of the thoracic aorta, pressing on or perforating the trachea, bronchus, or lung, may cause haemoptysis.

One of the most important conditions to exclude is mitral disease, whether secondary to aortic, or primary, and especially mitral stenosis. Mitral stenosis is probably the next most frequent cause of haemoptysis to pulmonary tuberculosis, and a common cause of mistake. Perhaps the heart is not examined at all, or if it be examined it is by no means rare for the characteristic murmur to be absent. Furthermore, because of the inhalation of blood there may be evidences of patches of consolidation, with alteration of the breath sounds and some accompaniments. The heart should be examined on several occasions for the characteristic murmur, the character of the pulse should also be noted, and whether there is a short sharp first sound or not.

Rarer causes are diseases of the blood, haemophilia, some acute specific fevers, suppurative processes in the mediastinum, foreign bodies in the air tubes, bronchiectasis, paroxysms of whooping-cough, injury, pulmonary syphilis, aspergillosis, actinomycosis, hydatid, new growth, and pneumonokoniosis. Lastly, Osler believes haemoptysis may occur in vascular degeneration.

Pleurisy.

The first symptoms and physical signs of tuberculosis may be those of pleurisy, either the dry or the serous form. These may persist until the symptoms and physical signs of pulmonary tuberculosis declare themselves, or the latter may only appear later. Tuberculous pleurisy most commonly occurs at the apex of the lung. Dry pleurisy limited to the axillary region or to the base of one lung is undoubtedly often non-tuberculous. Dry pleurisy limited to one apex is, in the absence of croupous pneumonia, almost certainly tuberculous. Bilateral dry pleurisy, or one that is widespread over the lung, is, in the absence of new growth, probably tuberculous; and quite two-thirds of the cases of pleurisy with effusion have the same cause, especially when, on microscopical examination of the fluid, the cells are found to be lymphocytes, in contrast to polynuclear leucocytes.

Physical Signs.

Although the symptoms are important, it is on the early detection of physical signs that the early diagnosis of pulmonary tuberculosis mainly rests. I would lay down two broad principles of vital importance:

First, the locality of the physical signs. In the great majority of cases the primary seat is from 1 in. to 1½ in. below the summit of the lung (rather nearer the posterior and external borders). A less common primary seat is the second and third interspaces below the outer third of the clavicle. The lower lobe of the same lung is usually affected early. It is best examined by getting the patient to place the hand on the opposite shoulder and examining the back internal to the vertebral border of the scapula. The common primary seat of the lower lobe is from 1 in. to 1½ in. below its summit, that is, opposite the fifth dorsal spine, midway between the spinous process and the vertebral border. The lesion then spreads along the line of the vertebral border of the scapula. This early affection of the apex of the lower lobe is of immense value in differential diagnosis. Occasionally a “crossed lesion” is found, that is, the lower lobe of the opposite lung is involved before that of the side primarily affected. The supraclavicular and infraclavicular regions, the supra-spinous fossae, and the interscapular regions opposite the fifth dorsal spine should always be very carefully examined.

Primary basal tuberculosis, apart from the pneumonic type, is very rare, and when it does exist there has usually been some antecedent damage sustained, as, for example, by pleurisy. What is called primary basal tuberculosis is often secondary to an apical lesion, which, because it has healed, or for other reasons, has been overlooked.

Secondly, a diagnosis of pulmonary tuberculosis from one single physical sign should never be made. Considerable variations from the normal are often met with in perfectly sound chests. As an example of this fundamental and all-important rule may be cited the fact that if distinct impairment of the percussion note is discovered at one apex, it should, if pathological, be accompanied by other physical signs, such as diminution of expansion, or alteration in the vocal fremitus or breath sounds; otherwise it may be merely indicative of curvature of the spine. Similarly, the mere presence of bronchial breathing may be due to an abnormal position or course of a bronchus; but when such breathing is pathological, the lesion which gives rise to it is always sufficient in degree to cause some other physical sign or signs. On the other hand, several slight abnormal signs, which when taken separately are of no pathological significance, if they all point in the same direction afford just grounds in making a positive diagnosis.

Another point of importance is to compare exactly corresponding points on either side. It is best to have the patient stripped to the waist, seated or standing, in a good light, and to adopt the usual plan of inspection, palpation, percussion, and auscultation.

One clavicle may be seen to stand out more prominently than the other, and there may be some hollowing of the fossae above and below; but these, and also displacement or uncovering of the heart, are evidences of fibrosis, which is a secondary change, and are not found in the very early stage of the disease. Diminution of expansion at one apex is a very early sign. It may be observed by standing in front of the patient, or sometimes it is better observed by

standing behind and looking down the front of the chest from above. There are various ways of noting it by palpation. The thumbs may be placed on the second ribs below the clavicles, the eyes being fixed on the thumbs when the patient takes a long breath; or the hands may be placed below the clavicles; or, standing behind the patient, the thumbs may be placed in the supraclavicular and the fingers in the infraclavicular regions, or the hands on the shoulders close to the neck, the fingers lying over the clavicles touching the infraclavicular areas and the thumbs lying on the supraspinous fossae.

Diminution of expansion at one apex is of great significance and is a very early sign. It is almost always found before any alteration in the percussion note and frequently as soon as any change in the character of the breath sounds. It may only amount to a slight lagging of movement behind that of the other side. If it merely indicates a localized thickening of the pleura without any lesion of the underlying lung, this apical pleurisy is almost always tuberculous. Normally the vocal fremitus is as a rule greater on the right side than on the left—almost certainly in more than 75 per cent. of cases. If it is found to be equal on both sides and well marked, there is probably some disease in the left upper lobe. If it is more marked, the probability is still greater. If the fremitus is equal on both sides but less apparent than normal, it almost certainly means some diminution at the right apex, and this signifies that at that part there is either a thickened pleura, a pleural effusion, or some localized emphysema. This localized emphysema is most frequently due to a deeper tuberculous lesion. Any changes in the vocal fremitus in the supraspinous fossae have the same significance.

When we come to percussion we have to deal with a difficult matter. There are some general rules which it is of very great importance to observe: First, percussion should be performed from the wrist-joint. When possible the examiner should stand immediately in front of the part percussed, percuss exactly corresponding points, and employ exactly the same percussive force on either side. When percussing in front the patient's head should be exactly in the middle line, and as a rule the best results are obtained by light percussion. When percussing behind, the patient should lean slightly forward and cross the arms, but letting the shoulders drop in order to relax the muscles. It must be remembered that as a normal variation one finds the percussion note below the right clavicle slightly raised in about half the cases of healthy chests. The upper border of the lung should first be ascertained. A lowering on one side is significant; but when it is bilateral its value is little, since the height to which the lungs ascend varies in normal individuals. In early pulmonary tuberculosis there is frequently diminished resonance. The note becomes higher pitched, shorter, sharper, lacks the normal tone, and there is a sense of increased resistance to the finger. This can usually be made out on shallow breathing, but occasionally it is more easily detected after the patient has taken a deep breath, owing to the diminished entry of air into the affected part. When impairment is more marked with light than with deep percussion, there is a strong possibility of the existence of a thickened pleura. The heavier the force required to bring out the lack of resonance, the deeper is the infiltration or consolidation. Sometimes the note on the affected side is not only as resonant as that of the other side, but it may even be hyper-resonant. This is indicative of a one-sided apical emphysema, and such a condition is almost always due to an underlying tuberculous scar. I have several times seen a positive diagnosis of pulmonary tuberculosis given from the mere fact that there was a difference in the notes of the two sides, though there were no other physical signs, and there was no pulmonary lesion. Even a considerable difference in note may be present when the two sides are not perfectly symmetrical. We should most carefully examine for the presence of scoliosis, for this condition influences the position of the ribs and the shape of the chest. It is not enough to inquire about the spinous processes. We should note whether the upper ribs are more curved on the one side and flattened on the other, and look for other signs of scoliosis. Even a very slight deviation of the vertebral column from the normal is sufficient to cause a difference in the heights of the two

supraclavicular regions and the two suprascapular regions, one side being more rounded than the other. The percussion on this more convex side is less resonant. The breath sounds may also be diminished in loudness.

In people who are extremely emaciated there may be well-marked impairment at both apices, and yet no pathological change to account for it. This impairment may be accompanied by diminution of the breath sounds. The explanation is difficult; but it may be that, on account of the weakness and rest, the apices are not properly filled with air, and therefore sink lower than normal.

If we follow certain rules, we get incalculable help from auscultation. We should first of all see that the patient breathes properly—that is uniformly, fairly deeply, and, if possible, through the nose. Some patients indulge in spasmodic movements of the chest, with the diaphragm fixed; others open their mouths and breathe noisily. Nervous persons, especially women, when asked to take a long breath sometimes produce a false movement of the chest, but at the same time close the glottis, and, since practically no air enters the lungs, the breath sounds are almost inaudible; or they may narrow the glottis, and, as a consequence, bronchial breathing is heard. These errors are avoided by noting that the abnormality in the breath sounds is bilateral, and by getting the patient to cough. In auscultation, the expiratory part should always be taken as the criterion as to whether breathing is truly bronchial or not. It is of far greater importance than the inspiratory part. In true bronchial breathing the expiration should be blowing, and have a uniform pitch and intensity through the whole act. Auscultation over any part of the lung without asking the patient to cough is of incomplete value, because in a certain proportion of cases accompaniments are only found during or immediately after the act of coughing, and also because the significance of râles largely depends upon whether they persist on repeated coughing and deep breathing. Normal, or vesicular, breathing is described by Fowler as a rustling sound, audible during inspiration, generally followed without any appreciable interval by a sound of lower pitch, shorter duration, and faintly blowing quality heard during expiration, but this may be absent.

In bronchial breathing the inspiration is blowing in character; the pitch is higher than in vesicular; there is a distinct interval between inspiration and expiration; the expiration is still more blowing and has a still higher pitch, and has an equal or longer duration than inspiration. As I have said, in true bronchial breathing the expiration should be blowing, and have a uniform pitch and intensity through the whole act. Bronchial breathing is normally heard over the seventh cervical spine, and this locality should always be taken as the standard. Broncho-vesicular breathing, in which the characters of bronchial are combined with those of vesicular breathing, is heard normally over the manubrium in front and the upper part of interscapular region behind. Now it is of the utmost importance to distinguish between the intensity and quality of the breath sounds. Negligence in this is one of the commonest sources of errors. Regarding the intensity of vesicular breathing, even in normal conditions there is a very great range of variation. In one chest it is very loud, in another it is quite faint. Pulmonary tuberculosis has frequently been diagnosed by mistaking harsh vesicular for bronchial breathing. The error can easily be avoided by comparing one side with the other; it is bilateral. Bronchial breathing should be diagnosed by the *quality* and not by the loudness of the breath sounds. But even here it should be remembered that normally the breath sounds on the right side are of slightly higher pitch, and the expiration is rather prolonged; beneath the right clavicle they may be even bronchial, and this has occasionally led to the diagnosis of consolidation or of a vomica in a healthy person. It is sufficient to note the absence of corroborating physical signs. Cog-wheel breathing, when heard equally over the whole of one lung, is of little significance; when limited to one apex it may be of diagnostic value, but even then it is by no means reliable, as it may be caused by irregular muscular contraction. Harsh breath-sounds, with prolonged expiration, limited to an apex, is generally the earliest auscultatory sign of pulmonary tuberculosis. There may be merely prolongation of expiration. If so, what we infer from it depends entirely on its character.

If it is only faintly blowing and of low pitch it probably indicates emphysema; if it is truly blowing and of high pitch it usually means infiltration.

Weak breathing is, in my experience, usually a later sign than harsh breathing. It is frequently overlooked. A marked diminution of the respiratory sound at one apex, especially the right, if persistent on repeated examination, is, in the absence of a localized condition of emphysema, thickening of the pleura, pleural exudation, or bronchiectasis in which the bronchus is plugged, in my opinion, extremely suspicious. It is of greater value when found at the right apex than the left, because normally the breath sounds are louder at the right than at the left apex. The breath sounds in the second and third spaces are frequently harsh, when those above and below the clavicle are feeble. The breathing over the opposite upper lobe may be harsh, owing to increased functional activity; and this may give rise to an error in diagnosis regarding the site of the lesion, because the feeble breath sounds of the side primarily affected are undetected. As the affected area becomes more densely infiltrated the breath sounds become truly bronchial.

Adventitious Sounds.

Adventitious sounds, when present and correctly understood, are probably of greater help in the early diagnosis of pulmonary tuberculosis than any other physical sign. First of all, get the patient to cough and to breathe deeply, for frequently these acts are accompanied by râles which are inaudible in quiet breathing; and, conversely, the significance of accompaniments often depends upon whether they disappear on deep breathing and on coughing. I have already said that a dry pleurisy limited to one apex is, in the absence of croupous pneumonia, almost certainly tuberculous. Also a bilateral dry pleurisy, or one that is widespread over the lung, is, in the absence of new growth, probably tuberculous. This pleurisy may reveal itself by pleural friction, either of the usual kind or of a crepitant character. As a rule, however, the first accompaniments heard in pulmonary tuberculosis are small crackling râles—that is, sharply-defined, crackling sounds, chiefly heard during inspiration; indeed they may be limited to inspiration. It is sometimes almost impossible to distinguish small crackling râles from crepitant pleural friction. The former are not likely to commence at the very beginning of inspiration on quiet breathing, and are not so frequently heard during expiration as are pleural sounds. If the sounds disappear after coughing, a pleural origin can practically always be excluded, and almost always if the accompaniments only occur after coughing. At a later period of a tuberculous invasion sounds of a clicking character are occasionally heard. Only one or two “clicks” are heard during the act of inspiration, and they are practically pathognomonic of commencing softening in a tuberculous focus. When softening has advanced numerous medium and large sized crackling râles may be audible.

Occasionally on listening over the upper part of the chest, both anteriorly and posteriorly, sounds resembling râles are heard immediately after coughing, which are oesophageal in origin, and due to the patient swallowing. They are recognized by the fact that they are bilateral, and are not heard when the patient is told not to swallow after coughing.

Apart from this possible source of error, and also apart from pleural accompaniments, it may be asked whether crackling sounds heard over one apex or both apices are pathognomonic of a tuberculous lesion. The answer is in the negative. First of all, the râles associated with mitral stenosis have been noted. Secondly, sometimes when a patient, who is accustomed to shallow breathing, takes a long breath, the air vesicles, which in a localized area were hitherto collapsed, become suddenly distended, with the result that crackling sounds may be produced; this is especially apt to occur in emphysematous subjects. Their significance may be ascertained by the fact that they do not persist after continued deep breathing and repeated coughing. But even the presence of persistent and strictly localized crackling sounds in the upper part of the chest is not absolutely pathognomonic of infiltration or consolidation. For I have seen a few cases in whom a diagnosis of pulmonary tuberculosis had been made because of the mere presence of crackling sounds over the

upper part of both lungs. There were no other physical signs, but the sounds persisted. The cases were watched, and all the channels of inquiry available for the diagnosis of pulmonary tuberculosis were taken advantage of, with a negative result. Such cases are occasionally met with. For this reason, as well as for others, as I have already stated, a cardinal rule should be that a diagnosis of pulmonary tuberculosis from one single physical sign should never be made. Instead of pleural friction or crackling râles, the first auscultatory sign may be the presence of rhonchi or bubbling râles, which frequently disappear on coughing. When these are strictly limited to one apex, and persist over a considerable length of time, the cause is, in my opinion, practically always tuberculous, and not a simple catarrh. You will thus see that it is the *locality* of accompaniments, whether pleural, crackling râles, bubbling râles, or rhonchi, which is important. Crackling sounds may be heard along the margin of the cardiac impairment, which, as a rule, are of no significance.

Regarding the vocal resonance, all that it is necessary to say is that any alteration of it has pretty much the same significance as alteration in the vocal fremitus.

Other Chronic Pulmonary Disease.

Having diagnosed the presence of a chronic disease of the lungs, affecting mainly or only the apex of one lung, the problem which remains is to exclude a non-tuberculous lesion, such as actinomycosis, hydatid, new growth, or bronchiectasis. This is practically always easy, and I need not go into the points of differential diagnosis.

Other Methods of Diagnosis.

There are a few remaining avenues of investigation which will be briefly dealt with. First, the Roentgen rays. An examination in early pulmonary tuberculosis may reveal a mottled or stippled shadow or irregular movement of the diaphragm on the affected side. As far as my experience has gone, I have never known of any case in which either of these has been demonstrated before physical signs could be made out on careful examination by a competent examiner. Furthermore, the same kind of a shadow may be obtained in new growth of the lung. And impaired movement of the diaphragm occurs in other affections than pulmonary tuberculosis.

Next, the use of Koch's Old Tuberculin subcutaneously as a means of diagnosis. Provided this is used only under certain well-defined conditions, in my opinion it may be employed with perfect safety. The conditions are these: Never use it if the mouth temperature reaches 100° F.; if there has been a recent attack of hæmoptysis; if there are evidences of extensive bronchitis or bronchiectasis, or definite physical signs of pulmonary tuberculosis or tubercle bacilli in the sputum. On the other hand, if these conditions are fulfilled and a certain diagnosis cannot be made by other means, it is of the greatest value and should be resorted to. It is true that cases have been recorded in which a definite reaction was obtained in apparently non-tuberculous subjects, especially in those who have had syphilis (although there was no certain proof that these cases were free from tuberculosis in some part of the body), and undoubtedly some cases of pulmonary tuberculosis do not show a reaction, yet it is an almost absolute test, and in clinical medicine there are very few tests which are absolute. It should be remembered that the reaction may be due to a tuberculous focus in some other part of the body than the lungs and pleura. It is best to begin with 0.001 c.cm. of tuberculin (1 mg.). If no reaction follows within forty-eight hours, administer a second dose twice as large. If there is even a slight rise of temperature following this, repeat the same dose in forty-eight hours. If the reaction to the second dose of the same size is more marked than was the reaction following the first dose, then you may be sure you are dealing with a case of tuberculosis. If these doses fail to produce a reaction, 5 and finally 10 mg. may be used. In regard to von Pirquet's cutaneous reaction and the conjunctival test (Calmette), unfortunately not infrequently it is difficult to say whether the reaction is positive or negative. In both there may be no reaction in the last stages of tuberculosis. Von Pirquet's test can be employed in pyrexial cases. It is more valuable in children, especially in the first three years of life, than in adults.

A reaction always occurs when there is pathological evidence of tuberculosis, though there may be an absence of clinical evidence. The ophthalmic-reaction should never be employed if any affection of the eye is present. Even apart from these cases it is undoubtedly attended with some risk. This test is more in harmony with clinical experience than von Pirquet's.

My opinion is that neither of these tests is nearly of such diagnostic value as the subcutaneous tests.

THE INFLUENCE OF STRONG, PREVALENT, RAIN-BEARING WINDS ON THE COURSE OF PHTHISIS.

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I HAVE repeatedly—and, I venture to believe, conclusively—shown that strong, prevalent, rain-bearing winds exercise a powerful influence over the prevalence of phthisis. I now propose to show that *probably* they also exert an influence on the course of the disease.

There are several lines of inquiry which may be followed in order to ascertain whether these winds do exert an influence on the course of phthisis or not. We may (1) make continuous clinical observations on phthisis cases in some single locality under varying conditions of wind and rain; or (2) we may clinically observe and compare the effects on a series of phthisis patients of residence in sheltered and exposed situations; or (3) we may compare the collective results obtained by similar methods at differently located sanatoriums—some in shelter, some exposed; or (4), lastly, we may compare the average durations of fatal cases whose course has been run in places which present a suitable contrast of shelter and exposure.

It is to this last line of inquiry that I would now especially invite attention. I shall only refer to the three others in order to make clear that they all, so far as I have been able to follow them, seem to lead to the same conclusion.

1. *Observation of Patients in a Single Locality Under Varying Conditions of Wind and Rain.*

This is a line of inquiry which lies peculiarly within the province of sanatorium physicians, and to them one must ultimately look for the width of experience requisite to enable us to form a judgement. My own opportunities have been necessarily very limited. But when, some years ago, the "open-air" treatment began to be introduced into this country and when we were treating a number of cases in connexion with the Royal Devon and Exeter Hospital, it seemed to me that windy and rainy weather unfavourably affected them. About the same time a friend of mine, who had medical charge of a "home for consumptives" at the top of a hill open to all winds, told me that his patients seemed to get on very well except when westerly winds were blowing. In 1904, Dr. A. A. Rodriguez, of Campo in the Azores, was so good as to make inquiries for me amongst the medical men practising in those islands, and told me that, whilst some had noticed no injurious effect from any wind, others had found their phthisical patients were worse when the warm and damp south winds were blowing, and others considered that these south winds did harm and that the north winds, which are dry, did good. A recent statement by Dr. H. Hyslop Thomson¹ that "the sanatorium physician knows well that such winds (strong, prevalent, and rain-bearing) are responsible for elevation of temperature and increased cough and expectoration amongst certain of his patients" is of special importance as embodying just the sort of experience which is necessary for the satisfactory following out of this method. Thus such evidence as I am aware of points in the direction of these winds affecting unfavourably the course of phthisis.

2. *Comparison of the Effects on Phthisis Patients of Residence in Sheltered and Exposed Situations.*

Here my opportunities have been more extensive, though my observations have been far too limited to be more than suggestive. It is obvious that the personal opportunities

of any single observer, not specially devoting himself to the treatment of phthisis, can scarcely, in a dozen years, furnish conclusive evidence by this method. A few examples will suffice to indicate my general experience.

A young lady, after a smart hæmoptysis, had been seen by me doing remarkably well in a wind-sheltered situation. But, being better, she took it into her head to go for a "bracing change" to one of our most westerly exposed hillsides. A fortnight later I was again sent for. The "change" had not been a success, and she had had another considerable attack of hæmorrhage.

A gipsy woman, camping in the open air on the summit of a wind-swept common, was quickly losing ground from phthisis. I got her to shift her camp to the leeside of the hill, and immediately she began to improve.

A man, who had been nursing his son who died of phthisis, developed an empyema. After this had been opened, he went to a very sheltered valley in South Devon, where he did exceedingly well. His second son came to me some time afterwards with early phthisis, and, at his father's request, I sent him to the same place. I have rarely seen so rapid and complete a recovery as the boy made.

A lady with very slight disease of one apex, which had steadily refused to clear up, spent the winter at Chagford, and when I saw her next spring the physical signs had practically disappeared. She went to an exposed health resort in another county, and there the disease broke out afresh.

This north-east slope of Dartmoor, to which I drew attention in 1900 for its comparative shelter from westerly winds and coincidentally low phthisis mortality, has since then been utilized for sanatoriums, which have given very satisfactory results, as I can testify from the cases which I have sent to them.

On the other hand, I have seen cases at various points along our southern sea-board, who had been sent from a distance to live on the actual sea-front, and had been promptly seized with acute chest complications or with hæmoptysis. Yet cases suitably located, in sheltered spots at these very health resorts, do exceedingly well. In one of them several excellent medical observers have noticed that cases of hæmoptysis which have proved intractable on the actual sea-front, lose their hæmorrhages when removed about a quarter of a mile inland, under the shelter of a considerable hill.

Long ago Dr. Thomas Shapter pointed out how undesirable it is to place a consumptive on an exposed sea-shore, and Dr. Ransome has more recently emphasized the seriousness of the consequences of locating such patients on an exposed sea-front.² It seems to me, however, that simple aspect has a good deal to do with the effect. Thus, I remember a patient who had greatly improved in hospital, insisting on returning home to live in the open air at a seaside place which faces almost due east with considerable western shelter. She continued to do very well so long as I had news of her. Also it is not entirely a matter of distance from the sea, for a case which did badly with repeated hæmoptysis, lived on a high hill facing west just above a sheltered seaside town, in which, not far from the beach, several cases I know have done satisfactorily.

An interesting observation comes from the Sandwich Islands. This group is said by Hann³ to present "the finest example of the contrast" between a wet and windy side and a dry and sheltered side. I therefore wrote to find out what was the experience there as to phthisis cases on these two so different aspects. Dr. Grace of Hilo very kindly replied, telling me that it was universally recognized, though the explanation was unknown, that "the windward is the bad side of the islands."

Thus this line of inquiry seems to point in the same direction as the last.

3. *Comparison of the Results of Differently Located Sanatoriums.*

Here one enters on especially difficult and delicate ground. Moreover, I imagine the time is scarcely ripe for drawing conclusions of value. Yet some hints may be gained by considering in what sort of situations the most historically celebrated phthisis health resorts have been established. Dr. E. Campodonico of Lima has recently been so good as to send me particulars of the celebrated resorts in the Peruvian Andes. He writes:

The most famous health resort for phthisical patients in Peru is the territory of Junin, where the towns Tarma, Jauja, Huancayo are located. This region, watered by the river Mantaro, stands at about 3,000 metres above the sea level, on the eastern side of the Andes. . . . These towns are protected