The early diagnosis of consumption / by John Hay.

With John Hay's Compliments

(7)

THE EARLY DIAGNOSIS OF CONSUMPTION

BY

JOHN HAY, M.D., M.R.C.P.

PHYSICIAN, LIVERPOOL STANLEY HOSPITAL;
ASSISTANT PHYSICIAN, HOSPITAL FOR CONSUMPTION AND DISEASES OF THE CHEST;
HON. PATHOLOGIST, DAVID LEWIS NORTHERN HOSPITAL

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THE EARLY DIAGNOSIS OF CONSUMPTION.[1]

BY

JOHN HAY, M.D., M.R.C.P.,
Physician, Stanley Hospital;
Assistant Physician, Hospital for Consumption and Diseases of the Chest;
Pathologist, David Lewis Northern Hospital, Liverpool.

GENTLEMEN,—Since the recognition of the fact that pulmonary tuberculosis is an infectious disease, its diagnosis, and more especially its early diagnosis, as a factor in the maintenance of the general public health, have become matters of supreme importance.

Again, the accepted fact that pulmonary tuberculosis, especially in its early stages, is frequently amenable to certain recognised forms of treatment, makes it most desirable that a correct diagnosis should be made at the earliest possible moment.

I have made the question of the early diagnosis of consumption the subject of this lecture, partly on account of its inherent interest and importance, and partly because it is one that is constantly brought to my notice in the large Out-patient Department of the Hospital for Consumption and Diseases of the Chest.

There the cases naturally fall into three classes:—

(1) Those undoubtedly phthisical, in which the diagnosis is easy.

"The cough, shortness of breath, and expectoration point to

[1] A post-graduate Lecture delivered at the Stanley Hospital, Liverpool, August 2nd, 1904. The lecture was illustrated by a number of patients suffering from pulmonary disease.

the respiratory organs as the seat of the disease; the loss of flesh and strength, to some chronic cachexia; the hectic, to some inflammatory disease; the physical signs which indicate consolidation of the lungs, with breaking down and contraction, commencing at the apex and gradually spreading downwards, render it highly probable that the disease is tubercular in nature, and the discovery of the bacillus makes it certain." [1]

(2) Those classes which one has no hesitation in classing as non-tubercular; and

(3) A number of patients in whom the diagnosis is not straightforward, and who cannot be placed offhand in either group.

There may be suspicious constitutional symptoms without definite physical signs, or there may be physical signs admitting of various interpretations. This group of questionable cases includes patients in the very earliest stages of pulmonary tuberculosis, also those of later stages in whom the disease has been arrested, and is at the time quiescent—cases of fibroid tuberculosis with emphysema or compensatory hypertrophy; and certain asthmatic patients, or again, patients in whom the obvious and predominant symptoms are those of chronic bronchitis—anæmia and debility, with possibly some wasting, and so forth. One could increase the list, but it is sufficiently long to serve its purpose and to suggest the class of patient to which I refer.

Pathology.—A brief *résumé* of the pathological aspect of the subject will be of advantage at this point.

The tubercle bacillus has two actions—one local, the other general. The former inaugurates changes in the lung which are revealed to us through variations in the physical signs, the latter produces a condition of toxæmia by the absorption of the products of the activity of the bacillus, giving rise to constitutional disturbance.

The local action of the tubercle bacillus causes certain changes in the cells in the immediate neighbourhood of the

[1] *West*, p. 527.

bacilli, resulting in the formation of what is called a miliary tubercle or nodule, a small mass of epithelioid cells, derived from the fixed tissue cells, and surrounded by a number of lymphocytes.

In the centre of this mass there may be one or more large cells, with numerous nuclei peripherally placed. Tubercle bacilli have been demonstrated in the interior of these cells.

It is interesting to note that a condition such as the one described can be produced by the injection of dead bacilli. One of two things may happen to this small 'tubercle,' the result depending upon the virulence of the bacillus on the one hand and upon the resistance and reaction of the tissues of the host on the other.

Passing through a stage of coagulative necrosis it may become dense and fibroid, and thus be locked up in tough tissue, or it may proceed to the characteristic lesion of tuberculosis—namely, caseation.

This condition of caseation, except in a very imperfect degree, cannot be produced by the injection of the dead organism, the toxins produced by the living bacillus being necessary for this further development of caseation.

We have therefore two processes proceeding side by side:

Fibrosis—the protective and conservative.

Caseation—destructive and progressive.

In the portions of lung tissue actually diseased it is noteworthy that the blood-vessels disappear, no new ones being formed.

However, in the adjacent areas of lung parenchyma, especially in the more acute forms of the disease, there is definite congestion both of the alveolar walls and of the bronchial tissues.

The gray and yellow nodules—avascular in themselves, but frequently surrounded by congested lung tissue—may increase in number and size, coalesce and form solid masses, or be scattered throughout the lung substance. They may soften and break down, producing cavities, or be enveloped in fibrous tissue.

The manner in which these pathological conditions can affect the physical signs depends largely upon their site, and it would be well to remember that the smallest bronchioles are most frequently involved, with the result that their lumen becomes diminished and possibly occluded—the disease may then spread to the alveoli, which become filled with epithelial cells and small-celled growth from the thickened walls. In the more chronic form of fibroid tuberculosis the disease beginning in the finer bronchi tends to extend to the peribronchial tissue, and, associated with fibrosis, a complementary hypertrophy of the lung parenchyma takes place.

The local action of the bacillus is generally first in evidence towards the apices of the lungs. It is generally accepted that infection is more usual through the air passages, and a focus once being formed, the process extends either by dissemination through the air tubes, by the blood stream or the lymphatics. Sometimes it is the abnormal physical signs in the lung which arrest our attention, at others, certain symptoms and the presence of constitutional disturbances lead us to suspect a lung lesion, and we then examine the chest for corroborative signs. The absorption and distribution throughout the system of the toxic products of the bacillus give rise to pyrexia, wasting, anæmia, and night sweats, with languor, debility, and inability for prolonged exertion. These inform us of the activity of the process, and therefore are most valuable in assisting us to form an accurate prognosis.

If active tubercular changes are taking place in a lung, there is almost always some pyrexia; rarely however, the temperature is subnormal.

The pyrexia is irregular, usually showing an afternoon or evening rise, and easily increased by exercise. The thermometer is invaluable in the early diagnosis of phthisis.

Phthisis rarely develops without symptoms. Frequently its onset is insidious, and those symptoms which are present are neglected or considered to be due to some transient and trifling cause.

Atonic dyspepsia in a young adult, accompanied by anorexia and a dislike for fats, especially if there be a steady loss of weight, should always be viewed with suspicion, and a careful examination of the lungs should be made.

If there be also a short ineffectual cough, the necessity for overhauling the chest is still more urgent.

The same urgent need exists where there is debility associated with loss of weight, and a tendency to sweat at night with occasional shiverings.

More frequently, however, the onset is associated with symptoms which definitely point to some trouble in the lungs or respiratory tract—as cough, hæmoptysis, pain in the chest, dyspnœa, or hoarseness.

Cough.—Cough is almost invariably present as one of the first signs of early phthisis; it is probably in some cases due to the congestion of the lung, in others to an accompanying catarrh of the smaller bronchi, or to pleurisy. Early involvement of the larynx may be the exciting factor.

When present, the cough is usually short and ineffectual; it may, however, result in the expectoration of small masses of mucus. One cannot over-estimate the importance of a careful enquiry as to the existence and character of a cough in these cases; a negative history would undoubtedly lend weight to a diagnosis from which phthisis was excluded. There must be many cases in your minds in which cough was the early outstanding symptom.

I remember a medical student, who remarked to me one day that if anyone ought to fear phthisis, he ought, for his family history was about as bad as it could be. He looked the last man to contract tuberculosis—short, thickset, an athlete, played half-back for his team, and was an oarsman. All through the summer, though apparently in the best of health, he suffered from a short occasional dry cough, to which he gave no heed until suddenly, and without warning, he had several attacks of hæmoptysis.

Another medical man, a personal friend, who suffered from

phthisis and is now cured, told me that for a whole year before it was recognised that he had pulmonary tuberculosis, he was troubled with the short superficial ineffectual cough.

There is no necessity for me to quote more cases to emphasise the value of this peculiar quality of cough as an indication of importance.

Hæmoptysis, if genuine, is of paramount importance in the history. In the earlier stages it may occur before there are any demonstrable physical signs in the lungs, and when associated, as in the case above described, with a history of a short cough, is almost pathognomonic.

It is rarely profuse in these earlier stages, sometimes being simply streaks of blood in the expectoration; at others, frothy bright red blood—the quantity not, as a rule, exceeding two ounces in the twenty-four hours.

When the blood appears as streaks in the sputum the hæmoptysis ceases after a few hours, and leaves no trace in the sputum; as a rule, however, when there is a more free hæmoptysis it lasts for a day or so, and then for the next few days the sputum is stained with altered blood.

Hæmorrhage from the lungs in pulmonary tuberculosis may be due to one of several pathological conditions:

Hyperæmia of the parenchyma or bronchial mucous membrane.

Softening of a small focus of disease and opening into a minute vessel.

Ulceration of a tubercular patch in the bronchial mucous membrane, or a small limited quiescent lesion which had formed insidiously, may suddenly be the cause of the rupture of a small vessel.

I do not consider it necessary to enumerate the many other causes of hæmoptysis, but one should always bear in mind mitral stenosis. It is the more important as the lungs in such a patient may give evidence of a slight consolidation with some alteration of the breath sounds; sometimes even apical crackling râles may be heard.

There is also frequently a cough in severe mitral lesions.

Fortunately on careful examination a typical case of mitral stenosis is as a rule easily recognised, but there are cases in which the typical bruits are absent, and the diagnosis has to be made on the quality of the heart sounds and a careful inspection of the præcordium with accurate definition of the deep cardiac dulness. The small volume and irregularity of the pulse in mitral stenosis would assist in the diagnosis.

Pain and tenderness in the chest may be of no moment, but, on the other hand, sometimes indicate pleurisy—the pleurisy giving rise to the first subjective and objective signs of the disease.

Dyspnœa is rare in the earlier stages, but I have met with it in patients who worked in an atmosphere of flint dust.

In these patients the respirations were between 30 and 40 per minute before there were any obvious physical signs of disease in the lungs. The dyspnœa was constant, and accompanied by a most troublesome, persistent short and ineffectual cough.

The progress of the disease was rapid—the dyspnœa persisting throughout.

Tachycardia [1] without pyrexia, but associated with constitutional symptoms, is occasionally the first symptom noticed.

In these cases the toxæmia is so profound that the circulatory centre is attacked; later the respiratory centre is involved, giving rise to an accompanying tachypnœa.

Patients suffering from apyretic tachycardia go steadily from bad to worse.

I had such a case under observation for some months in the beginning of the year. The pulse was constantly at or above 120 per minute—the temperature being normal throughout, although there was a progressive lesion at the right apex.

When first seen the physical signs were slight, and his

[1] Vialard, "De la tachycardie du début de la tuberculose," *Bull. gén. de Thér.*, vol. 145, p. 277. 1903.

medical attendant had given a favourable prognosis in spite of the ominous apyretic tachycardia. The patient was dead in a few months.

Another form of onset to be remembered is the 'influenzal.' It is now well recognised. In the *Liverpool Medico-Chirurgical Journal* for March 1901, Dr Buchanan records twelve cases in which there was a history of influenza as the first sign of illness.

It is to be noted that—

(1) The initial symptoms of tubercular infection may be diagnosed as an attack of influenza.

(2) The patient may have actually suffered from an attack of true influenza, which has so debilitated him that he has become infected with tuberculosis.

(3) Influenza may have lit up a quiescent lesion into fresh activity.

Whatever its manner of action we should attach great weight to a history of a previous attack of influenza.

Without having attempted to exhaust the modes of onset, I have briefly touched on some of the more important.

Let me emphasise, before beginning the discussion of the physical signs, that in all cases of doubtful diagnosis the family and past history of the patient is of the greatest importance.

Phthisis is a house- and factory-bred disease, and we must enquire for possible sources of infection. Is there anyone suffering from chest trouble in the office, workshop, or in the house? Is the patient's bed-fellow healthy? These and similar queries may often give valuable information.

One may take it that the majority of town dwellers are exposed to infection even if they do not become infected, and that the really important matter is rather one of soil—the personal factor. It is essential, therefore, to find out if the patient's vitality has been reduced by alcoholic excess, by starvation, by mental worries, over-work, or over-lactation; in fact, by any cause of debility and lowered vitality which might render him an easy victim to tuberculosis; for if you cannot

digest the bacillus it will digest you. The sputum must be examined for tubercle bacilli in every case where there is the slightest ground for suspicion.

Frequently—and sometimes in cases where one least expects it—the examination of the sputum yields positive results.

A certain number of patients present themselves in the pre-bacillary stage, or there may be no expectoration; in such cases the diagnosis must and ought to be made irrespective of the sputum.

I intend now to devote the remainder of our time to the diagnosis of pulmonary tuberculosis, based on the physical signs found on examining the chest.

Your suspicions as to the nature of the case have been aroused by some of the symptoms already described, and you turn to the chest for confirmation or the reverse.

In considering what value we are to place on physical signs in the diagnosis of phthisis, we must bear in mind—

1. That the localisation of physical signs is of much greater moment than their quality.
2. That it is a safe plan to "attach much weight to the presence of certain signs, little weight to their absence." [1]
3. That it is always unwise to base a diagnosis on any one physical sign.
4. That one frequently meets with very definite variations from the normal in perfectly healthy chests.

The recognition and full appreciation of physical signs can only be acquired by constant examination, frequent palpation and percussion both of the healthy and diseased chest, till one has become quite at home with the normal variations from the normal standard.

It necessitates the highest training of the fingers and ears to perceive the earliest variations, and a judgment trained by long experience and constant practice to discriminate in each case between the important and unimportant physical signs.

It is advisable to have the patient stripped to the waist,

[1] Lindsay, *Lungs and Heart*, p. 129.

seated or standing in a good light, the light coming preferably a little from one side.

Carefully inspect the chest, noting the general shape and conformation, whether it be a long, sunken, alar chest, or broad, deep and emphysematous. The former is more likely to be the chest of a tubercular patient, though tubercular disease is not infrequently found associated with general emphysema.

Notice any hollowing about the clavicles. It is most important to notice whether expansion is good and equal.

Is there lagging or delay in the movement of any part? The apices should be carefully observed for this.

One must not forget that basal conditions, such as thickening of the pleuræ or the presence of fluid may cause an over-action at the corresponding apex.

This over-action being taken for normal sometimes gives rise to the inference that the normal, and therefore less active, apex is diseased. This false inference may find support on auscultation, as the breath sounds will in all probability be weaker over the less active normal apex.

Diminution or delay in the movement of the chest can be estimated by placing the hand on the infraclavicular regions of the chest, the variations from the normal being felt as well as seen.

The upper portion of the chest may be examined with advantage by looking down on the shoulders from above when the patient is seated in a chair.

Another method, and a most valuable one, is to stand behind the patient and place the hands on the shoulders close to the neck, the fingers lying over the clavicles and touching the infraclavicular areas, the thumbs being tucked comfortably into the supra-spinous fossæ. Yet another method of examination is to grasp the upper portion of the chest by placing a hand in each axilla and the thumbs on the infraclavicular area. As the chest expands the fingers and thumb of each hand are separated, the extent of the separation being felt and any difference in expansion between the two sides easily appreciated.

In early phthisis diminution of expansion or some lagging at the diseased apex is not infrequent, the functional activity of the diseased lung being much diminished.

Alteration in expansion is sometimes more marked and of considerable value in diagnosis in a quiescent lesion, where compensatory hypertrophy or emphysema may have masked any loss of percussion resonance, and auscultation gives little or no assistance.

Of course, with extensive consolidation there may be serious and obvious loss of expansion, but there is then no difficulty in diagnosis.

I think hardly enough attention is paid to the total possible expansion of the chest as measured at a level of the fourth costal cartilage.

After excluding cases of emphysema, one notices the frequency with which early phthisis is associated with an extremely small range of variation between inspiration and expiration, the amount often not being greater than half an inch.

The next step is to test the *vocal fremitus*. Here the normal variation is marked, and in the large majority of cases is greater on the right side.

Squires,[1] in *The British Medical Journal*, states that 86 per cent. of men and 77·5 per cent. of women show a dextral excess. One should also remember that the "excess of the right fremitus is physiologically greater from the tenth to the twenty-fifth year"[2]—in other words, the normal variation is more marked during adolescence.

In women it is frequently difficult to obtain a satisfactory fremitus; it is then advisable to ask them to sing the sound "ōō" in as bass a voice as possible.

As a rule the vocal fremitus is everywhere greater on the right side than the left over any symmetrical portion.

If, therefore, we find the vocal fremitus equal at the two apices, it suggests the possibility of disease, while sinistral

[1] Squires, *B.M.J.*, p. 1242. 1904.
[2] Berkeley, *Med. News*, p. 1010.

excess is practically always pathological. As a rule the vocal fremitus is not of much aid in the earlier stages, but is looked upon more as a sign corroborating other physical signs.

Percussion, on the other hand, frequently yields most valuable information.

The portions of the chest to which greatest attention must be paid are the following:—the supraclavicular areas covering the extreme apex of the lung, the infraclavicular areas in their outer third, the clavicles themselves *in their outer half*, and also the supra-spinous fossæ.

The apex of the axilla, including, as it does, the first and second spaces, may repay examination.

Frequently, while the apex of an upper lobe yields only indefinite signs, examination of the apex of the corresponding lower lobe may reveal some impairment of note; this combination almost invariably signifies tuberculosis.

The examination of the apices of the lower lobes is most simply carried out by requesting the patient to place each hand on the opposite shoulder, and then percussing down the chest internal to the vertebral border of the scapulæ, comparing one side with the other. Occasionally disease at the apex shows itself on mapping out the upper limit of the lung on the neck, the level on the diseased side being at a lower level than that of the healthy.

In comparing one side of the chest with the other, exactly corresponding areas should be examined. All the muscles should be relaxed and the patient breathing easily.

Light percussion is the most valuable, but heavy percussion is occasionally of value in the examination of the supra-spinous fossæ, more especially in muscular or stout individuals.

Direct percussion, using all four fingers of one hand as a plessor, assists in the appreciation of the extra sense of resistance occasionally met with as an evidence of disease at the apex, even when the percussion resonance is only very slightly or doubtfully impaired.

This direct percussion frequently gives positive results,

not only by affording an increased sense of resistance, but also by a definite impairment in the percussion note, even in cases when no alteration in the note can be elicited by the ordinary methods.

When in doubt as to variation in percussion resonance, I have frequently found the following method of assistance:—

Leave the binaural stethoscope in position in your ears, the tubing and chest piece hanging free; then gently percuss the suspected area, and compare the note produced with the one elicited from a corresponding area on the other side of the chest. The difference, if any, between the two is then more easily appreciated, the lower-pitched elements in the sound being filtered out.

As a normal variation one finds the percussion note slightly raised at the right apex in about 50 per cent. of healthy chests. This occurs more frequently in men than women, and must be borne in mind when basing conclusions on variations in percussion resonance.

Auscultation.—The most valuable of all methods of examination is, in my opinion, auscultation, and there are some authorities—such as Sir Isambard Owen[1]—who advise a reversal of the usual method of examination, and suggest that it is wiser and speedier—after a cursory inspection of the chest—to begin with auscultation; then, having obtained a lead as to the site of the disease, to proceed to careful inspection, palpation, and percussion.

The first thing necessary is to see that the patient is able to breathe satisfactorily.

Many persons, when asked to take a deep breath, think it necessary to open their mouths and respire in a loud noisy manner, or to make spasmodic movements of their chest, while their diaphragm remains more or less fixed.

It is useless to auscultate until such individuals have learnt to breathe through their nose slowly, quietly, and if desired, deeply.

[1] *B.M.J.*, p. 766, April 2, 1904.

Quantity.—When auscultating, first determine the intensity of the breath sounds; later, the quality.

Is less air entering the apex than is normally the case? Are the breath sounds weakened? If so, what may be the pathological condition causing this diminution in the normal intensity of the breath sounds?

Tubercle bacilli may have lodged in the bronchial mucous membrane and caused thickening and consequent obstruction to the entry of air, or the bronchi may be completely blocked with secretion and swollen diseased mucous membrane. Another possible cause is the congestion already referred to as occurring in the more acute cases. This congestion interferes with and diminishes the normal elasticity of the lung, and the breath sounds are consequently weakened. In the early stage of pneumonia the same phenomenon is met with.

A localised condition of emphysema, or thickening of the apical pleuræ, may also diminish the normal intensity of the breath sounds. Weakening of the breath sounds at an apex is frequently one of the earliest physical signs of phthisis, and is of more value when noticed at the right apex than the left, on account of the greater intensity of breath sounds normally present at the right apex.

On the other hand, instead of the diminution of intensity, the breath sounds may be louder and harder over the diseased area. According to J. K. Fowler[1] this harsh breathing precedes the feeble, and one may find feeble breathing below the clavicle accompanied by moist sounds after coughing, while in the second and third spaces the breathing is harsh, and free from moist sounds.

Lindsay disagrees with the above, and holds that either harsh or feeble breathing may be the earlier sign.

The feeble breathing that one hears occasionally in the more advanced cases is probably due to obstruction or blocking of the bronchial tubes, or to a lesion arrested in an early stage and the formation of emphysematous bullæ and puckerings of the lung.

[1] Fowler and Godlee, *The Diseases of the Lung*, p. 374.

Cogwheel breathing is sometimes an indication of early phthisis, but is more frequently caused by irregular muscular contraction due to nervous conditions.

Normal or vesicular breathing is described by Fowler as follows: "It is a soft rustling sound, audible during inspiration. It is 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."

If the quality of the breath sounds varies much from the normal, and if this variation be localised to an apex, one must infer some pathological condition.

Expiration may be prolonged, and instead of being lower pitched may become of the same or even higher pitch than inspiration—a pause may appear between inspiration and expiration at the expense of inspiration.

Breathing such as the above, especially if heard at the left apex, would be strongly suggestive of commencing infiltration.

I say 'at the left apex' advisedly, because it frequently happens that one finds, especially in young women, a small area below the right clavicle where the breathing is as described above, the chest being normal.

When such breathing exists in a more marked degree, and the expiratory murmur is blowing in character, of increased and continued intensity, and of a pitch higher than inspiratory, you have typical bronchial breathing such as one hears on listening over the seventh cervical spine. The lung is then past the early stage, and one is dealing with definite consolidation.

Faint bronchial breathing is occasionally met with at an apex, and is generally caused by consolidation with some obstruction to the entry of air, or the consolidated tissue is some little distance from the surface of the lung.

The normal variations, which may be marked, are more frequently met with in women than men, and generally take the form of some prolongation of expiration of a blowing character at the right apex, and most intense in the infra-

clavicular area. Here, as already noted, the breathing may be bronchial in character, even in a healthy lung.

Adventitious Sounds.—Adventitious sounds, when present, are of perhaps more actual help in the diagnosis of the early condition than any other class of physical sign.

Frequent and careful examinations may be required before they are elicited, and may I emphasise again that it is the localisation rather than the quality of the physical sign that is of value, though the quality in itself may sometimes be almost pathognomonic, as, for example, the 'clicking râle.'

A slight pleural rub at an apex one may consider as a sign of tubercular disease, double apical pleurisy still more so, and a double diffuse dry pleurisy should always be looked upon as a most suspicious condition.

Crepitations.—'Crepitations,' and here I refer to the fine crackling sounds simulated by the rubbing of hair between the fingers, are occasionally heard as one of the first signs of phthisis. They are recognised towards the end of inspiration, and are probably due to a congestion of the alveolar walls having caused a certain stickiness. The alveoli are distended by the entering air and the sudden separation of the alveolar walls causing the fine crackling sound.

The first stage of acute pleurisy and the congestion stage of pneumonia may give rise to a similar sound.

If there is a catarrhal condition at an apex or a tubercular focus breaking down, you will generally find râles, and less frequently rhonchi, their quality and size depending, of course, on the condition of the lung surrounding the tissue in which they are produced, and also on the calibre of the bronchus or bronchiole.

A persistent catarrh localised at an apex, even without impairment of percussion resonance, is usually tubercular.

Softening of the tubercular focus first occurs in a few cases at that portion of the upper lobe just above the intralobar fissure where the lung is covered by the axillary wall. Auscultation in this region will then reveal it.

The râles associated with mitral disease have been mentioned.

The swallowing of frothy saliva or mucus causes sounds simulating râles, heard in the interscapular region. This should not be forgotten.

Crepitations may be met with along the margin of the cardiac dulness; they are of no moment.

Emphysema, with or without pleural adhesions, gives rise at times to fine or medium-sized crackling râles, chiefly inspiratory, but also expiratory. They sound as if close to the ear, and are not affected by coughing, remaining fairly constant; occasionally somewhat localised, more frequently scattered and diffused.

During the examination of a chest never forget to ask the patient to cough, and also to take several deep inspirations.

How often, when quiet breathing reveals little or nothing abnormal, a cough is followed by a shower of râles on inspiration, or just that little significant click so valuable in the diagnosis of early lesions.

It is wise to have some routine procedure with regard to this, such as always asking the patient to cough after testing the vocal resonance.

I well remember watching a Canadian friend examining some chests. He was the superintendent of a large State sanatorium, and part of his routine during the examination was, at a certain point, to address each patient thus: "Say wan, tew, three. Corf."

Another and valuable method of auscultation is the direct, or listening at the patient's half-open mouth.[1] Râles, rhonchi, and crepitations can then be heard, some of which are much less audible by stethoscopy. If this be the case, it points to a lesion situated probably in the finer bronchioles, and surrounded by relatively healthy pulmonary parenchyma. Their presence in the case of a patient suspected of a bacillary infection will confirm in an absolute manner the diagnosis of a tubercular pulmonary affection.

[1] Veillard, *Rev. méd. de la Suisse Romande*, p. 350. 1904.

Vocal Resonance.—The vocal resonance varies with the condition of the lung and bronchi, consolidation and patent bronchi producing bronchophony, occlusion of the bronchi or thickening of the pleura diminishing the vocal resonance.

It is normally louder at the right apex, where one may find bronchophony occasionally, even in healthy chests.

It is of value in the diagnosis of quiescent or deep-seated lesions associated with emphysema. Here there may be no marked dulness, possibly no impairment of percussion resonance, and yet bronchophony may be present accompanied by râles and altered breath-sounds. This association of signs should always suggest a tubercular lesion, possibly one of some standing.

Many men with a large practice find it impossible to devote sufficient time to examine every chest case as thoroughly as I have suggested. I would remind them of a motto given me some thirteen or fourteen years ago by an old Scotch fisherman in Kincardine: "Noo, laddie," he said, "let me gie ye a worrrd o' advice—a' through life keep yer eyes peeled an' yer ears cockt."

I hand on the advice to those about to examine a chest. As to method, I would suggest, after a rapid but careful inspection of the bared chest, and having considered the general appearances and tendencies of the patient, that they should at once auscultate those areas already mentioned as important, not omitting the apices of the axillæ. Follow this with palpation, in order to estimate the general and local expansion during respiration.

Then rapidly percuss the *outer half of each clavicle*, the supra- and infra-clavicular areas and the supra-spinous fossæ.

If these procedures yield negative results and the expansion is good and equal, the probability is that there is no pulmonary tuberculosis.

If, as the result of the examination, your suspicions are roused or strengthened, it is imperative that you proceed to a full and complete examination.

If after a prolonged and careful examination there is still a doubt, I would suggest the following course :—

Note your findings on a chest chart. Instruct the patient to keep a record of his morning and evening temperature, also to record it after exertion. Have the patient weighed every week. Examine the morning sputum frequently, and finally obtain the opinion of an X-ray expert both as to the movement of the diaphragm and also the question of any opacity or shadow at an apex.

At the end of a month again examine the patient's chest, and compare the result with the findings charted on the previous occasion.

Then with the data of the month's records, you will in all probability be in a position to give a definite opinion.

In conclusion I would like to point out that I have considered chiefly the question of the diagnosis of phthisis as it arises among patients who are up and about and able to come to one's consulting room or out-patient department.

There are many other cases where difficulties of diagnosis may and do arise in acute forms of the disease, or in the periods of convalescence; such I have not attempted to consider.