A Lecture

ON

THE DIAGNOSIS OF PULMONARY TUBERCULOSIS.

Delivered at the Medical Graduates' College and Polyclinic.

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LECTURE II.

My first lecture* on the diagnosis of pulmonary tuberculosis was devoted to the conditions of tuberculous consolidations, discrete and in masses. I now propose to discuss the diagnosis of excavation in its various aspects, to point out what is the true significance of certain well-known physical signs, and to note how far they are to be depended on when we attempt to track out the course of the disease. Excavation sometimes takes place in a lung on a small scale without giving rise to any signs or symptoms. Small tubercles may caseate, soften, and, coming into connexion with a minute bronchus, be removed by expectoration. But softening, if it involve any considerable-sized tuberculous mass, or, if the mass be superficial, gives rise to unmistakable physical signs.

The process of softening in its simplest form appears to be a necrobiosis of the alveolar wall from the results of the epithelial proliferation caused by the irritation of the tubercle bacillus, and possibly, as Mr. Watson Cheyne thinks, owing to a chemical change in the cells themselves produced by the tubercle bacilli; where the process is on a larger scale there is more or less ulceration and masses of tubercle undergo caseation, soften, and are expectorated. Therefore caseation may be partly the result of mechanical pressure, and partly of the chemical process of the cells, and the liquefaction of the caseous mass, which is true softening, is only a further step of disorganization.

The process of softening is often, but not always, accompanied by pyrexia, by increased cough, and by a change in the character of the expectoration, which becomes more abundant and purulent, and is occasionally blood-streaked. It often contains the brownish specks which are held to be characteristic of lung tissue.

Microscopically, large quantities of lung tissue are found either in the form of hooklets or even of entire transverse sections of alveoli. Tubercle bacilli are generally very abundant, and sundry other organisms, such as streptococci and staphylococci, and the micrococcus tetragenus, are found.

The physical signs of softening generally consist in the replacement of smaller and scattered crepitation by coarser and larger crepitation, as *râles*, and then comes the ominous click sound on cough so characteristic of softening, followed by the croak and the loud tubular sound, which soon passes into the various modifications of the cavernous sound, and these become very distinct after coughing.

Where catarrhal sounds are present, as in some forms of lung tuberculosis, the sounds distinctive of softening are often masked, and cavernous breathing becomes apparent almost as a surprise, generally after a severe fit of coughing. The sonorous rhonchus disappears, and a loud croak is audible. Where the area of softening is small, the percussion sound is unchanged, but, if a large cavity is forming, dullness often gives rise to limited resonance, probably owing to the large masses of tissue being removed and replaced by air. The percussion sounds which a recently-formed cavity gives rise to depend partly on its distance from the chest wall. The well-known cracked-pot sound is not an index of the size of the cavity, but indicates that it is superficial, and generally immediately underlying the spot struck, and to evoke it properly the mouth must be kept open. In many cases, where the cavity lies deep and there is much infiltration around it, or the pleura is greatly thickened, the percussion sound over the region of the cavity is dull. Inspection of the chest wall often discovers retraction where a cavity has formed, and this is to be best seen in the first or second intercostal spaces. Sometimes considerable flattening of the chest wall takes place, which generally indicates that a cavity of some size has formed and is beginning to contract.

With regard to the relations between the size of the cavity and the percussion note, undoubtedly large amphoric cavities,

when superficial, give a hyper-resonant note and have been mistaken for a pneumothorax.

But to return to the subject of softening. Generally speaking, lung tubercle softens in the order of its formation, and therefore excavation is commonest and earliest in the upper portion of the upper lobes, but the auscultatory sounds of softening are best heard in the first and second interspaces and not in the suprascapular regions, where the sounds are often not very clear. Another region of search should be the interseapular, if the march of tuberculosis has advanced beyond the interlobular sulcus.

As I have stated, softening need not necessarily be accompanied by many constitutional symptoms, but it is invariably attended by some increase of cough and expectoration. The prospects of the patient when a cavity has formed cannot be considered so favourable as if there be only tuberculous consolidation, as a suppurating surface suitable for the breeding of the tubercle bacilli is at once established, and the chance of reinfection and spread of the disease is largely increased. The physical signs of a cavity being so familiar need not detain us long, but the caution as to not coming to any conclusion as to the absence or presence of a cavity without making the patient cough, is as important as in the first stage of phthisis. Often over large tracts of the thorax where resonance and absence of breath sound are all that can be detected, a good cough will discover dry or moist cavernous sounds, the reason of the former silence being that the bronchus leading to the cavity was blocked by a plug of mucus which a sharp cough displaced, and this may account for the cavities which are from time to time discovered in the *post-mortem* room and have escaped detection during life. The loudness of cavernous sounds is not always an index of the size of the cavity, as superficial caverns, even of a comparatively small size, if surrounded by much consolidation, give rise to louder cavernous sounds than deeper cavities of large size which are not so surrounded.

Tuberculous cavities vary not only in size and situation, but also in kind; there are secreting cavities with smooth surfaces which look like lining membranes and are not so, but from whose interiors a large amount of matter exudes; there are dry cavities whose walls are principally composed of fibroid material and contain little or no secretion, and are probably the forerunners of the caseous masses embedded in fibroid tissue and unconnected with bronchi, which are so often found at necropsies. There are the primary cavities of the ordinary type, and the secondary cavities of which more presently. Basic cavities are also a division, and these may either be primary or secondary; and there are the contracting cavities which we always are on the look-out for by reason of their happy augury; and, lastly, there are the amphoric cavities, arising from such extensive lung excavation that the organ is often converted into a mere bag of pleura. Let us now consider them *seriatim*.

DRY AND SECRETING CAVITIES.

The secreting cavity is generally found in connexion with acute forms of the disease, as in acute tuberculo-pneumonic phthisis, and often extends rapidly. The secretion consists of the detritus of the softening process, broken-down lung tissue, abundant pus cells, and tubercle bacilli. The walls of these cavities are often the seat of much inflammatory and ulcerative process, and the rapid laying bare of the blood vessels and their perforation by erosion often gives rise to large hæmoptysis.

The dry cavity is generally the result of chronic disease and remains in a stationary condition for months, and often for years, giving rise to no symptoms beyond morning cough and expectoration. The presence of a large amount of fibrosis will often convert the dry cavity into the contracting cavity, but in many cases, owing to pleuritic adhesions or the absence of the fibroid element, the dry cavity will remain patent for long periods, in fact for years. In many of these patients the disease is unilateral and limited to one lobe, and the discharging cavity resembles a sinus from a tuberculous lesion of the bone. On the walls of these dry cavities are often to be noted aneurysms of the pulmonary artery branches, the rupture of which often causes death in cases which have appeared promising.

SECONDARY CAVITIES.

These are a class which Dr. William Ewart was the first to describe and explain, and my experience amply confirms his conclusions. Secondary cavities are formed through the re-inhalation of the secretion or sputum of primary cavities.

This is partly the result of the prone position of the patient in bed and of weakness of expectorating power, and partly owing to the suction power of the middle portions of the lung being stronger than that of the apex. Hence the secretion from an apex cavity, instead of being discharged through the wind-pipe and mouth, descends into an adjoining bronchus, reaches the terminal bronchioles and alveoli, and sets up fresh tuberculous disease in the neighbourhood. A cuboidal mass of tubercle is formed as the result of the infection, which again breaks down and is expectorated, and thus another cavity is formed. These secondary cavities follow the lines of certain bronchi, principally where the suction power on coughing is greatest, and the principal secondary cavities are formed in the dorso-axillary region, the mammary regions, and at the base of the lung, and are remarkable for being devoid of lining membrane. The dorso-axillary region is the commonest spot for a secondary cavity to commence in and should be carefully examined in chronic cases, but a large number of secondary cavities are also detected in the mammary region. The basic cavities are not so common. Secondary cavities are unfavourable to a good prognosis, as they seldom contract.

AMPHORIC CAVITIES.

In some patients tuberculization is followed by so extensive an amount of excavation that the presence of air in the cavity gives rise to the phenomena of amphoric breathing and metallic tinkle, which latter is the echo of drops of fluid falling into the cavity from its walls and bronchi.

These cavities are diagnosed from pneumothorax by the absence of the fluid in the pleura and its characteristic signs, and by the different position of the auscultation sounds, though there are cases of localized pneumothorax where the diagnosis is very difficult and often impossible.

CONTRACTING CAVITIES.

The phenomena these give rise to may be traced step by step by physical signs. Cavities contract sometimes rapidly, and I have known considerable sized cavities contract in three months, but as a rule the process is a slow and gradual one, depending on the amount of fibrosis in the lung and the reacting power of the patient against the ravages of the tubercle bacillus. The chief signs of a contracting cavity are:

1. Flattening of the chest walls overlying the cavity, and often contraction of the chest circumference of the side.
2. Displacement of neighbouring organs.

When the contracting cavity is on the left side, the left lung is retracted towards its root, and the right lung becomes hypertrophied, and is drawn across the median line of the sternum, and may be traced through percussion by a line of resonance reaching half-way across the clavicle. The heart is raised, its apex being tilted towards the axilla, and sometimes it describes an arc of a considerable sized circle, of which the centre is the fixed portion of the heart and arteries. The heart's impulse may be felt first in the fourth space to the left of the nipple, and even traced into the third space, always tilted towards the axilla. The stomach is drawn up, and the note is to be distinguished quite high up in the thorax.

When the cavity is in the right lung, the heart is generally drawn to the right and under the sternum, the liver is raised, and the left lung is drawn across the median line, and reaches half-way across the clavicle, or even further. The lung on either side contracts towards a fixed point, sometimes towards a spot where the pleura is adherent, but more commonly towards the root of the lung; and the cavernous sounds, at first becoming less distinct in the first and second interspaces, become more so in the suprascapular and interscapular regions, where they linger some time, and gradually disappear. Cough and expectoration may cease, or the latter sometimes becomes fetid from retention through narrowing of the bronchus. Occasionally the contraction of cavities causes even greater changes, and the shoulder of that side is depressed, the spine being curved towards the contracting side. The worst of it is that contraction of the cavity in one lung does not prevent the formation of fresh tubercle in the opposite lung.

DIAGNOSIS OF CAVITIES.

The diagnosis of tuberculous cavities from bronchiectasis is based on several points of distinction.

First, on the age of the patient and on his different appearance in the two diseases. Children and middle-aged people are liable to bronchiectasis, but as a rule not young adults, which is the class most frequently attached by tuberculosis.

Patients with bronchiectasis, except when suffering from septicaemia, show no sign of wasting or anaemia, but generally have a healthy exterior and are commonly gaining flesh.

Secondly, on the appearance and examination of the sputum, which in the case of bronchiectasis is frothy and mixed with pus and more or less offensive, and, though containing numerous septic organisms, is free from the tubercle bacillus.

Thirdly, on the character of the cavernous sounds and their locality. The majority of bronchiectatic cavities are basic, whereas tuberculous cavities are generally at the apex. Bronchiectatic cavities may, however, be detected in the first and second interspaces, where they are always nearer the median line than tuberculous cavities, and though they give rise to noisy, jarring, cavernous sounds, they are never audible over such extensive surfaces of the thorax as in tuberculous cavities. The percussion sound is not as a rule dull, but resonant from the accompanying emphysema.

The Harveian Lectures

ON

THE TREATMENT OF SOME ACUTE VISCERAL INFLAMMATIONS.

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LECTURE I.

ACUTE RHEUMATIC CARDITIS AND PERICARDITIS.

The treatment of acute cardiac inflammation is, in the great majority of cases, the treatment of an infection with the organism of acute rheumatism, and we have to consider what therapeutic agents, general and local, are available to assist us.

First and foremost, we must determine the value of sodium salicylate as an internal remedy in acute rheumatism. It is believed by some that the value of salicylate in the treatment of rheumatism is limited to the reduction of temperature, the relief of pain, and the diminution of arthritis. But a very short experience in the wards of a hospital for children suffices to show that this view is quite inadequate. For in the rheumatism of childhood arthritis is often absent altogether, and pain may be slight, while cardiac disease is severe. Yet the good effects of salicylate in children are quite as well marked as in adults. The temperature falls, the general condition improves, torpidity gives place to a bright expression, and the cardiac state is ameliorated, the area of dullness tending to diminish, and the strength of the ventricular contraction to increase.

Observation of the effects of salicylate in the treatment of acute rheumatism both in children and in adults has led me to the conviction that it is as truly antirheumatic as quinine is antimalarial or mercury antisymphilitic. But it must, of course, be given in adequate doses and its use must not be too soon relinquished. For an adult 20 gr. of sodium salicylate with 40 gr. of sodium bicarbonate should be given every two hours during the day and every four hours during the night. For a child of 6 to 10 years old 10 gr. of salicylate and 20 gr. of bicarbonate at the same intervals; this amounts to a daily dose of 100 gr. of salicylate. After a day or two 15 gr. of the salicylate with 30 gr. of bicarbonate may be given, and if necessary this may be increased to 20 and 40 gr. respectively, the total daily amount of salicylate being 150 or 200 gr. Children require proportionally larger doses than adults, for in them the rheumatic infection is much more intense. It is rare for an adult to die from his first rheumatic attack, but this disaster is by no means uncommon in childhood. Fortunately children usually bear salicylate well; in them it seldom causes much vomiting, often none at all, and they hardly ever complain of the deafness, tinnitus, and headache which are troublesome in adults, nor do they often manifest the mental symptoms and the tendency to delirium which are sometimes caused by the drug in later life.

There is, however, one effect occasionally produced by salicylate which demands special mention, for it is a sym-