Clinical Lectures

ON

CERTAIN DISEASES OF THE CHEST.

Delivered at the Liverpool Northern Hospital.

BY

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LECTURE II.—PNEUMONIA (*continued*).

BEFORE I proceed to speak of the treatment of pneumonia, I wish to say a few words in reference to the early physical signs of the disease, and the morbid conditions by which they are produced.

The general symptoms and signs which characterise the onset and progress of pneumonia are so well described in your various systematic works, and will be so frequently illustrated by the cases which I shall have to detail, that I shall purposely abstain from any regular description of them here. I must, however, refer at some length to a phenomenon which I have noticed, and about the existence of which there is some difference of opinion.

I mentioned in the last lecture, that I am of opinion that engorgement is not the earliest morbid condition of pneumonia; and I also believe that crepitation is not the earliest physical sign of the disease. Crepitation is the auscultatory sign which characterises the stage of engorgement, and practically is the first sign on which you can depend as indicating the existence of pneumonia. I shall have to speak of it again, and point out to you that it may be heard when no pneumonia is present.

But, of the earliest morbid condition; I agree with the conclusions arrived at by Dr. Stokes, that there is a stage prior to that of engorgement, characterised by dryness, intense arterial injection, and, consequently, a bright vermilion colour, of the pulmonary membrane. In proof of the probability of this condition, I must appeal to the facts furnished by auscultation; viz., the existence of a harsh, loud, puerile respiratory murmur, preceding the crepitating *râle*.

It is very rarely that an opportunity is afforded us of making an examination of the chest in incipient pneumonia; and to this fact we must, I think, attribute the differences of opinion which have been expressed as to the earliest physical signs of the disease.

I have had two cases under my care in this hospital, in which I noted the existence of a loud, harsh respiratory murmur as an initial physical sign of pneumonia. In both cases, there was acute primary pneumonia occurring in lungs previously healthy. I think it is important to note this; for, to render the observation of this particular phenomenon perfectly trustworthy, it ought to be made on a case, not where there is progressive inflammation, nor yet where there are consecutive attacks of inflammation, for the cause of the phenomenon might, under such circumstances, admit of some doubt; but where, the lung being in a healthy condition, inflammation of the organ comes on suddenly. Let me refer you to the following cases.

CASE I. P. F., a carter, was admitted into the hospital, under my care, on August 8th, 1864. On the day of admission, at an early hour, he was out in a shower of rain, got very wet, and did not change his clothes. In the course of two or three hours, he felt pains about the limbs, and had severe rigors.

When admitted into the hospital about midday, he was seen by the house-surgeon. He then complained of pain in the lower part of the left side. There were no febrile symptoms, and no abnormal physical signs about the chest.

On the following day, about noon, his condition was as follows. The pulse was 120, and full; respirations 32; skin very hot and dry; tongue coated with a white fur. The pain in the left side had increased. There was no cough, but much dyspnœa. The percussion-sound and movement of the left side of the chest were natural. *At the lower and back part of the left lung, a loud, harsh, peculiar respiratory murmur was audible.* No such sound could be heard elsewhere. The patient was ordered a grain of opium three times a day, with small doses of tartar emetic.

The next day, the pain in the side was almost gone. The pulse was 104; the respirations were 28. The physical signs were as follows: deficient movement of the left side, dulness at the left base, with crepitating *râle* over the lower half of the left lung. The crepitating *râle*, which was distinctly of a pneumonic character, occupied, in fact, this day, the seat of the harsh loud respiration of the preceding day.

It is needless to follow the history of the case further. The crepitation was succeeded by bronchial breathing and all the symptoms of confirmed pneumonia. The patient made a satisfactory recovery, and was convalescent on the eighth day of the attack.

CASE II. D. M., a Frenchman, was admitted into the hospital, under my care, on January 23rd, 1865. Two days before admission, he was perfectly well. He complained of dyspnœa and pain in the chest. On examination, *a loud harsh respiratory murmur* was heard over the lower and back part of the left lung. The movements of the side were good, and there was no dulness. The breath-sounds over the opposite lung were normal. On the following day, the physical signs were as follows: slight dulness at the base of the left lung, and well-marked crepitation over about the lower half of the same lung. In fact, as in the preceding case, the loud respiration of one day was replaced by the crepitating *râle* on the next. The patient subsequently had all the symptoms of confirmed pneumonia—dulness, bronchial breathing, and rust-coloured sputa. He was convalescent about the tenth day.

From the observation of these cases, I cannot entertain the slightest doubt that neither is the crepitating *râle* the earliest physical sign of pneumonia, nor engorgement its first morbid condition. It is true that I have never been able to demonstrate, by a *post mortem* examination, the dryness of the pulmonary membrane and the arterial injection, which I believe to exist prior to the stage of engorgement; nor, indeed, would it, I think, be easy to satisfy the minds of those who are sceptical on the subject by any such examination; for they might consider the appearances the result of mere congestion. At the same time, this absence of *post mortem* proof must not blind us to the facts which clinical experience teaches us.

As I have already mentioned, there is much difference of opinion as to the existence of this phenomenon; but before I speak of the objections which have been brought forward against the possibility of its occurrence, I wish to explain the way in which, I believe, this harsh respiration is produced, and to point out to you the condition in which I suppose the pulmonary membrane to be; and I shall take this opportunity of explaining to you what I consider to

be, 1. The cause of the ordinary respiratory murmur; and, 2. The cause of the crepitating *râle*.

First, as to the respiratory murmur:—Various causes have been, from time to time, assigned for its production; and although, in a practical point of view, its exact seat and proximate cause may appear unimportant, provided we are familiar with the sound itself, and can rightly interpret the modifications of it which result from disease, yet it must be confessed that clear views of the physical phenomena of all healthy organic actions are very desirable; and just as our knowledge of the simple manner in which the sounds of the heart are produced has facilitated our diagnosis of cardiac diseases, so more precise information than that we already possess, with regard to other points of a similar nature, cannot fail to be followed by beneficial results.

To the physical condition of the lung it is obvious that we must look for an explanation of the cause of the respiratory murmur; and there is one anatomical point, either unknown to those who have given their attention to this subject, or overlooked by them, which appears to me to offer a satisfactory solution of the phenomenon.

Without attempting to examine critically the opinions of others, I must content myself with observing that I believe the air-sacs of the lungs to be the seat of the murmur; and I shall now proceed to point out the arrangement which exists at the mouth of each air-sac, to which arrangement I am of opinion that the sound is due.

I have pointed out elsewhere the manner in which each bronchial tube terminates in a series of air-sacs; and the passage which has the most important bearing on the question of the cause of the respiratory murmur is the following.

"The air-sacs consist of somewhat elongated cavities, which communicate with a bronchial ramification by a circular opening, which is usually smaller than the cavity to which it leads, and has sometimes the appearance of a circular hole in a diaphragm, or as if it had been punched out of a membrane which had closed the entrance to the sac."

This arrangement is best seen in the lungs of children and of adults. In old age it has frequently disappeared, more or less. It may be often well seen in a piece of lung, the blood-vessels of which have been injected with coloured size, and which, after being dried, has been subsequently soaked in spirit. By careful dissection under a microscope the membrane, guarding the mouth of the sac, and narrowing the entrance to the cavity, is easily demonstrated. The membrane forms a part of the aërating walls of the air-sac, and has branches of the pulmonary artery ramifying in it.

It is obvious that a condition of this kind must have an influence on the passage of the air into the air-sac; that, to a certain extent, it must produce an impediment to the current of air, and thus give rise to a sound.

As the air is moved along the bronchial tubes it meets with no obstruction to its passage; but at the commencement of the air-sacs an opening exists which is smaller than the cavities between which it is placed. As the air-sacs expand with each inspiration, air must pass through the constricted opening. I believe that, in the passage of the air through this opening, the main element of the respiratory murmur consists.

The following facts appear to me to afford arguments in favour of the view I have advanced: the respiratory murmur is loud and well marked in infancy and childhood; it becomes modified in adult age, and in old age it is frequently very feeble. In the infant the membrane placed at the mouth of the air-sac is well marked and uninjured; the opening in it has a clearly defined and sharp margin; and, moreover, it is smaller—not only absolutely, but I believe also relatively—than in after life. In the adult, the air-sacs have undergone enlargement, and the membrane at their entrance is more or less perfect according as the lung is in a more or less healthy state; whilst in old age, the membrane has often, to a great extent, disappeared, apparently as the result of the wasting and absorption which so frequently occur in the lungs of those advanced in life.

Further, the changes which take place in the character of the respiratory murmur in emphysema of the lungs afford an additional argument in support of this view. In this disease, in consequence of distension, rupture, and absorption, the air-sacs become much altered in character, and the membrane guarding the entrance to them entirely disappears as the disease progresses. The obstacle to the passage of air is therefore removed; and hence one reason of the extremely feeble respiratory murmur which characterises the affection.

And now let me explain to you the way in which, I believe, this healthy respiratory murmur passes, first of all, into the harsh puerile respiration of incipient pneumonia, and subsequently into the crepitating *râle*, when the disease is fully established. It appears to me that the first phenomenon, which is merely an exaggeration of the healthy sound, is the result of the dry and swollen condition of the pulmonary membrane; that this gives rise to a constriction of the mouths of the air-sacs, and approximates them, therefore, to the condition which they present in childhood, when a loud respiratory murmur is usually heard. I see no reason to doubt that there is a dry stage in pneumonia, as well as in inflammation of mucous membranes. It is said that every stage of inflammation of serous membranes is marked by exudation; and it has, therefore, been inferred that such must be the case in pneumonia. But, although the lining membrane of the air-sacs resembles to a certain extent a serous membrane, yet it does not possess all the characters of such membrane. It consists, as I have already mentioned, of some yellow elastic fibres, a very delicate basement membrane covering the blood-vessels, and a layer of epithelium having somewhat the character of the epithelial cells found on serous membranes, but being by no means identical with them.

It has been objected to the view that there is a puerile respiration preceding the crepitating *râle* in pneumonia, that the sound which is thus described is nothing more than the result of a supplementary movement in parts around a spreading obstruction; that when this sound is heard, and afterwards is followed by crepitation, there has been, at the time when it was heard, consolidation of the lung in adjacent, more deeply seated, portions. I think that the circumstances under which the sound was heard in both my cases negative the possibility of such an explanation of it. Take the first case. The patient is admitted at noon on the 18th of August, having got wet early in the morning, previously being in good health. He is carefully examined, and nothing abnormal is found about the chest; nor is there any fever present. It will scarcely be inferred that pneumonia was present at that time. Twenty-four hours afterwards, he is again examined. There is a good deal of fever present; the respiration is hurried; and there is pain in the chest. There is no dulness; but a harsh respiration is heard over the back of the left lung. Now, is it at all probable that, during the short period that had elapsed since the man's attack, consolidation of the lung could have occurred—espe-

cially taking into consideration the subsequent progress of the case? For, after the lapse of twenty-four hours more, we have the stage of engorgement established in the more superficial portions of the lung, but no consolidation. I need not refer to the second case, for it presents features similar to those of the first.

I feel convinced that, in the two cases which I have detailed to you, this harsh respiration was an initial symptom of pneumonia; and, although it may not be a constant precursor of the crepitating *râle*, I believe it would be much more frequently met with, if we had more opportunities of auscultating our pneumonic patients in the early stages of their disease.

But now, as to the manner in which the crepitating *râle* is produced: I believe that its seat is in the air-sacs, and that it is caused by their expansion at the time when their walls are covered with the secretion which is poured out upon them. The expansion of the sacs at the time when they are partially filled with fluid appears to me to afford the conditions necessary for the production of the *râle*. That it has its seat in the finest bronchial tubes, I cannot admit; for in some cases these tubes are found after death free from exudation.

There are conditions under which the crepitating *râle* may be heard when no pneumonia is present. In certain cases of œdema of the lung, I have heard a crepitation as pure as anything I have ever heard in the most typical pneumonia; and trusting, therefore, to this sign alone, you might in some cases be misled as to the nature of the disease; but, generally speaking, there is no difficulty. The ordinary symptoms of pneumonia are absent in these cases; there are dropsical effusions in various parts of the body, and other conditions which enable you to form a correct diagnosis. Still some cases are very puzzling, and at first are apt to mislead us; such, for instance, was that of Scott, who died in L. Ward, and who, whilst in the hospital for valvular disease of the heart and dropsy, was seized with pneumonia. When I first heard the crepitating *râle* in this man, I thought it was the result of œdema of the lung; and it was only when other symptoms and signs developed themselves, that I became sure of the existence of pneumonia.

You may perhaps ask me how it happens that we hear the same sound in œdema of the lung as in pneumonia. The fact is, that the seat of exudation in the two diseases is the same; and in both conditions we have present, in the air-sacs, a certain amount of air and liquid exudation; the only difference being, that in one instance the liquid is somewhat more viscid than in the other.

RATE OF MORTALITY IN NEW YORK. Dr. Harris, the registrar of vital statistics, states in his report to the New York Board of Health that the number of deaths in the year 1866 was 21,206; this would make the death-rate about 34 to 1,000, which is greater than that of London, and double what is considered a normal rate in England. Nearly one-half (43·73 per cent.) of all the deaths are of those under five years of age, amounting to 10,123; while 29·51 per cent. are of those in the first year of their existence. Dr. Harris states that there is little doubt that of the 2500 children born alive each year, death takes nearly one-third before they reach their first birthday. In New York, one child is lost for every 75 or 80 of the population. There is no such infant mortality known anywhere in the Christian world; and, the registrar observes, it is considered the most sure indication of the growing insalubrity of the city.—*New York Paper.*

ON THE

INTERNAL USE OF TARTAR EMETIC IN ACUTE INFLAMMATIONS.*

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A LITTLE more than three months ago, I attended a lady, comparatively young, in her eighth labour. She had recently come to Bath, and it was therefore the first labour in which I had attended her; but she gave a history of severe and even dangerous floodings after nearly every childbirth, and this time was no exception to the rule. Life seemed in peril for an hour or two, but she ultimately rallied completely, and at the end of forty-eight hours she did not appear much injured by the great loss of blood.

Now, in all cases of unusual *post partum* hæmorrhage, it is well to be watchful for events of a pyæmic kind. These consist either of what is called pelvic cellulitis, or of diffuse inflammation of the breast; the latter is probably more common, and is erysipelatoid in its suddenness and activity. And it is highly philosophical to speak (with Dr. Barnes) of inflammation of the breast occurring very soon after labour, as essentially a form of puerperal fever.

On the afternoon of September 5th, exactly fifty-eight hours after delivery, the patient whose case is my present text began to show signs of mammary inflammation on the left side. Late at night, or about six hours afterwards, I was summoned to see her on account of the violence with which this inflammation had set in. Before leaving home, I pondered what I should do. Calomel was out of the question; saline purges seemed inapplicable by reason of the recent hæmorrhage; nauseating doses of antimony appeared no less improper; and if any one has faith in belladonna for curing these cases, I pity his credulity. In quiet despair I took up the fourth edition of Dr. Churchill on *Diseases of Women*, and on page 752 I found an apposite quotation from Dr. Beatty, who says that, on the accession of inflammation in the breast, he has given one-sixteenth of a grain of tartar emetic every hour, with the result that in ordinary cases the pain and fever are mitigated, and the breasts are smaller and softer. He says that these doses may induce slight nausea, but never or very rarely free vomiting. Dr. Churchill ratifies Dr. Beatty's opinion by saying that tartar emetic, given in this form, has a more powerful effect in abating inflammation of the breast than any medicine he has ever tried.

Armed with this knowledge, but slightly sceptical as to its entire truth, I visited my patient, and discovered that acute lobular inflammation of the breast had vehemently set in, and was marked by all the usual symptoms. Fifteen drops of tartar emetic wine (one-sixteenth of a grain) were ordered to be given in half a wineglassful of water every hour through the night, until 11 o'clock the next morning, a period of exactly twelve hours. Nothing local was applied, except a piece of hot wet flannel covered with oiled silk.

After twelve doses of this medicine, administered with unfailing punctuality, it is no exaggeration to say that the inflammatory hyperæmia was almost gone; the breast was only a little more swollen than the other, and there was scarcely any pain. It is pleasant to add, that there had been not only not the

* Read before the Bath and Bristol Branch, Dec. 13th, 1866.