Clinical Lectures

DELIVERED AT

CHARING CROSS HOSPITAL.

BY

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LECTURE VII.—ON PLEURISY.

Serous Inflammations point to Blood-poisoning.
Case 1. Prognostic Value of Change in Physical Signs. Ægophony dependent on Thickness of Fluid. Ægophony remaining after Absorption of Fluid; its Explanation. Increased Depression of Inter-costal Spaces in Receding Hydrothorax. Ægophony not generated at Edge of Fluid.

THE subject of to-day's lecture, gentlemen, is one which I think very well illustrates the general truth that Pathology is subordinated by Physiology—that the latter dominates the former—that the circumstances that determine the diseases to which a part is liable, and the particular kind of morbid agencies to which it is obnoxious, are the physiological anatomy of that part, and the function which it fulfils. Parts that are physiologically analogous are affected with similar morbid tendencies; and thus a natural physiological scheme becomes a natural, and the only natural, nosological one.

If I were asked what was the commonest morbid cause, which, above all others, had a tendency to generate serous inflammations, I should say, without hesitation, some state of blood-poisoning. We have serous inflammation from uræmia; we have serous inflammation from pyæmia; we have serous inflammation from the specific toxæmias of the eruptive fevers; we have serous inflammation from the presence in the blood of the *materies morbi* of rheumatic fever; and in the cases before us we have serous inflammation from a condition which I shall show you presently is, essentially, a veritable blood-poisoning.

The cases to which I wish to direct your attention are cases of pleurisy; by which we mean, as you know, inflammation of the membrane that lines the cavity and covers the viscera of the thorax. One case is that of a man lying in bed No. 8 of Robertson Ward; the others those of a young man and young woman whom we have seen among the out-patients, and who have refused to come into the hospital.

CASE 1. George Franklin, aged 32, by occupation a wine-cellar-man, engaged in washing, corking, and packing bottles in a damp cellar; married; has had two children; is what he calls moderately temperate—that is, is not frequently drunk, but thinks he averages a bottle of wine a day, which his occupation gives him great facilities for obtaining;—in fact, the men engaged in his business are not limited as to what, or how much, they drink, as any attempt at restricting them would be in vain. His health has always been good, with the exception of severe colds, to which he has for some years been liable, and which he attributes to his

occupation, which exposes him to great alternations of temperature, throwing him into sweats from the loads he carries, and then plunging him into cold damp cellars to pursue his amphibious occupation of bottle-washing.

Six weeks ago, in this way, he caught one of these colds, was feverish, with pains in his back and limbs, and suffered from cough, spitting, and shortness of breath. He worked on, however, through it for a week, but was then obliged to give over; and, after applying to a hospital and obtaining no relief, went home, took to his bed, and placed himself under medical treatment. In a fortnight he was sufficiently well to resume his employment, though very weak. On returning to his work, he was seized with stabbing pains in his right side, and an increase of the shortness of breath he has suffered from from his first cold. The pain was not constant, but only occasional, and was chiefly brought on by exertion and attempting to take a deep inspiration. It was as if two knives were plunged into him, one beneath the right floating ribs, between them and the crest of the ilium; and the other just to the right of the right nipple. The pain was so sudden and so severe that it would cause him to cry out, and bring him short up, whatever he might be doing. In the morning he could hardly get out of bed on account of this pain, and, when out, it was some time before he could get himself upright. He went on working for ten days in this way, and then was so bad that he applied to this hospital, and was admitted, eleven days ago—that is, last Friday week, May 8th.

I did not see him until the following Sunday afternoon, two days later, when, happening to be in the Ward, my attention was attracted by a man who was constantly yelling out and starting with pain. This man was the patient, and this was my first introduction to him; I found that he was placed under my care. I immediately set to work to discover what was the matter with him, and found I had not far to search. From his rapid breathing, and from the temporary stoppage of his breath that each plunge of pain produced, it was quite clear that his malady was respiratory. I at once stripped him, and discovered the following physical signs:—Absolute dullness over the whole of the back of the right lung; no respiratory sound whatever in this situation; no vocal fremitus; voice-sound distant and hardly audible, but not otherwise modified; no ægophony. In front on the same side, the dullness extended as high as the fourth rib, up to which situation the voice was ægophonic; above this point percussion was resonant, and the voice-sound natural or slightly bronchophonic; but there was hardly any breath-sound of any kind. On the left side, the breathing was in every way natural, except that it was compensatory, and that at the lower margin of the lungs behind there was a fringe of moist subcrepitus. Decubitus dorsal; can lie neither on the right side nor on the left. Expectoration small in amount, and large bronchial in character—firm grey mucus. Cough but slight; pulse 115; respiration 31, superficial; tongue dry; thirst; no appetite; physiognomy anxious and exhausted.

The physical signs left no doubt that the right pleura was full of serum, the result of acute pleurisy, from which the man was still suffering. I ordered him ten minims of tincture of opium, the same quantity of chloric ether, and a grain of quinine, every four hours; turpentine fomentation to the right side and back night and morning.

May 11th. Much the same; complains of feeling light-headed, and of seeing a lot of people sitting down and talking to him whenever he dozes. Pain a trifle better. A trace of ægophony could be heard at the top of the right lung behind.

May 12th. Pain decidedly better. Ordered to take the sedative only three times a day, instead of every

four hours as before. On examining the right lung behind, a considerable change is found to have taken place. Ægophony is heard over nearly the whole of it, passing at the apex into bronchophony, and at the base into loss of all voice-sound; bronchial breathing of flat pitch, characteristic of hydrothorax, coextensive with it, and the dullness of percussion considerably reduced in the supraspinous region. Crepitus at base of left lung increased. Dyspnoea less urgent.

May 13th. The patient has now had on five turpentine fomentations—on Sunday night, Monday morning, Monday night, Tuesday morning, and Tuesday night; and so effectually have they acted that they have taken the skin off his back. The pain is nearly gone, but still gives him occasional stabs when moving or taking a deep breath. Still feverish and semi-delirious if he dozes. Physical signs much the same.

On the 15th, the report of the physical signs was as follows:—Percussion dull in the lower two-thirds of the right side behind; ægophony from the base of the lung to a point opposite the posterior border of the scapula, midway between the inferior angle and the base of the spine; breathing highly bronchial coextensive with the ægophony; on the left side, respiration above compensatory, below accompanied by slight moist sounds.

On the 16th, the report is, that the other physical signs being much the same, the level of the limit of ægophony has receded nearly to the inferior angle of the scapula. Compound iodine ointment to be rubbed into the left side of the back night and morning.

On the 18th, the report is, that, the area of dullness is rather less, and that the level of the limit of ægophony has subsided still lower. Pulse 90; respiration 24. Ordered—the opium to be reduced from ten minims to five, to be taken with two grains of quinine and half a drachm of compound sulphuric ether three times a day.

May 20th. The bronchial breathing of the affected side has nearly disappeared, and in its place normal respiratory murmur is gradually emerging. In the lower part of the lung, there is still no breathing. Percussion-dullness undiminished. The ægophonic twang not so marked, but its area undiminished—distinctly heard as high as between the lower angle and spine of the scapula. Vocal fremitus still abolished below, but exaggerated in the upper fourth of the lung. The signs in front are the same as behind, but at a lower level. In his general condition, the man is improved in every way. Pulse 84; respiration 20.

There are several interesting points in relation to the physical signs in this case.

1. The *order* in which these physical signs have changed, and the prognostic value of these changes. When I first saw this man on the Sunday afternoon, there was a general negation of everything on the right side of the chest—no heart-sound of any kind, no vocal fremitus, no voice-sound, and dullness absolute and everywhere, except at the apex in front. Soon, however, the voice-sound has reappeared, but found to be of a peculiar character, having a bleating twang which we call *ægophony*; respiratory sound is again audible, but it is a tube-sound, and not a parenchyma-sound, and shows merely that there is a certain air-tide in the bronchial tubes, and not that the spongy lung is re-inflated; at the apex vocal fremitus has reappeared, while percussion-dullness has receded from it. Still later, true respiratory murmur has emerged from its temporary obscuration, while the tube-sound that had supplanted it has vanished; nevertheless, the percussion-dullness has hardly appreciably diminished, and the ægophonic twang, though less marked, and the absence of

vocal fremitus, still continue. Thus, as far as this case has progressed, we may divide the physical signs into three stages:—

First Stage.

Percussion—Absolutely and universally flat.

Palpation—No vocal fremitus.

Voice—Distant, hardly audible; no ægophony.

Breath—No breath-sound of any kind.

Second Stage.

Percussion—Still dull over lower three-fourths of chest.

Palpation—Fremitus reappearing above; none below.

Voice—Ægophony well developed.

Breath—Bronchial blowing of flat pitch.

Third Stage.

Percussion—Dullness receding, but very little.

Palpation—Fremitus exaggerated above, still none below.

Voice—Ægophonic twang less strong.

Breath—Respiratory murmur reappeared. Bronchial breathing nearly gone.

Now what do we learn by all this?

We learn, in the first place, that an absence of normal sounds indicates a worse state of things than a modification of them; that the worst of all physical signs are negative ones; that a total absence of voice-sound indicates a more aggravated condition than ægophony, and a total absence of breath-sound than bronchial breathing. When I listened to this man's chest on the Sunday afternoon the serous effusion was so extensive—the stratum of fluid between the lung and the parietes of the chest so thick—that no breathing sound could penetrate it, and probably the lung was so pressed upon that very little air entered it at all, and so no breath-sound was produced, even if it could have been conducted to the surface. But by the Tuesday, two days later, so much of this fluid had been absorbed that the lung had regained a good deal of its lost volume, and was able to follow the respiratory movements of the thoracic parietes. Thus the respiratory tide was re-established in the bronchial tubes, while the stratum of fluid surrounding the lung was so thinned that it was able no longer to isolate the sounds within the lung, but conducted them with a peculiar modification to the chest-wall. Just the same explanation applies to the appearance of the ægophony three days after the man's admission. On the Sunday afternoon, with all the signs of abundant serous accumulation within the pleura, there was no ægophony; on the Tuesday, when the physical signs indicated a diminution of the effusion, ægophony was strongly developed. Hence we learn that the absence of ægophony does not show the absence of pleuritic effusion, but, on the contrary (if other signs exist) asserts it more strongly than its presence, and that the very excess of the condition to which the sign points extinguishes the sign. Thus, ægophony, in a case of hydrothorax, instead of being a bad sign, may be a good sign—a *redux* sign, pointing towards a restoration to a state of health; and there can be no doubt, in the case of this man, considering the physical signs he presented on the Sunday, that the appearance of the ægophony on the Monday, and its full development on the Tuesday, was the very best sign that could possibly have shown itself.

We learn too, I think, from this case, that there is a certain thickness of the stratum of fluid that

gives rise to a maximum development of the ægophonic twang. The sound was most strongly developed at a certain zone, above which it diminished and below which it diminished—above, as the stratum of fluid got thinner, fading into the natural voice-sound with a slight bronchophonic character; below, as the stratum of fluid got thicker, becoming fainter and more distant, but still retaining the ægophonic character. As the effused fluid has been gradually lessening in quantity this zone of maximum ægophony has gradually descended, because the point at which the stratum of fluid was of the exact thickness for its highest development has also gradually descended. What is the thickness of the layer of fluid that most powerfully generates this peculiar sound I cannot say, and to determine it would be a problem of very difficult solution.

From what I have just told you, we see that normal breath-sound obliterated by hydrothorax is restored *vis* bronchial breathing, and that normal voice-sound obliterated by hydrothorax is restored *vis* ægophony, just as normal breath-sound obliterated by hepatisation is restored *vis* crepitation.

Again, we learn from this case, if we look at what I have called Stage 3, that the respiratory murmur may be resumed, and therefore the lung have acquired its normal volume and inflation, and therefore the fluid from that situation have disappeared, and yet ægophony remain. Now what does this seem to show? That the presence of fluid is not necessary to the development of ægophony. To-day, as I have read to you in the report of the case, there is very fair vesicular breathing over the middle two-thirds of the affected side; in some parts so clear and strong, and so evidently immediately beneath the stethoscope, that it is impossible to believe that there is any fluid there, and yet at this very spot—yes, and even above it—there is unmistakable ægophony. How is this to be explained? Thus, I think: The pleural surfaces are generally covered, to a thickness proportionate, I believe, to the length of time the effusion has existed, with a gelatinous leathery coating of fibrine. When the effusion subsides there still intervenes a double layer of this gelatinous material between the lung and the normal elements of the chest-wall; and as this material is saturated with fluid, it may, I think, be sufficient to generate the ægophonic tremor. This is merely an hypothesis; but I think it not an unreasonable one. The necessity of adopting it turns, of course, upon whether the presence of ordinary respiratory murmur negatives the possibility of there being fluid over the seat of it. If it does, as I cannot but believe that it must, then a layer of free liquid is not necessary to generate ægophony. If, on the other hand, ægophony inevitably implies the presence of a stratum of free liquid at the point where it is heard, then you may have respiratory murmur of natural character beneath pleuritic effusion. On the showing of this case, there is no escape from one or other of these alternatives.

You might have observed in this man that not only was there an absence of that fulness of the intercostal spaces which is generally described, and which often exists in cases of abundant pleuritic effusion, but that during forcible inspiratory efforts the lower intercostal spaces of the affected side were conspicuously more drawn in than on the sound side. This arose from the resistance which the fluid con-

tents of the diseased half of the chest offered to the sudden enlargement of its cavity. In healthy breathing the rising of the parietes in inspiration is immediately followed by, or rather simultaneously accompanied by, the easy and unresisting expansion of the lungs. But when the pleural cavity is occupied by fluid, this easy enlargement of the thoracic contents in obedience to the inspiratory movements is no longer possible, because, in proportion to the serous accumulation, in proportion as the lung has been supplanted by fluid, in such proportion has an expansible been substituted by an inexpandible material. The effused fluid is absolutely inexpandible; the compressed lung no doubt retains a considerable degree of expansibility, for unless it did no air-tide could be generated, and not even bronchial breathing would be audible; but the expansibility is always greatly inferior to that of healthy lung, and sometimes, when the compression has been considerable and of long duration, almost annihilated. Thus, partly by the diminished expansibility of the lung, and partly by the composite nature of the thoracic contents, a drag is put on the inspiratory movements, and the intercostal spaces are sucked in, so to speak, at each inspiration.

You must be prepared then to find that not only will there often be no filling out or bulging of the intercostal depressions in cases of hydrothorax, but that they will be more depressed and conspicuous at inspiration on the affected than on the healthy side; and that thus not an effaced condition but an excessive conspicuousness of these spaces may point to pleuritic effusion. This is a sign that is necessarily more marked in a diminishing than in an increasing hydrothorax, and therefore is not without prognostic import.

The whole bearing of this case favours most strongly Laennec's theory of ægophony. With the single hypothetical exception I have mentioned, the one thing that seemed to regulate it was the presence and quantity of the fluid; when the effusion was too great in quantity there was no ægophony, when it diminished the ægophony appeared, when it got down to a certain standard the ægophony was at its maximum, and as it subsided the ægophony descended. And even supposing, as I have suggested, that a gelatinous coating of fibrine may be adequate to the production of the ægophonic quake, it seems to me no violation of Laennec's view, for such a material is mainly fluid, and may be capable of experiencing vibrations similar to those of a fluid.

This case also clearly shows that the ægophonic twang is not generated at the *edge* of the effused fluid, as some have supposed, for it was the most marked a good two inches, I should say, below the level which the physical signs pointed to as that of the edge of the serum.

[To be concluded.]

MEDICATED CIGARETTES. *Mercurial Cigarettes.* Dissolve three drachms of red precipitate in three drachms of nitric acid, and add enough water to make up the quantity requisite to saturate 100 slips of paper. They will contain about three grains of the nitrate of mercury. *Nitre Cigarettes.* Dip the paper in a saturated solution of the nitrate of potash before rolling. *Balsamic Cigarettes* are made by giving the dried nitre cigarettes a coating of tincture of benzoin.