Clinical Lectures

DELIVERED AT

CHARING CROSS HOSPITAL.

BY

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LECTURE VI.—ON CAPILLARY BRONCHITIS.

Comparative Rarity.—History of Case.—Peculiar Sputum.—Post-Mortem Appearances.—Points of Diagnosis from Ordinary Bronchitis.—Rapidity of Respiration.—Pulse-Respiration Ratio.—Relation of Moist Sounds to Sputum.—Resemblance of the Condition to the State of Animals Drowned.—Treatment: the Question of Alcohol and of Bleeding.

I wish to call your attention today to a case of bronchitis that has been recently under our observation. And perhaps you may wonder at my making a single case of so common a disease the subject of a clinical lecture. But this case was altogether of a different type and kind from the ordinary cases of bronchitis with which we are so familiar. This variety has been named *capillary bronchitis* from its extending to the smallest ramifications of the bronchial tubes, and was known to the last generation of doctors as *peripneumonia notha*. Bronchitis as it generally shows itself is a long way the commonest affection we have to treat. I do not think I am overestimating it when I say that, in the winter months, one-half of the cases that come under our observation are cases of bronchitis; sometimes the out-patient room presents us with little else. But although I have given clinical lectures here for seven years, this is the first case of this particular form of the disease that I have had an opportunity of bringing before you.

In one unfortunate respect the case is strikingly exceptional—its speedy and fatal termination. *Ordinary acute bronchitis does not kill*; of the hundreds of cases I have seen, I have never seen a patient die of it, except at the extremes of life; infants die of it, very old people die of it, and at all ages the chronic affection, which it often leaves behind it, proves largely fatal. This chronic bronchitis decimates the poor; it is a veritable scourge. But the acute affection, as I have said, does not kill, except at the extremes of life. Its tendency is to do one of two things;—either to terminate in perfect recovery, leaving perhaps only a greater tendency to the affection than existed before; or else to run on into the chronic form, of which the patient may, at some remote period, ultimately die. Whereas, we see in the case before us a healthy man, as is shewn by his nutrition, in the prime of life, coming into the hospital within two days of the supervention of his first symptoms, and in four days he is dead.

But besides its rapidly fatal issue, we shall see, as we proceed, that the case is peculiar and distinctive in many other respects—in its clinical history, its physical signs, and its *post mortem* appearances.

The history of the case is as follows:—

Francisco Rondi, aged 24, presented himself among the out-patients at the hospital on December 22, 1862. His face was pallid, his expression anxious and exhausted, and he was obliged to be supported into the room. His aspect indicated such depression that at the first moment of seeing him I suggested he might be going to have fever. His tongue, however, had not at all the fever character, and inquiry immediately showed that his symptoms were thoracic. His breathing was very much accelerated, and he had a constant short cough, which, without bringing anything up, gave him no pause or rest. This constant coughing somewhat embarrassed our auscultation. However, the signs were so plain and so pronounced that there was no misreading them: they were simply these. Breathing hurried and laborious; respiratory murmur clear but exaggerated at the apices, but from that situation downwards, everywhere, front and back, supplanted by an abundant, moist, crunching crackle, like squeezing a sponge half saturated with water, especially loud at inspiration, but also audible at expiration. At the upper boundary of this sound the respiratory murmur was heard through the crepitus, but as you descended it became less and less audible and the crepitation louder, which, in the lower half of the chest, drowned everything. It was clearly produced by the bursting of multitudinous bubbles, and resembled pneumonic crepitation in its character—in the idea that it gave—and in being mainly an inspiration-sound; but it differed from it in not being fine enough—it was decidedly coarser. It was manifestly produced by drawing air through innumerable capillary tubes occupied with fluid. There could be no doubt, then, that the man was suffering from intense and almost universal capillary bronchitis. I at once ordered him into the hospital. His pulse was rather above 120, not small. The respirations I did not count. Although the morbid sound was of so moist a nature, there was no expectoration—the short, incessant, hacking cough did not bring up a particle of sputum. No sternal pain. Percussion-sound normal. I should mention that the part of the lung that seemed the most free was the upper part of the left lung in front, where, from the clavicle to the fourth rib, the respiratory sound was almost natural except that it was strongly compensatory.

I ordered *atheris chlorici* ℞; *ammoniae carb.* gr. v; *tinctura scillæ* ℞xx; *decocti senegæ* ʒj; to be taken every four hours.

Turpentine fomentations to be applied to the chest, front and back.

This man being an Italian, I could get but little history out of him; but I ascertained that his illness was but of two days standing, previous to his application at the hospital, that it seemed to be the result of cold, and was ushered in by shivering and headache. Our patient is one of the unfortunate race of organ-grinders, and no doubt owes his attack to the mingled wet and cold which has prevailed lately, and to which his occupation has necessarily exposed him.

On going into the wards the next day, I found the patient evidently very much worse. He was manifestly in a state of impending suffocation; dyspnoea extreme—laborious and making sixty-eight respirations a minute; face dusky, or rather, deeply cyanosed; the conjunctiva even pervaded with the same leaden hue, turgid and suffused; lips purple. But he was lying quite flat on his back. I immediately ordered him a bed-chair and a shawl-blanket, and had him propped up in bed. This seemed to give him some slight comfort. On examining his chest I found precisely the same signs as on the day before, only still more pronounced, and the area of uninvaded lung still smaller—confined to a small part of the left apex in front. The same fine churning crepitation, mainly inspiratory, everywhere else—above and below, front and back. There was audible, however, now, in addition to the fine sound which alone existed

yesterday, a larger moist sound about the middle zone of the lung; indicating either that the larger tubes were implicated, or that the exudation thrown out in the smaller tubes was passing along the larger in its progress towards discharge. The action of the extraordinary muscles of respiration was very strong, the inter-sternomastoid and the inter-scalenal pits deeply sucked in at each inspiration. The same sucking in was seen at the lower part of the chest—scrobiculus and cartilages of the false ribs—in fact, along the entire attachment of the diaphragm. The expectoration had just, and but just, appeared: I was shewn a single pellet of stiff mucopus, attached to the bottom of the spittoon, as the only portion that had been spat up. The respiration, as I have mentioned, was 68, the pulse 136; the pulse-respiration ratio, therefore, was exactly 2. The pulse of good volume and strong. Urine scanty, intensely dark, but clear.

I ordered the mixture to be given *every alternate hour*, and the turpentine fomentation to be frequently applied, front and back.

Dec. 25th. Much worse; cyanosis still deeper; surface damp with perspiration; aspect heavier and more exhausted, at the same time more anxious; the dyspnoea was so intense that the patient could only get a word out now and then. The constant "hack" has ceased, and instead there is a frequent cough in short paroxysms, attended, on each occasion, with the discharge of expectoration. This free discharge has come on since my visit yesterday; I was shewn a spittoon containing half a pint of it, and the material is so peculiar as to deserve a particular description.

On taking up the spittoon I not only observed the frothiness of the sputum, but was struck with the lightness of the spittoon, considering how full it was. On pouring the material from one spittoon to another this was at once explained;—the whole consisted of nothing but a sort of foam, and was as spongy and full of air at the very bottom as at the top; it was like whipped syllabub, or that beaten white of egg and cream which they pour over trifles, only more ropy; but equally spongy, and aerated throughout. I have never seen any sputum exactly like it; generally, however frothy, the froth is confined to the top, and the lower strata are free from it: but this was not so. On pouring it from one vessel into another it "flopped" out in an elongated mass, leaving the bottom of the empty vessel as clean as if it had had nothing in it. There was not the slightest trace of rusty tinge about it. I regret to say I did not examine it microscopically.

Pulse 128, respiration 60; pulse-respiration ratio, therefore, 2:13. The pulse was voluminous, but flabby and compressible. In consequence of the man's increased weakness I ordered him, in addition the diligent maintenance of the previous treatment and great external warmth, an ounce of wine every alternate hour.

Dec. 26th. The man is dying. He sits supported in bed with his head thrown back, in a state of unconsciousness, and with a loud rattling respiration; eyes half-closed, pupils contracted; face darkly cyanotic, and sweating profusely, as is his whole body. All efforts at rousing him produce hardly any effect. The temperature has fallen, being at the groins 92° Fahr. He ceased to be able to swallow, to expectorate, to cough, or to articulate, early this morning, about eight hours ago; since that time he has been gradually sinking into a state of deeper unconsciousness. Pulse 120, respiration 44; pulse-respiration ratio 2:72.

Death took place at six o'clock P.M., four hours after the last visit.

Post-mortem examination, nineteen hours after death. On opening the chest, the first thing that struck me was that the lungs did not collapse; they maintained their full size in the living thorax; the part of the heart left uncovered did not exceed the natural region of precor-

dial dulness. The upper part of the anterior surface of both lungs presented an appearance of a slight general emphysematous enlargement of the air-cells, and the surface here was pale; lower down this distended condition of the parenchyma did not exist, and the surface was red and vascular—in some parts bright red. The pleural surface of the lungs was uneven, some of the lobules standing out in relief from the others, or rather, I should say, some of them not coming up to the pleural level; but there were none of them anything like "carnified"—all crepitated, and were full of air. On pressing the lung with the finger the impression remained, giving it a doughy, pitting character. On being withdrawn from the body the bulkiness of the lungs, and their refusal to collapse, were very conspicuous. Their posterior parts were seen to be of a chocolate colour, passing into a brighter red both forwards and upwards.

On cutting into the lung the whole structure was seen to be of a deep dull red, everywhere spongy, unusually so, and tolerably dry. Very little material escaped from the cut ends of the air-tubes, but on squeezing the lungs, a viscid frothy muco-purulent matter shewed itself at innumerable points. I apprehend that the reason why it did not flow from the tubes without pressure was from its great viscosity. On cutting open the larger bronchial tubes, they were seen to be of an even deep rose red, and this colour extended up into the windpipe, where, on a section, the redness of the mucous membrane, in its entire thickness, presented a strong contrast to the whiteness of the cartilaginous rings. The same deep redness extended down to the finest bronchial tubes that could be cut open. Nowhere was there anything like ulceration, or even abrasion of the surface.

On examining the white opaque froth that everywhere filled the bronchial tubes, large and small, it was found to consist of two materials only—pus-cells and columnar epithelium—suspended, of course, in a clear fluid (see fig.); but so numerous were these bodies that they covered the field with hardly any interspaces. The material was perfectly free from blood-cells.

Such is a picture, from beginning to end, as well as I can give it, of this fatal affection. There are many points of interest about it: let us see what they are.

In the first place, I would indicate to you the points of contrast between such a case as this and one of ordinary bronchitis—what would be called now-a-days, by a pleonasm, their points of differential diagnosis. These are of three kinds;—as seen in the clinical phenomena, in the physical signs, and in the *post mortem* appearances. The great distinctive clinical phenomenon was the intensity of the dyspnoea. The respiratory changes were suspended to a degree never seen in ordinary bronchitis: the man was blue from the day of his admission; death

by suffocation was manifestly impending over him from the time he came in till he finally succumbed. The clinical fact, too, of this rapid succumbing was of itself, as I have stated before, a distinctive clinical feature. Again, the sputum we must reckon distinctive; ordinary bronchitis would never yield such a material. The peculiarity of the physical sign (for there was but one) was; first, its character—that it was a capillary crepitation, non-pneumonic, as distinctive and as individual, in my opinion, as the true crepitation of pneumonia; secondly, its distribution—that it was not patchy or partial, but universal; and thirdly, its singleness—that it was this one sign alone, not, as one gets in common bronchitis, mixed moist and dry sounds of various sizes. The distinctive *post mortem* appearances were, universal non-collapse of lung, universal florid redness of the respiratory mucous membrane, and universal blockage with a viscid froth.

I think, too, that the point where the inflammation commenced and the course that it travelled were peculiar. We know that ordinary bronchitis travels downwards. The bronchitis of catarrh, of whooping-cough, of measles, always travels in this direction. A man catches cold to-day; to-morrow he has a sore throat; the next day he is hoarse; the next, he has sternal soreness, indicating an affection of the lower windpipe and large bronchi; the next, dry or moist sounds, showing that the affection has reached bronchiæ of smallish size. Now, if we may judge by the physical signs, the inflammation in this case would appear to have started in the capillary bronchules, for the first-heard sounds were seated in them; as the case advanced, the sounds became larger; and, on examination after death, we found that the inflammatory action had involved the whole of the respiratory mucous membrane, even into the windpipe. I do not mean to assert positively that the different parts of the respiratory mucous tract may not have been affected simultaneously; and the reason why the crepitus appeared in the smallest tubes first may have been merely because they were the smallest, and therefore the earliest infarcted with a sufficient amount of mucus to produce a moist sound; the larger tubes, from their wider calibre, requiring a more abundant exudation before they could be the seat of similar sounds. But the fact admits of another interpretation; viz., that the inflammation extended from below upwards, and that the moist sound registered its progress. At any rate, there was none of that "creeping down" which seems to be the law of ordinary bronchitic progress.

Another circumstance to which I would call your attention is the extreme *rapidity* of the respiration. On the 24th, it was 68 in a minute—a most unusual rapidity in an adult, and a circumstance that of itself would constitute a most unfavourable prognostic sign. I do not know the law of the rapidity of respiration in the different forms of dyspnoea, nor do I know that it has ever been worked out; but it seems to me to be, approximatively,—that the nearer the mischief is to the parenchyma of the lungs, the more is the breathing hurried. Thus pneumonia appears to present us with the most rapid of all forms of dyspnoea; capillary bronchitis, as in this case, gives us a high degree of acceleration; in ordinary bronchitis, the breathing is generally by no means much quickened; in asthma, I have often noticed it not at

all so; while in chronic laryngitis, I have seen the respiratory interval actually lengthened. In this respect, rapidity of respiration appears to be in inverse proportion to respiratory distress and violence of respiratory efforts, both of which I have noticed to be more intense and agonising the nearer the seat of the difficulty has been to the orifice.

Closely connected with this is the ratio existing between the rate of respiration and the pulse,—what has been called the pulse-respiration ratio. We know that in health we have four beats of the pulse, or four and a half, to each complete respiration; whereas, in this case, a complete respiration, consisting of an inspiration and expiration, was performed for every two beats of the pulse, although the heart was acting at the rate of 136 pulsations in a minute; or, if we reckon each respiration as a double act (an expiration and an inspiration being each a respiratory act), there was a respiratory act to every heart's beat, or 136 in a minute. In this case, then, the pulse-respiration ratio was on the 24th exactly 2, instead of 4.5. I am unable to offer you any facts as to the law of the variation of this ratio—why it should be very much disturbed in some pulmonary affections, and not at all in others. Nothing but close and prolonged clinical observation, directed to this particular object, will furnish the clue to such a law, supposing it to exist. I would only commend to your memories the clinical fact illustrated by this case, and mentioned by Dr. Walshe, that in capillary bronchitis the ratio of the respiration to the pulse is very high.

Let me also call to your attention, in connexion with this subject, the curious fact, that on three days, the 24th, 25th, and 26th, while the man was going from bad to worse, and the condition of the lungs daily aggravating, the rate of the respiration, and consequently the pulse-respiration ratio, was constantly approaching a more natural standard. Thus the respirations per minute, on these three days, were 68, 60, and 44; and the pulse-respiration ratio 2, 2.13, and 2.72 respectively. This falling of the ratio of respiration as the condition becomes more aggravated, and death approaches, has also been referred to by Dr. Walshe.* What can be the explanation of it? Why, as the state of the lungs gets worse and worse, the respiratory changes more and more in arrears, and the demand for air increasingly urgent, should the respiratory efforts become less frequent? I believe the reason to be, that the increasing carbonic narcosis accompanying the deepening asphyxia renders the patient less and less sensitive of the respiratory arrears, and therefore tends to diminish those respiratory efforts of which the sense of arrears, or, in other words, the respiratory distress, is the immediate stimulus. Whether the poisoning of the blood with carbonic acid could have any similar lowering effect on the heart's action, and thus account for the diminished rate of the pulse, I am not prepared to say. I have never directed my attention specially to the effect of cyanosis on the cardiac pulsations.

[To be continued.]

* Pulse-respiration ratios of 3.0, 2.5, or 2.35 to 1, are not uncommon; but sometimes, even as much as forty-eight hours before decease, the frequency of the respiration falls, while that of the pulse continues to rise; under these circumstances, I have known a ratio of 4.5 to 1 (that of health) coincide with a pulse beating 144 per minute.