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A COURSE OF LECTURES ON CLINICAL MEDICINE.

By W. R. BASHAM, M.D., Physician to the Westminster
Hospital.

LECTURE VI.

Case of Drunkard's Delirium, with pneumonia and bronchitis: History of the case; character of the delirium; stethoscopic signs; condition of the sputa; treatment: death; post-mortem appearances.—Tendency of drunkards to collapse; urgency of inflammatory symptoms, when supervening in such cases. Physical signs of pneumonia and bronchitis.—Pathological condition of pneumonia; cause of the rust-coloured sputa; progress of the disease.—Effects of bronchitis, complicated with pneumonia; discretion required in the treatment of such cases.

Gentlemen,—You have been present this day at the *post-mortem* examination of two cases of acute disease of the pulmonary organs, which, though they do not exhibit any uncommon features, are nevertheless of interest to those who are engaged in pathological studies; for every opportunity of making *post-mortem* examinations, affords us the valuable means of either confirming or disproving the diagnosis made during life; and what is still more important, we are presented with a view of the organic processes which constitute the disease, and learn the direction in which those organic changes proceed. The probable fatal or favourable issue of parallel cases is thus more easily determined, and the nature and direction of our therapeutical efforts more clearly defined.

R. H., aged 29, a green-grocer, had been drinking for the last three days, and during that time, in a continuous state of intoxication, was brought to the hospital, on the night of January 5th, at half-past twelve o'clock, by a neighbour. On admission into Burdett ward, he is reported to have presented all the symptoms of a case of *delirium tremens*. His manner was much excited; his answers to questions were hurried and indistinct; there was great trembling of the limbs, particularly of the hands; the tongue was tremulous, was quickly protruded, and as suddenly withdrawn; the mind rambling incoherently on a variety of subjects; the pupils were contracted, and the skin bedewed with a clammy moisture. He was ordered a dose of opium. In the morning, the report book states that he had been very restless all night; no sleep; much excitement and violence of manner;

so much so, as to render necessary confinement by the strait waistcoat. Suffers from an exceedingly troublesome and suffocative cough, with frothy sputa, stained with blood. He had been ordered opiate draughts every hour, which he had taken up to the hour of the daily visit. A careful examination of the chest quickly decided that a different and very energetic plan of treatment was necessary. *Auscultation*:—The left side was duller on percussion than the right, and particularly on the mammary and lateral region; throughout the right side the resonance was clearer. On the left side, no respiratory vesicular murmur could be detected; bronchial respiration was everywhere, but especially in the mammary region laterally; respiratory murmur was also detected here, accompanied by some moist mucous gurgling. On the right side, the vesicular murmur was veiled by the presence of bronchial respiration and mucous rhonchi, in the large as well as the small tubes. The pulse was 120, full and elastic, round and compressible; the tongue moist, but furred; the respiration 48; frequent urgent cough; pupils contracted to a point; manner very restless, and much incoherence. With these physical signs there could be no doubt that acute inflammation of the bronchial mucous membrane existed, combined with pneumonia of the left side, probably at the root of the lung.

Here was a fearful combination of evils to contend against,—a drunkard, intoxicated for more than three days, continuously an habitual drinker, suffering from acute pulmonary inflammation. Such individuals will scarcely, under any circumstances, bear depletory measures; nevertheless, in such a case as this, they are imperatively required, and you must be prepared to treat them with the cupping scarificator, and antimony, calomel, and opium, in one hand, and with ammonia and other stimuli, if necessary, in the other. Accordingly this patient was cupped to twelve ounces between the shoulders, a large blister covered the left side of the thorax, and Hydrarg. Chloridi, gr. j.; Opii, gr. j.; Antim. Pot. Tart., gr. ʒ, given every four hours. The pulse was directed to be watched; and if, after the cupping, it continued full and hard, a farther amount of blood was to be abstracted.

On the visit the next day, it was reported that the cupping had relieved the breathing and frequency of the cough, that the respiration had fallen from 48 to 40, and that the sputa were still frothy and sanguinolent. The pulse had improved in character after the cupping.

and was neither so full nor so hard as previously; the prognosis of the case, notwithstanding, still continued most unfavourable. At the afternoon visit the state of the chest was similar to that of yesterday; and owing to the blistered surface of the left side, could not be minutely examined. Bronchial respiration, with moist crepitations, were heard posteriorly on the left side; on the right side, mucous rhonchi were heard in each region. The sputa were copious, frothy, and of a more dusky dirty hue than yesterday; the tenacity was not very great, and they did not partake so much of the characteristics of pneumonia as of bronchitis. The pulse was 140, small and compressible; countenance anxious; eyes glazed; pupils contracted to a point; tongue moist; odour from the body offensive and cadaveric. The symptoms continued thus till the evening, when he changed, became comatose suddenly, the vital powers quickly failed, and he died at seven p.m.

Sectio cadaveris eighteen hours after death. Body well formed; limbs muscular, and rounded by subcutaneous fat. On opening the cavity of the chest the lungs did not collapse, but were adherent to the pleura costalis by firm attachments of fibrinous tissue; no evidence of the formation of recent plastic exudation: the right lung was dense and gorged, but crepitated throughout on pressure, and floated in water; no trace of fibrinous exudation could be detected in any part of this lung; when squeezed a white frothy fluid oozed out from innumerable openings of the more minute bronchi: the left lung crepitated only at its apex and the inferior margins; the central parts felt dense and carnified; its condition was such that it easily tore or broke down into a granular mass, and it did not float in water; this portion of the middle lobe was of a dark-brown, red, dirty colour, it constituted an example of lobular pneumonia in the second stage; the central portion of the hepatized mass was softer than the circumference, and appeared to be passing into the stage of purulent softening. Submitted to examination under the microscope granular corpuscles and blood-discs were alone observed; in some few of the former acetic acid developed central nuclei. The mucous membrane of the trachea exhibited a fine bright-red capillary injection; passing into the right bronchus the colour became more of a madder hue; and in the smaller ramifications of the tubes in this lung of a still more dusky appearance. The left bronchus exhibited much the same condition as the right; but the mucous membrane of the subdivisions had here become of a purple tinge, which colour was maintained even into the hepatized mass. The liver was much enlarged; its inferior margin extended to within a quarter of an inch of the umbilicus; a section exhibited a bright, sienna-orange colour; it had also a granular appearance; no evidence of portal congestion; under the microscope it presented innumerable fat vesicles; its weight was ninety-two ounces. The kidneys were healthy, presenting evidence of venous hyperæmia; right kidney weighed five ounces and a half; left kidney six ounces and a half. The brain presented no aspect of disease; its weight was forty-six ounces.

A prominent feature of interest in this case was the complication of acute pneumonia, with a condition which, if not strictly *delirium tremens*, was closely allied to it, being a state of great physical and constitutional disturbance brought on by the inordinate use of intoxicating liquors. The effect of alcoholic stimuli in excess, is at first to produce great cerebral excitement, with exaltation of the circulating powers; to this invariably succeeds a state of great prostration, and extending in some cases to collapse,—in fact, a failure of the powers and functions that have been thus unnaturally excited, is the usual characteristic of the drunkard's excess. Now, in the case under consideration, superadded to this dangerous tendency to prostration and collapse, were unequivocal symptoms of intense pulmonary inflammation, requiring the most energetic measures, and the unhesitating use of agents, which, without this pulmonary complication, the pre-existing state of the patient would most emphatically forbid. The prospect of a favourable issue was from the first extremely limited, for the pneumonia of drunkards is specially a fatal disease; they are proportionably more prone to the disease, and in them its progress is unusually rapid and uncontrollably fatal.

On approaching the bed-side of this patient on the morning after his admission, you witnessed his hurried and alarmed looks,—his restless, yet vacant gaze on those around his bed,—a certain amount of incoherence in his words, yet sufficiently conscious to protrude the tongue when asked; this organ quivering, furred, white and moist; the pupils contracted; and the hands, when released from the strait waistcoat which his violence rendered necessary, agitated and tremulous. He coughed frequently and had no sleep. You could not fail to recognize these as the expressive symptoms of the drunkard's disease; and inquiring no farther, you might have imagined that opium and ammoniacal stimuli were all that this case would require. But in the investigation of a case you must carry your inquiries beyond those limits which the eye merely reaches, the senses of hearing and of touch must lend their aid, and by them you must satisfy yourselves that no complication exists,—no lurking mischief in deep-seated organs, working insidiously to a fatal termination, while you have been busy only with those symptoms that lay on the surface, and are most easily and obviously detected. Here the respiratory movements of the chest were so much accelerated, that even without the stethoscope, pulmonary disease was to be suspected.

On carefully examining the chest, first by percussion, the left side was duller than the right, and this dullness was specially in the mammary and lateral region; on the application of the stethoscope over this spot, bronchial respiration, with a dry crepitating murmur was heard. The presence of bronchophony, and the total absence of any vesicular murmur, convinced me that the central portion and probably the root of the left lung was the seat of the inflammation. Superiorly and in the axilla of the left side, mucous rhonchi of small bubbles veiled all other respiratory sounds. The resonance of the right side was not much affected, but the mucous gurgling was heard in every region. This

physical state of the chest indicated the presence of broncho-pneumonia,—pneumonia situated probably at the root of the left lung, and an inflamed state of bronchial mucous membrane throughout its entire extent, the intensity being greatest in the smaller tubes; and I formed this opinion upon the absence of any moist gurgling of large bubbles being heard in the regions where the presence of secretion in the larger tubes is most readily detected—namely, posteriorly between the scapula, and anteriorly in the mammary region. The character of the sputa corroborated to a certain extent this diagnosis. They were copious and much was of a pure white frothy aspect, without much tenacity; but a portion was also frothy and somewhat rust-coloured, and this portion was more tenacious than the rest. Now, the pathological conditions on which these phenomena depend are these:—In that portion of the lung in which pneumonia exists, a retardation or stagnation of the blood in the arterial capillaries first takes place; what is the primary cause of the retardation or arrest to the even current of the capillary circulation, is not known or even likely to be. All that we can attain to is a knowledge of the conditions of any given phenomenon; the causes of those conditions are among the impenetrable mysteries of nature, and it is a useless expenditure of time speculating or guessing at it. All that we can state with certainty, is, that the earliest change observed in the capillary vessels of a part, the seat of inflammation, is a stagnation of the blood-corpuscles; their onward progress is arrested, and as an effect of, or concomitant with, this diminished motion, is the escape of a fibrinous spontaneously-coagulable fluid, which, filling the surrounding interstices of the tissue, consolidates the part, and gives firmness and solidity to what was previously elastic and extensible. In the pulmonary parenchyma this exudation not only fills the bronchial intercellular passages but the air-cells also. This accounts for the denseness and increased specific gravity of a portion of inflamed lung; and it also explains the presence of the dry crepitating sound on applying the ear to the region immediately over the seat of inflammation. This fibrinous exudation, the special product of capillary hyperæmia, contains dissolved in it much of the hæmaphrein of the blood; its presence gives to the inflamed part the dark brown-red colour always observed in the first stage of pneumonia. A portion of this colouring matter, (hæmaphrein,) dissolving and mixing with the bronchial mucus, tinges that fluid, a brown, red, or rust colour, and when expectorated presents us with the sputa so familiar in this disease. This constitutes the first stage of pneumonia. The next stage is the softening or breaking down into a purulent degeneration of this fibrinous deposit. In the case before us this condition had scarcely commenced, as the granular state and colour of the part testified, for as softening proceeds, all the hæmaphrein of the blood is absorbed out, and a grey cheesy mass is left. When examined under the microscope only a few corpuscles that developed nuclei by the action of acetic acid were observed, the mass being composed of granular exudation corpuscles.

Now, it was not pneumonia only that the stethoscope detected; the other physical signs implied that the bronchial mucous membrane, especially in the smaller tubes, was in a state of inflammatory congestion. The *post-mortem* examination confirmed this, and exhibited to you morbid complications not unusual in the pneumonia of drunkards. The immediate cause of death in these cases is asphyxia, or death beginning at the lungs. The accumulated secretion of mucus in the air-tubes as effectually obstructs the passage of oxygen to the blood as any other asphyxiating agent; and when such conditions exist, coma usually for some hours precedes death. Venous blood continues to circulate till the patient dies literally "*poisoned by his own blood.*"

Many circumstances in this case co-operated to bring on the fatal issue. In the first place, the inordinate use of intoxicating drinks at all times tends to the creation of an inflammatory diathesis. Again, the season of the year, and the particular condition of the atmosphere—frosty, foggy, with a very elevated barometer, most unquestionably predispose the lungs to become the seat of inflammatory action. You daily witness, among both out and in-patients, how the season of the year predisposes to pulmonary disorders. Many circumstances co-operating, a series of morbid effects followed, from which it was not reasonable to expect that the patient could survive. I have already had occasion oftentimes to remark to you, that the co-existence of acute inflammatory action, with a general adynamic condition of the system at large, is by no means rare. It is this antagonistic state of things that for the most part renders inflammatory diseases fatal; for if the powers of the system be sufficient to permit those remedies to operate, which only can arrest the progress of inflammatory action, all may do well; but if the powers of life be low, these measures often add to the prostration, while they fail to check the advance of inflammation. Nevertheless, in such cases they must not be neglected; and indeed it is not unfrequently necessary to cup, blister, and give mercurials with one hand, while the system is supported by ammoniacal stimuli with the other.

The treatment of these cases requires great discretion. To bleed from the arm is not advisable; rapid subsidence of the vital powers often follows abstraction of blood from a vein. The most effectual and least hazardous mode of relief is by cupping between the shoulders, or over the affected side. Blistering is also desirable. Calomel, antimony, and opium, should be cautiously administered, and ammoniacal salines given at intervals the moment the pulse, by its softness, indicates their necessity.

I wish to direct your attention to the case which died in the same ward on the same day,—one also of pulmonary disease; but an emphysematous state of the lungs, with chronic bronchitis, were the characters determined by the stethoscope, and at our next lecture I wish to point out to you some of the chief distinguishing features of that case.