Clinical Lectures

DELIVERED AT

CHARING CROSS HOSPITAL.

BY

HYDE SALTER, M.D., F.R.S.,

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS; ASSISTANT
PHYSICIAN TO THE HOSPITAL.

LECTURE I.—REGURGITANT AORTIC DISEASE.

CASE. *The Three Varieties of Orthopnoea. Explanation of the Peculiarities of Heart-Dyspnoea. The Pulmonary Congestion of Lung and Heart-Disease contrasted. Cardiac Cough. Aortic Regurgitant Pulse: its Mechanism. Circumstances conspiring to Heart-Displacement. Cardiac Sputum. Diagnostic Value of Age. Treatment.*

GENTLEMEN,—The case to which I wish to-day to call your attention is that of a man lying (or rather sitting, for he cannot lie) in bed No. 9 of Bow Ward. The case is one of regurgitant aortic disease, and its history is as follows.

Robert Carey, aged 40, a widower, by occupation a bill-poster, and has been engaged in that occupation seven years. He has been what he calls a temperate man, taking, on the average, "two pints of porter and a drop of gin" a day, and rarely exceeding that quantity.

Up to three years ago he was a healthy, strong man; at that time he had rheumatic fever, which affected his head, back, and every joint in his body, and for which he was under treatment seven weeks in the Kingsland Road Infirmary. When he came out, he was quite well in every respect except his heart, which had suffered greatly during the rheumatism, and which was still the seat of pain when he left the infirmary. He suffered a good deal too from palpitation, which to a certain extent embarrassed his respiration and incapacitated him from violent exertion. These symptoms gradually diminished, and almost vanished; but he has never been quite the man he was before. From his occupation he was very much exposed to wet and cold, and frequently caught colds. These, with occasional palpitations of his heart, obliged him two or three times during the past year to lie up for a day or two; otherwise, he went on well till about six months ago, when he took a violent cold, which affected his chest and breathing a good deal, but for which he did not give up work. But ever since this his breathing has been worse than it ever was before, and he has suffered more from palpitation; but he slept, and ate, and drank, as well as usual. He continued his pill-posting up to three weeks ago, when it was that his present condition came on him. It developed itself gradually, and consisted in an increase of his former symptoms. He coughed more; his breath was shorter; he had more palpitation; was unable to lie down at night; the feet took to swelling; the face also; and he experienced great incapacity of exertion; it took him three-quarters of an hour to walk from Temple Bar to Hungerford Market. He was then not utterly unable to lie down at all, but slept about an hour and a half, and then was suddenly awoken with a sense of impending suffocation and choking. In this state he came to me this day fortnight.

Such is a brief history of his case. His condition since his admission is such as you have watched from day to day, and such as I shall make it the purpose of my lecture to-day to unfold and explain to you.

The first thing that would strike you on looking at this man would be his dyspnoea. There are great differences in dyspnoeas, according to the different causes that produce them, and which suggest many interesting physiological questions, into which, however, I must not enter. Now this patient's dyspnoea is eminently *cardiac*. The peculiar characteristics of cardiac dyspnoea are these:—It is rather a breathlessness than a difficulty of breathing; it has a panting, gasping character; the sensation complained of is that of oppression, not tightness, and has a strangling, choking throat-feeling about it. The breathing is always greatly accelerated; many dyspnoeas are not—laryngeal dyspnoea, for instance, which is often slower than natural; but cardiac dyspnoea always is. This man is breathing forty-six respirations in a minute.

Another peculiarity of heart-dyspnoea, well illustrated in this case, is its great intolerance of movement, or any exertion whatever. Very often, in heart-dyspnoea, exertion is the only circumstance under which the dyspnoea is felt. As long as the patient is at rest there may not be the slightest sense of difficulty of breathing, and the respiration may not be distinguishable from that of a person in ordinary health; but the moment he exerts himself, especially if ascending a hill or going up stairs, his breath is gone. So it is with our patient. Although his breath is always short, even when perfectly at rest, it becomes frightfully difficult if he attempts to move. He declares that merely turning his head brings on the dyspnoea; speaking is almost impossible; he cannot rise from his bed, or move two steps, without being completely *hors de combat*.

Another peculiarity of the dyspnoea of heart-disease is its intolerance of the recumbent posture; and this peculiarity has given its name to one of its most characteristic forms—*orthopnoea*. The word really signifies merely "upright breathing"—compulsion to assume the erect position in order to breathe; but clinically it stands for a good deal more—for a recognised and well marked group of respiratory phenomena. Our patient is an example of it in its severest form; he has not lain down to sleep for ten days and nights; and if he momentarily dozes in even a semi-reclining posture, he is instantly awoken by impending suffocation.

There are three forms, or rather degrees, of orthopnoea: the first, in which the patient may suffer little or no dyspnoea when awake, even when exerting himself moderately, or ordinarily when sleeping in the recumbent posture. He lies down and goes to sleep as if in health; but occasionally, in the middle of the night, he is suddenly awoken with a strangling, gasping breathlessness; starts up, flings the clothes from him, and struggles for his breath as if he were dying. Very commonly the patient jumps from his bed in the effort to get more air, and sits gasping on the side of it, or leaning on a chair. It is often a quarter or half a minute before he can "turn his breath"; and, when he does so, his respiration is hurried and panting. As the circulation through the lungs becomes freer this gradually subsides, and the breathing becomes as tranquil as ever. This may happen at long intervals, or frequently; very often it occurs regularly every night, and sometimes many times in a night. The fear which patients exhibit, after such a paroxysm, of going to sleep again, lest another should

come upon them, shows how terrible is the sensation that accompanies the struggle.

Another form is that in which the patient can breathe well in any position as long as he is awake, and can sleep perfectly well sitting up, but cannot sleep lying down for a moment, the attempt to do so being immediately attended with the characteristic paroxysm. In these cases, the patient is obliged to sleep in the erect posture, and in that way gets pretty good nights.

The third and most intense form is that in which sleep is altogether impossible in any position, even sitting up: the moment the patient dozes, on comes the struggle; he is wide awake in an instant; sleep is hopelessly banished from him. If you stand by and watch a person in this state, which is a most painful thing, you see his drowsiness gradually overpowering him; his eyes close; his breathing seems momentarily more tranquil, and you would fancy he was just falling off into a refreshing slumber; but, a moment after, he starts up with a look of horror, and his quiet breathing is instantly exchanged for the orthopnoæal struggle.

This last is the form of orthopnoæa from which our patient suffers; he has not had ten minutes sleep, night or day, for nearly a fortnight; he is dying with sleepiness, but dares not yield to it; and, if it overcomes him, he is unconscious but a few seconds before he is broad awake in a death-struggle for breath. Such cases show with how little sleep a person can go on living. I think it is impossible, in the whole range of the incidents of disease, to imagine any suffering that can compare with this: the thirst of Tantalus could not be worse. It is not wonderful that the sufferers long for the sleep of death.

And this leads me to speak of what is the absolute nature of heart-dyspnœa.

The essential condition of tranquil respiration is a free renovation of the two fluids, air and blood, in the lungs: the essential condition of dyspnœa is an impeded renovation of one or both of these fluids. In lung-dyspnœa, the fluid whose renovation is impeded is the air—as, for example, in bronchitis, asthma, emphysema; in heart-dyspnœa, it is the blood. The immediate cause, I believe, of the sensation of want of breath, in any form of dyspnœa whatever, is congestion of the capillaries of the lungs, dependent generally on the presence of venous blood in those vessels. How this is produced by shutting off the air we understand perfectly well: the oxygenating agent is not supplied; the appropriate changes do not take place in the blood; and the capillaries, exercising upon it some obstructive force the exact nature of which we do not know, refuse to give it free transit, and it accumulates in them and in the pulmonary veins behind them, constituting the congestion of asphyxia. In the lung-congestion from disease of the left side of the heart the sequence of events is quite different. The point of obstruction here is not in the capillaries of the lungs, but at the heart; so that the whole of the pulmonary circulation, veins as well as arteries, is affected by it. The capillaries become loaded, not from the blood being in a state unfit to pass from them, and from an opposing action to its free transit exercised by themselves, but from finding in front of them loaded pulmonary veins. Moreover, in cardiac congestion of the lungs, until ulterior changes take

place, the access of air to the pulmonary capillaries is free, and the blood in them therefore arterialised. Thus, in the congestion of asphyxia, the capillaries are loaded with venous blood; in that of heart-disease, with arterial. But we know that impediment in the capillary circulation of an organ as inevitably involves suspension of its function, as suspension of its function involves impediment in its capillary circulation. We know, for instance, how mechanical congestion of the kidney diminishes the quantity of the urine. And, although the blood in the congested pulmonary capillaries in heart-disease may be arterial, yet being itself unable to pass on, from the obstruction in front, it prevents the venous blood behind it reaching the seat of arterialisation, and so suspends the function of the lungs, and loads the branches of the pulmonary artery with venous blood, as much as in true asphyxia. And it is not long before the congested capillaries relieve themselves by serous transudation, giving rise to vesicular and interlobular œdema, and other ulterior changes, so that respiration becomes directly impeded. Nevertheless, there is at first that difference between cardiac and asphyxial lung-congestion which I have indicated; and the points of contrast may be tabularly represented thus:—

<i>Pulmonary Congestion of Asphyxia.</i>	<i>Pulmonary Congestion of Heart-Disease.</i>
Suspended function producing capillary stasis.	Capillary stasis suspending function.
Point of obstruction the pulmonary capillaries.	Point of obstruction the heart.
Pulmonary veins emptier than usual.	Pulmonary veins loaded.
Blood in pulmonary capillaries venous.	Blood in pulmonary capillaries arterial.

Since, then, in the first stages of cardiac dyspnœa, the blood in the capillaries of the lungs is arterial, the presence of venous blood in them cannot be necessary to the sensation of want of breath: impeded transit of blood through the lungs, and a loaded state of the pulmonary circulation, would appear to be all that is necessary.

The extreme dyspnœa from which our patient suffers suggests the question as to what form of cardiac disease the most embarrasses the breathing; for we often see patients dying of heart-disease with much less dyspnœa than we see in this poor man at the present time. This question I am not prepared to answer. I should have imagined that mitral regurgitation, constituting an obstruction that the most directly tells back upon the pulmonary circulation, would have the greatest tendency to engender dyspnœa, as it certainly has to produce pulmonary hæmorrhage; yet here we have what we may call acute orthopnoæa, easily induced by a valvular derangement from which the lungs are protected by the intervening healthy mitral valve. I profess myself, then, unable to answer this question; but it is one well worth thinking of, and well worth making the subject of observation.

I have detained you thus long in analysing one of this patient's symptoms—the dyspnœa—because it is, as I hope I have shown you, so full of instructiveness, physiological as well as pathological; but there is another symptom that would strike you as immediately as the dyspnœa—the *cough*. This is the true cardiac cough—a single hacking cough, not in par-

oxysms, but iterated, keeping on ; never urgent, yet not passing half a minute without occurring ; feeble, with a certain throat-hoarseness about it, having rather a choking character, and with no expectoration as its result. When I say *no* expectoration, I should say there *was* no expectoration ; but a little is now appearing.

[*To be continued.*]

Clinical Lecture

ON THE

TREATMENT OF RHEUMATIC FEVER,

AND OF THE USE OF ALCOHOL IN SERIOUS CASES OF THIS DISEASE.

DELIVERED AT KING'S COLLEGE HOSPITAL,
WINTER SESSION, 1861-62.

BY

LIONEL S. BEALE, M.B., F.R.S.,

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS ; PHYSICIAN TO
KING'S COLLEGE HOSPITAL ; PROFESSOR OF PHYSIOLOGY AND
OF GENERAL AND MORBID ANATOMY IN THE COLLEGE.

Complicated Cases of Rheumatic Fever. Two Classes of Cases.

CASE II. *A boy aged 16. Joint affection. Pericarditis with effusion. Slight pneumonia. Pulse 132 ; respiration 72. TREATMENT as in Case I, but less opium was given, and only four ounces of brandy in twenty-four hours. Convalescence a fortnight after the disease was at its worst.*

General remarks on the treatment pursued. On the propriety of giving stimulants in cases of acute rheumatism.

[*Concluded from page 83.*]

LET me now ask your attention while I give a very brief outline of another case of the same malady, in which the same plan of treatment was carried out ; but, as the patient was much stronger, it was only necessary to give small quantities of brandy.

CASE II. The patient is a boy of the name of Alfred Davies, in No. VII Ward (vol. iv, p. 36). He is 15 years of age ; and, as far as the morbid processes are concerned, he was in much the same state as the case I have already adverted to. There was joint-affection, with loud to-and-fro rubbing sound over the heart, followed by effusion in the pericardium. There was some extent of pulmonary affection.

Two days after admission, the pulse was 132, and the respiration 72. The same plan of treatment was pursued as in the case of the girl ; but it was not necessary to give so much opium ; and as the powers of the patient were good, as his voice was strong, as he could move about in bed, and sit up, and as his pulse, though quick, was strong, no alcohol whatever was given for the first nine days ; but then his strength began to fail ; the pulse was still as high as 128, and much weaker ; so that I thought it desirable to give him small quantities of stimulant.

From the 2nd (the day he was admitted) to the 9th November, the pulse had varied slightly in frequency from day to day ; but the lowest was 104, and the highest 132.

On Nov. 10, the pulse was weak, 128 ; and he was ordered to take four ounces of brandy in the twenty-four hours.

On Nov. 11, it was 124 ; on the 13th, 104 ; and by the 19th, it had fallen to 96.

On Nov. 21st, a further diminution in frequency had occurred, and the brandy was reduced. A manifest and rapid improvement in the condition of the patient accompanied the diminution in frequency of the pulse.

A gradual diminution in the frequency of the pulse, and an increase in its power, were observed for the four days succeeding that on which he commenced taking four ounces of brandy.

By Nov. 26, he was able to resume ordinary diet, and was convalescent. The rubbing sound and the pericardiac dulness gradually diminished, and a systolic mitral bruit became audible. His strength gradually improved, and he left the hospital on December 14, just one month from the time when the disease was at the worst.

The essential nature of the morbid changes in the two cases I have brought under your notice in this lecture was the same. In each there was pericarditis, followed by regurgitant mitral bruit. The general plan of treatment was the same ; but, as the exhaustion was far greater in the first case than in the second, it was necessary to give a much larger quantity of alcohol, and keep it up for a longer time. In the latter case, a marked improvement was observable in the patient's state very soon after he was put upon four ounces of brandy in the twenty-four hours ; for from this time the pulse steadily diminished in frequency (from 128 to 96) and increased in power.

General Remarks on the Treatment Pursued. I hope I have succeeded in my effort to explain clearly the principles which have guided me in the treatment of these cases. I did not attempt to treat the disease ; but endeavoured to ascertain precisely in what way a fatal result was likely to occur ; and did all I could to avert it or postpone it. I have not cured the malady ; but I think we have been the means of keeping the first patient alive while the disease passed off. From the observations of others, and from having watched the course of many cases myself, I knew that the absorption of fluid and lymph in the pleura or pericardium could not take place very rapidly, and I knew that if the effusion increased beyond a certain point death must result. No treatment could possibly produce any sudden alteration in the morbid conditions which tended to kill the patient.

If we had concentrated our attention solely on the special disease ; if we had considered only what should be done for the pneumonia, for the pericarditis, for the rheumatic state, the patient might have died from prostration. She might have succumbed to the depressing effects resulting from the morbid condition. True, if we could remove the cause of the prostration, it would be better than relieving the last by artificial stimulus ; but we cannot remove the cause. We know that the disease must disappear slowly ; we know that when the case is fatal we lose the patient more commonly from exhaustion than from disease. Now, in alcohol we have a remedy which, of all others, acts rapidly on the flagging powers of a patient worn-out by disease. It does not increase inflammation. It cannot increase the tendency to formation of lymph. By restoring the powers, it undoubtedly tends to diminish the tendency to effusion. Alcohol is not an *antidote*. It does not destroy a poison in the system ; neither does it combine with one and render it inert.

I have given you the history of but two cases,