brain"; any motion or coughing jarred and hurt his head, or rather its contents. His head, however, was not tender to tapping. He had no rheumatism; no syphilis; no paralysis. His intellect was clear; but he had sensations of things crawling about the lower part of his forehead. The age of this patient would make one suspect calcification of the cerebral arteries; and this might occasion the symptoms complained of. However this may be, there can be little question that, owing to some defect in nutrition, the cerebral nerve-tissue became intolerant of slight commotions, which ordinarily it could have endured very well.

But, to return to our patient: I do not think the peculiar dysæsthesia of which he complained—the jarring sensation—was of any special significance. No doubt, some minute modification of molecular arrangement corresponds to each of the manifold dysæsthesiæ which such sufferers experience; but these are far beyond our ken, and it is not of any vital importance to the practitioner to be acquainted with them. The subjective phenomena (the same is true of the objective) are no sure guide to the *quality* of the disorder; and this is what especially concerns us. You will never treat derangements of the nervous system successfully, if you suppose that all phenomena having the same outward appearance are really alike. Cases of delirium, of spasm, of neuralgia, differ very greatly, though, as far as phenomena go, they are identical. In the case of H. G., I was led into error at first by the history and by the evident signs of prostration of nerve-power. These led me to give tonics and stimulants, which availed nothing for the relief of his head. When, however, I administered an unexciting calmative, aided by a counterirritant (for such I consider the sternutatory), improvement ensued rapidly. You may perhaps think that such a remedy ought, on rational grounds, to have been employed at once; and that stimulants were inappropriate to the symptom *pain*. A little experience, however, will show you that, in a multitude of instances in which debility constitutes a prominent feature, stimulants are most efficacious sedatives. Two or three glasses of port wine will remove, for the time, neuralgia in some cases. All neuralgic pain, however, is not to be thus dealt with; and you may need, to allay it, remedies which are very much the reverse of stimulants, such as hydrocyanic acid, bromide of potassium, aconite, and moist warmth. When I find such means as these successful, I think the condition is thereby at once differentiated from that in which our ordinary tonics avail; just as much as two different saline solutions are distinguished by chemical tests.

As I have above said, I cannot tell you in what the essential difference consists; but I find it of value to keep in my own mind the two conditions separate, and to denote them by some such terms as neuralgia and hyperæsthesia. It may aid us in forming more definite conceptions of these two states, if we call to mind their analogues among motor disorders, such as choreal paralysis and jactitation, which, though extremely unlike, undoubtedly originate in the same morbid process, and are, so to speak, truly homogeneous. It is a fact, that all causes which impair strength are very prone to generate hyperexcitability, which may show itself in the sensory, or motor, or intellectual departments.

Lastly, as to the sternutatory. This must be regarded as a form of counterirritation; having this advantage, however, that it is capable of repeated application within a short time; and that it provokes repeated diversions of blood, with mucous exudation. The formula which I employed is that recommended by Dr. Laycock; and you see that it was very positively more efficacious than common snuff. Its good effect in our patient depended, I think, on its producing reflex stimulation of the vaso-motor nerves of the cerebral arteries, and lessening the blood-flow, which was probably excessive for the *weakened* tissue. I emphasise the word *weakened*; for I hold it well ascertained, that an organ whose vitality is vigorous can bear and employ beneficially a much larger supply of blood than one which is in an opposite state. The nerves of the Schneiderian membrane are perhaps better fitted than any to exert this constricting influence. At the same time that the sensory nerves of this membrane are excited, its vaso-motor are, in all probability, relaxed; so that the vessels become suddenly congested, and modified blood-fluid and corpuscles escape. This seems to be the general rule in all localities, that tissue-excitement determines vasal relaxation. Fatal cases of tetanus and chorea afford marked examples; the vessels of the cord or other nervous centres being engorged, and rupture and extravasation having ensued in some places. To an hyperexcitable organ, an undue amount of blood-flow is certainly injurious; so that it is good practice to try to calm tissue-excitement while we constrict vessels.

I do not know that I can altogether explain the mode of action of counterirritants; but their utility, in spite of recent scepticism, I do not doubt; and I strongly advise you not to doubt.

To conclude, let me counsel you to familiarise your minds with such common things as the management of headache in its various forms. Many of you may never perform lithotomy, nor perhaps amputate; yet

students crowd to gaze at the great operations, of which they can see next to nothing, and which they are little likely ever to perform; while they can hardly be brought to give a thought to the treatment of a malady which they may have to deal with any day of their lives.

CASES AND COMMENTARIES.

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

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No. I.—*Case of Double Aortic Disease.—History of Case and peculiarities of Physical Signs.—Diastolic Impulse.—Relations of Diastolic Murmurs to Diastole.*

HENRY BENNETT, aged 38, a tall, spare, sallow man, with thin, lank, black hair, by occupation an engine-fitter, temperate, married, with four children, all healthy, was admitted into Charing Cross Hospital on December 28th, 1869. He has for years suffered from an undue beating of the heart, but not enough to interfere with his work; a twelve-month ago this palpitation became more violent; at that time, he had an attack of bronchitis, from the heats and chills to which he was exposed in his occupation, and it was on recovering from the bronchitis that he noticed that the palpitation had increased. Eight weeks ago he experienced a further and sudden increase of it, without any bronchitis, or any assignable cause; and now to the palpitation was added dyspnoea—he was unable to walk quickly, or work at his employment, and on lying down at night his breath was very short, and his heart seemed beating in his throat, so that he was obliged to jump up in bed again; from that time, too, he has, almost every night, suffered from orthopnoea, being aroused from his sleep by a sense of suffocation, starting up and labouring for his breath with the utmost violence; especially does this occur if he lies on his left side. He has, too, what he never had before, a sense of uneasiness in the epigastrium accompanying these attacks of palpitation and breathlessness. He has never had rheumatic fever, or rheumatism in any form.

Physical Examination.—On stripping the man, two things are at once seen; first, a strong pulsation of all the arteries, at the root of the neck, arms, etc.; and, secondly, an undue visibleness of the heart's movements over the whole of the front of the chest, especially in the intercostal spaces of the left side, and the scrobiculus. The apex is seen strongly beating between the sixth and seventh rib, at a distance of four inches in a vertical line dropped from the left nipple, and seven inches and a half from the base of the heart. A fluctuous brush of wave-like movement, passing from the region of the apex to the left at each pulsation, is very striking and conspicuous in the fifth and sixth intercostal spaces. On listening at the base, a double murmur is heard, the two running into one another—the first soft and faint, the second loud, rough, and terminating abruptly. On checking these with the pulse, the first is found to be systolic, the second diastolic. There is, therefore, double aortic mischief, constrictive and regurgitant. It would be difficult, perhaps, to say at which margin of the sternum the basic murmur is the loudest; I think the left; but it is decidedly of deeper and softer tone at the right, flatter and harsher at the left, and more conspicuously abrupt in its termination. At the apex, a loud single murmur is heard, accompanying the stroke of a very strong impulse, so strong as to raise the stethoscope with great force against the ear. This impulse has all the characters of a heaving thumping systole, but on checking it by the pulse, I found, to my surprise, that it was diastolic, while at the systole there was no perceptible impulse whatever. About the diastolic period of this violent impulse there could be no doubt. I checked it in every way. On applying the fingers of one hand to the apex, and of the other to the wrist, the alternation of the heart's stroke and that of the pulse was very clear and striking; and on applying the stethoscope to the apex, and allowing its free end to move freely as a sphygmoscope, and comparing its movements with those of the visible pulsations of the radial artery, their alternation was equally conspicuous. I do not know that I have ever felt an impulse of greater strength, more thumping character, and wider distribution; and yet with this impulse the systole has nothing to do. At the period of the systole there is no trace of impulse, and hardly a trace of sound; what little sound is perceptible appears to me to be rather of the nature of a very soft and almost inaudible systolic murmur than having any of the character of a natural first sound. It is, however, so faint and so masked by the loud diastolic murmur, that it is difficult to say anything as to its character. Nothing could be more misleading as to the heart's rhythm than such a diastolic impulse as this; not only its extraordinary strength, but its *singleness*, gave an impression of its systolic character that was almost irresistible;

and no one watching it, or feeling it, or listening to it, unchecked by the pulse, could resist the conviction that it *was* systolic, and that the murmur that accompanied it was a systolic murmur. No case could better illustrate the importance of checking the cardiac movements by the arterial movements. The arteries of the body seem everywhere alive. Pulse 96; respiration 20; pulse-respiration ratio, therefore, $\frac{5}{2} = 2\frac{1}{2}$, when sitting and tranquil.

The patient became progressively and continuously worse from the day of his admission. He suffered greatly from a pain through his chest, as if it were in a vice; this was a source of great torture to him. For ten days before his death he suffered from constant orthopnoea, and for several nights had no sleep whatever. An eighth of a grain of morphia injected hypodermically gave relief to the anginal agony, but gave him no sleep. The heart's action was very violent, from 120 to 125 in a minute. During the last two days of his life he was troubled with constant vomiting, and took no food. At 8 o'clock on the morning of January 17th, having been asleep about ten minutes, he suddenly woke up, gave a shriek, threw up his hands, fell back, and died almost instantly in the arms of those who were supporting him.

On examination after death, the heart was found to weigh twenty-four ounces; the aortic valves were a mass of atheroma, thickened, stiffened, and puckered; the valves of the pulmonary artery were healthy; the mitral valve was thick and leathery, but not contracted, and was evidently adequate to the closure of the orifice.

There are five points to be noticed in this case.—1. The greater loudness of the diastolic than the systolic element of the double aortic murmur. Many writers insist on the greater loudness and strength of systolic murmurs than diastolic, and maintain that certain characters attach to systolic murmurs exclusively, because they *are* systolic, and because other murmurs are not developed by a force sufficient to elicit them, such as thrill and musical tone. It is impossible, say they, that a mere passive relaxation can generate a blood stream of the same force and rapidly as an active contraction, and therefore we must not look to any but systolic murmurs for the highest expressions of endocardial blood-friction. No mistake can be greater than this; it is founded on an imperfect comprehension of the physics of the case, and is contrary to clinical experience. One of the most musical murmurs I ever heard, of the clearest and highest pitch, was a diastolic aortic murmur which I heard last week; and I have recently pointed out that presystolic murmur is peculiarly characterised by thrill. This is not the first, nor the second, nor, I think I might say, the tenth case, in which I have found the diastolic element of a double aortic murmur predominant. Even lately we have had several cases in this Hospital in which it was so; in the cases of Samuel Easton and George Castle, to such a degree that the loud diastolic murmur almost masked the systolic, so that the latter was at first overlooked; it was so, also, in the cases of Michael Barret and William Brown. I am inclined to think that it is the *rule* for the second half of a double aortic murmur to be strongest of the two, and that it is greatly the exception to find the reverse. If so, what is the explanation of it? I think it is this. The force in each case is equal—on the principle that action and reaction are equal to each other. The force exerted by the ventricle is spent in imparting movement to the blood in the proximal portion of the aorta, in resisting its tendency to backward flux, and in further distending the arterial system—in other words, in overcoming, directly or indirectly, the elastic contractility of the arterial wall. This force, thus dealt out, is stored up, so to speak, in the arterial wall, ready to be dealt back again (and, in fact, *is* immediately dealt back again) in impelling the blood onwards if the aortic valves are sound, and in driving the blood back again into the ventricle if they are incompetent. The force, therefore, with which the blood is driven back into the ventricle in aortic regurgitation is simply the very same force with which it has just been driven forward. So far, then, *ceteris paribus*, the sound ought to be the same—the regurgitant murmur just as loud as the constrictive. But why should the regurgitant be louder? The reason, I believe, is that the forward stream is an opposed stream, and requires the sustained action of the ventricle to carry it onwards; it is, as it were, laboriously pushed forward against the blood in front of it, which it has to propel a stage further along the aorta; while the backward stream in regurgitation is an unopposed stream—there is no tense blood in the dilating ventricle to delay or impede its back-rush. The consequence is that the stream that generates a systolic is less rapid than that which generates a diastolic murmur, and therefore produces less friction and develops a feebler sound. The explanation is exactly the same as that of the greater loudness of the expiratory than the inspiratory sound in bronchial breathing; inspiration has the elastic contractility of the lung to overcome—whereas expiration is an unopposed act; expiration, therefore, takes half the time of inspiration, and its air-stream is twice as rapid, and therefore twice as sound-generating.

2. In one sense the regurgitant murmur in this case might be called pre-diastolic—that is, it preceded the loud and strong second sound which suddenly terminated it, and up to which it ran. It is evident that an aortic regurgitant murmur cannot *really* be pre-diastolic, as it cannot commence till the ventricle begins to dilate, that is, ceases to contract, and its commencement must mark, and exactly coincide with, the commencement of the ventricular diastole. But in relation to the diastolic sound, it *may* be pre-diastolic, for the regurgitation may take place before the injured valves fall together (as far as they *can* do so), and, indeed, may delay their falling together, and thus delay the production of the second sound which suddenly terminates the murmur. That the injured valves did partially close in this case, and thus put a stop to the principal regurgitation, was shown by the strong and tolerably sharp second sound with which the murmur terminated. It is clear, then, that the interval between the first and second sound may, at the base, be occupied by two distinct murmurs—one, the ordinary aortic systolic murmur (A), running *from* the first sound into this in-

Diagram representing the relations to the first and second sounds, of (A) a systolic murmur running *from* the first sound, and (B) a diastolic murmur running *up* to the second sound.

terval; the other, the murmur which we find in this case (B), commencing in the interval and running *up* to the second sound. It is quite clear, since the murmur cannot take place till the ventricle relaxes, and since the sound marking the closure of the valves follows it, that the closure of the valves cannot coincide, in this case, with the commencement of the diastole, as it is generally supposed to do. It is very possible, as I have suggested, that the diseased condition of the valves and orifice may retard the falling in of the valves, and thus that we have in such a case a postponed second sound. If so, the murmur is really the true diastolic event, and the second sound in such a case is *post*-diastolic. The thickened and stiffened condition of the aortic valves in this case is just such as one can imagine impeding and delaying the falling together of their segments—in fact, such as must necessarily have been incompatible with that quick movement and instantaneous apposition which the valves effect in their natural thin and flexible condition. There is yet a second corollary that may be deduced from this case—that an aortic regurgitant murmur may have two relations to the second sound—may precede it or succeed it; may run up to it, as here, or run down from it, as is ordinarily the case. In the former case, that of this man, the regurgitation takes place before the valves can fall together, and is sufficiently arrested by their closure to stop the murmur; in the second, the ordinary case, the valves fall together, as far as they can, the moment the ventricle relaxes, and then, their closure being imperfect, the arterial tension continues to squirt the blood back through the chink, till, the ventricle being filled from behind, a balance of pressure is arrived at.

3. This case illustrates the point, that the rule about right and left margin of the sternum, in diagnosing aortic and pulmonary-arterial disease, does not apply to regurgitant murmurs, but only to direct ones, as the murmur was perhaps slightly louder on the left (the pulmonary) side of the sternum, whereas the regurgitation was aortic.

4. The conspicuousness of the murmur at the apex, though in its genesis a basic murmur, illustrates the influence of convection on the localisation of cardiac murmurs.

5. This diastolic impulse, without a trace of systolic—what is it? What is its nature? What produces it? Has it anything to do with regurgitation—with the sudden filling of the ventricle with a “flop” of blood from the aorta, or with the concussion of the two streams meeting one another in the ventricle, one rushing back from the aorta, the other forwards from the auricle?