ON THE OCCURRENCE OF A DIASTOLIC MURMUR OF AORTIC ORIGIN APART FROM AORTIC INCOMPETENCY OR ANEURYSM.

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In discussing the diagnosis of cardiac disease, I have been in the habit of insisting very strongly on the gravity and importance of a diastolic murmur, contrasting it with the comparative uncertainty of systolic murmurs, which are not unfrequently of but trivial import. The diastolic murmur seemed, however, to be always due to important organic disease, and to be diagnostic of aortic valvular incompetence, except in those cases where it was due to aneurysm, or dependent on pericardial friction. A theoretical provision had also to be made in favour of incompetency of the valves of the pulmonary artery, and perhaps of some other pathological rarities. With these exceptions, a diastolic murmur (as I believed) invariably indicated regurgitation through the aortic orifice into the left ventricle.¹

Some years ago, in conversation with Dr. Gairdner at a clinical examination, I gathered that my way of putting the case was somewhat stronger than the actual facts warranted; but, although I expressed myself more guardedly after this, nothing had occurred under my own observation, till last winter, to impress me with the danger of relying on this sign. Curiously enough (although such chances are common in medicine), I came across a second case within a few months; and, warned by the recent experience, I was not so far misled the second time.

The first case was one of extreme dyspnoea, with symptoms of the angina type, and very marked evidence of enlargement of both sides of the heart. There was likewise albuminuria, but scarcely any dropsy. The patient was a seaman, and was 47 years old. There was no history of acute rheumatism—only some vague pains for the last three years, which never laid him aside from work. There was no indication of aneurysm, nor did the history suggest such a thing. When, therefore, I found a very loud double murmur, audible all down the sternum, I concluded at once that we had to deal with the well known aortic regurgitant disease. The urine was albuminous; but this seemed very likely a secondary phenomenon, or at least a merely concurrent disease. The notes bore that there had been frequency of micturition, and that the quantity was supposed to be a little greater than usual. Unfortunately, the amount was not measured. The specific gravities varied from 1015 to 1020, and it was specially noted that there was no lumbar pain.

The dyspnoea and orthopnoea became more extreme; remedies had but little effect, and before death the excitement and delirium of the patient almost amounted at times to mania, such as I have seen several times in cases of the uræmic poisoning of Bright's disease.² The patient frequently took off his clothes almost completely, climbing up on the windows and mantlepiece, thinking, apparently, he was on his steamer; he was often completely beyond the control of the attendants. He died ten days after admission, the whole illness being stated by him as only of about three or four months' duration. The lungs seemed but little implicated on admission, a few dry rales being only noted. Occasionally, the rhythm of the respiration assumed the Cheyne-Stokes character.

Unfortunately the pulse-tracings of his case were lost, and no note of them was entered in the journal. After death, I could not rely on my memory in describing their character.

The post mortem examination showed that the diagnosis was essentially wrong. The heart was, indeed, notably enlarged (27½ oz.); the hypertrophy involving the left ventricle more than the right; but the primary mischief seemed to be in the kidneys, both of which were contracted and granular, with abnormal development of the connective tissue. In the pelvis of the left kidney, which was distended, a pretty large calculus was found, and the atrophy of this kidney was much more considerable than that of the other. The aortic valves were examined very carefully, and tested with water poured into the aorta; and the perfect apposition of the curtains, as seen from a somewhat enlarged aorta, without the slightest indication of any chance of regurgitation, was most striking. There was no indication of disease in the valvular structures. The aorta was dilated, and the

surface rough and atheromatous. The muscular tissue of the heart seemed normal to the naked eye, but, on microscopic examination by Dr. Steven, there seemed the earliest possible indication of fatty change in the central parts.

Here, then, we had cardiac murmurs, most strongly suggestive of aortic incompetency, without the least indication of such a defect at the post mortem examination. The murmurs were, very plain, and were used by Dr. Samson Gemmell and myself in our demonstrations of typical cases of cardiac disease. How far, then, are we entitled to trust to this sign?

Dr. Gee,³ in his admirable text-book, recognises the existence of a diastolic murmur apart from aortic incompetency as well established; but he says that such cases have always been associated with distinct disease in the aorta itself. The gravity, therefore, of a diastolic murmur still remains as an evidence of organic disease; but we must include along with aneurysm, amongst possible causes of the murmur, a dilated, or rigid, or atheromatous condition of the aorta.

Dr. Bellingham⁴ seems to have been the first to call attention to this murmur from disease of the aorta, apart from valvular lesions; and he explained it as due to the onward passage of the blood against the dilated, rigid, and diseased aorta during the systole of the heart, and to the regurgitation of the blood over the same surface, coming from the large branches into the rigid tube of the aorta, prior to the closure of the semilunar valves. Hence we may have, according to him, a systolic murmur, or a systolic and a diastolic murmur, in such cases, much in the same way as we may have single or double murmurs from aortic aneurysm, apart from any imperfection in the aortic valves themselves.⁵ This view of Dr. Bellingham's has not been overlooked, but does not seem to have gained general acceptance. Dr. Hayden, for example, says he has never met with a case in point.⁶

Dr. Law communicated to the Dublin Pathological Society⁷ a case of double murmur without aortic incompetency; and, in discussing the subject, he adheres to Dr. Bellingham's views. Dr. Cockle,⁸ likewise, gives an instance of double murmur in a case of diseased aorta, but with the valves apparently quite intact. At a meeting of the Society of Biology of Paris, a somewhat doubtful specimen was shown in illustration of this subject; but some of the speakers seem to have met with cases leading them to believe that double murmurs might depend on disease of the aortic wall without valvular lesions.⁹

Dr. Austin Flint¹⁰ has taught for many years that disease of the aorta may give rise to what he calls a "prediastolic" murmur, "caused by the retrograde movement of the column of blood, the latter caused by the recoil of the arterial coats directly the systolic ends, and which produces the closure of the aortic valve. If there be aortic insufficiency, this murmur is lost in the aortic regurgitant murmur. The name prediastolic denotes its occurrence just before the aortic sound. It is preceded by a systolic murmur, referable to the aorta." This statement may be regarded as essentially confirming Bellingham's views, although differing in the naming of the murmur, and in insisting on its preceding the second sound. Dr. Walshe¹¹ refers somewhat doubtfully to this subject, and quotes Skoda as believing that a diastolic murmur may be due to the friction of the blood against a roughened aorta before the closure of the aortic valves produces the accentuated second sound.

The second case occurred in my wards three months later, while my attention was still being directed to the subject. The patient was a man, 44 years of age, an upholsterer, admitted with considerable dyspnoea and anginalike suffering. He had never had rheumatism, and there was no history of strain, or of venereal aores. The physical signs gave no evidence of aneurysm, although a slightly increased dullness in the upper part of the sternum, with booming second sound at the aortic cartilage, suggested the idea of dilated aorta. The heart's action was heaving, and its dullness extended widely both to the right and left. At times, it was very irregular, and a very marked redupli-

¹ Auscultation and Percussion. Second edition. London, 1877. P. 283.

² A Treatise on Diseases of the Heart. Dublin, 1883. Part I, pp. 162-164.

³ The occurrence of such murmurs in aneurysmal disease, apart from valvular defects, is stated by eminent authorities to be much rarer than my own experience would have led me to suppose. I find, however, that M. Marey alleges that, in many cases of aneurysm, he has found such murmurs where the necropsy showed no valvular defect; and he proposes the indications of the cardiograph, showing the very sudden filling of the ventricle, as a new sign of aortic insufficiency. (Gazette Médicale de Paris, 1868. P. 548.)

⁴ Hayden: Diseases of the Heart. Dublin, 1875. p. 84.

⁵ Proceedings of the Pathological Society of Dublin, vol. iii, p. 263. Dublin, 1883.

⁶ Contributions to Cardiac Pathology. London, 1880. P. 12.

⁷ See abstract report in Edinburgh Medical Journal, vol. vi, p. 470; November, 1860, taken from Gazette Médicale de Paris.

⁸ Clinical Medicine. London, 1879. P. 247. The quotation here given is from

this volume; the subject is also discussed in his *Practical Treatise on the Diagnosis, Pathology, and Treatment of Diseases of the Heart* (second edition, Philadelphia, 1870, p. 213. One of his cases bears the date 1866.

¹¹ Walshe: Diseases of the Heart. Fourth edition, London, 1878. P. 103.

¹ This sentence expresses the opinion of the late Dr. Hilton Fagge. See his article on Diseases of the Valves of the Heart, in Reynolds's System of Medicine, vol. iv, p. 647.

² Illustrations of Epileptic Mania and of the Automatic Phenomena of Epilepsy; likewise of Maniacal Attacks following, and also taking the place of, Uræmic Convulsions. (Glasgow Medical Journal, December, 1880. See also Raymond, Archives Générales de Médecine, Mars, 1882.)

cation of the first sound, as heard to the left of the xiphoid cartilage; was often present. An interesting fact was noticed repeatedly, namely, the disappearance of the reduplication while the patient held his breath, although he could only do so for a very short time, owing to the dyspnoea from which he suffered. Occasionally, he had suffered from attacks of bronchitis. During his stay, the respiration approached at times the Cheyne-Stokes type; he had no hæmoptysis. The urine was albuminous and scanty. The patient's face was habitually florid, but appeared unduly congested. When, with all this, the limbs began to swell, as they did soon after admission, the idea of mitral disease was most strongly suggested to the mind. But the murmur, instead of being systolic or presystolic, was obviously diastolic; a pretty long, soft, or whiffing murmur, running off from the second sound, which could still be heard at the aortic cartilage with a booming quality. The murmur itself was audible both at the aortic cartilage and at the very tip of the xiphoid cartilage, perhaps most plainly at the lower half of the sternum. The idea of mitral disease, and the improbability of aortic incompetency, were so strongly impressed on my mind, that I considered again and again if this murmur could not be the diastolic portion of a prolonged murmur of mitral stenosis; but its complete dissociation from the first sound seemed to negative this conclusively. On trying the pulse critically, both with the finger and with the sphygmograph, no characteristics of the aortic regurgitant pulse were found. The idea of its being due to disease in the aorta, similar to the case already reported, next suggested itself; although it was a very unlikely occurrence to have another such case so soon after the first, it was discussed with the students in attendance. In this second case, however, we had only one murmur—not two as before—and it was of diastolic rhythm. Now, so far as I had seen the records of the kind of cases referred to, there had always been two murmurs found; or if one only existed, it was systolic; and the explanation offered by Dr. Bellingham and others, as to the production of the murmur, rendered it very unlikely that a diastolic murmur could be produced by a rough dilated and rigid aorta without a systolic murmur being present also. With this in view, I listened again and again for a systolic murmur at the base, but had to enter in the notes that no such murmur could be heard. The patient lived about six weeks in the ward, with increasing dyspnoea and dropsy; this, indeed, became extreme. The total duration of the illness was about six or seven months. He died suddenly, apparently from syncope.

The post mortem examination showed the heart greatly enlarged, the right side even more than the left; weight, 20 ounces. Before opening the organ, the aortic valve was tested with water, and found perfectly competent. The valvular structures were not thickened anywhere, but the mitral and tricuspid orifices were enlarged. The aorta was much dilated, and highly atheromatous, with calcareous plates and deposits of fibrine. A large thrombus occupied the right auricle, penetrating into the auricular appendage. Hæmorrhagic infarctions were present in both lungs. The spleen, liver, and kidneys had the appearance of chronic congestion.

These two cases afford, to my mind, conclusive evidence of the existence of a double murmur, and particularly of a diastolic murmur, of aortic origin, apart from aneurysm and apart from valvular incompetency. Of course, it may be contended that, with a dilated aorta, the valves were inadequate to prevent regurgitation during life. In reply to this, I can only say that the valves and aortic orifice appeared to the pathologist and myself, during the testing, so completely adapted to each other that, with every desire to do so, we were unable to admit such an explanation.

ON THE VALUE OF QUININE AND SOME OF ITS CONGENERS AS PARTURIENTS.

Read in the Section of Obstetric Medicine at the Fifty-second Annual Meeting of the British Medical Association.

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It has been said that, as civilisation advances, labour, on account of the more complex and artificial mode of living, from being a normal and physiological, tends to become more and more a pathological process. Whether this be so, it would be very difficult to prove, as many factors come into play, many of them opposed to each other. However, it may be, most medical men in large midwifery practice have met with many cases more or less tedious, yet without complication, which lost them much time and cost them anxiety. Active interference by any of the agents

usually described, medicinal or instrumental, was contraindicated, and the medical man would fervently re-echo the wish of the patient or her friends that something could be done for her, without knowing how or with what it was to be done. If the medical man be near home, he may leave the patient, with directions that he should be sent for when labour has fairly set in. But if he be many miles from home, and the patient be a multipara, he will feel difficulty in deciding to do so. If he return, he will be anxious as to how the patient is getting on. Of course, the medical man has no right to do anything which might endanger the patient for his own convenience, but this delay is often as bad for the patient as inconvenient for the doctor. The patient becomes exhausted with hope deferred and with frequent resort to various homely devices for quickening labour which have little power to effect it.

The causes of delay in the cases to which I refer are, in primiparae chiefly, some rigidity of the os or cervix, which I would call firmness, in contradistinction to the other term, which suggests greater degree of condensation of tissue, and, in multiparae, want of contractile power in uterus from loss of tone (1) caused by bad health, or want of rest; (when the latter is very markedly the cause, a dose of opium or chloral, and some hours' rest are indicated); (2) from anxiety or timidity; (3) inertia, cause undiscoverable.

In some of these cases, Barnes's dilators are of service; but anything that looks like instrumental interference, especially in an early stage, and when not urgently required (in their eyes, at least), would be received with much distrust by very many, and, in fact, would not be permitted; in other cases, they would not seem to be suited—certainly not so well as the means I propose.

It is generally accepted that ergot is contraindicated until the os is two-thirds dilated, and certainly if there be any rigidity. Now, here was a case, which I take from my note-book, a typical case, but resembling more or less closely a vast number I have met with.

CASE I.—I was sent for at 7 A.M. to see Mrs. B., primipara, a strong healthy young woman, who had labour-pains during the night. The patient lived four miles from my house. The os admitted the tip of the finger; the vagina was rigid. I left at 12 noon, and was sent for again next morning. The patient had pains during the night, now few and weak. I gave a hot enema. The os remained rigid. At 1.15 P.M., I gave five grains of quinine in powder; pains soon came. In half an hour, I repeated the dose; strong pains set in; the os dilated; the head descended to the perineum; but the pains failed somewhat. A third dose of quinine was given; the pains improved. The child being very large, the delivery was completed with forceps. All went well. Now, in this case, ergot was absolutely inadmissible; had it been used, one result of it from what I have had too good reason to know of its action, would have been the death of the child. The use of Barnes's bags would have been very difficult.

CASE II.—Mrs. C., in her second confinement, had had pains off and on for a week; she had little sleep at night. The pains were now (12 o'clock at night) few and weak. The vagina was not relaxed; the cervix was a tough or semi-rigid tube, admitting two fingers. At 1.40 A.M., five grains of quinetum were given, and two like doses at intervals of three-fourths of an hour. Pains set in strongly; the os gradually dilated. When the distended bag of waters began to press on the perineum, the pains slackened a little, and the patient asked for another dose of the medicine. I did not give it, but completed delivery by the vectis, about 7 o'clock. The uterus contracted well. What would have been the result of giving ergot here? On first seeing the patient, I had much debate with myself whether I should not have given opium or chloral to produce sleep, and then have had to wait many hours, or to go away with some anxiety of mind.

CASE III.—Mrs. W., multipara, in her eighth confinement, was a strong woman, but excitable and fanciful. At 9 P.M. labour was slow; the pains were inefficient. The head was at the brim; the membranes were whole; the os was of the size of a florin, dilatable. At 1 A.M. five grains of quinetum were given; in twenty minutes strong pains set in, which brought the head well into the pelvis. The child being large, delivery was completed by forceps.

CASE IV.—Mrs. M., in her eighth confinement, was a thin, anæmic creature. Labour had been lingering for four days; the pains were few and inefficient. The membranes were whole; the cervix was tubular, admitting two fingers. At 3.30 A.M., eight grains of quinetum were given; good pains started; the dose was repeated after some time. When the head was on the perineum, I gave ergot. She made a good recovery.

I could give other cases, showing that quinine or quinetum strengthened, where ergot failed; but it is better to state the conclusions derived from its administration in many cases during the last seven years.

On account of cost I tried murate of cinchonin, but it produced