

ON THE PULSUS BISFERIENS OF AORTIC REGURGITATION.<sup>1</sup>BY J. MICHELL CLARKE, M.A., M.D. CAMB.,  
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SHORTLY after reading the very instructive and interesting papers of Dr. Graham Steell on this subject,<sup>2</sup> and to which I here express my indebtedness, a well-marked example of the somewhat rare type of this pulse described by him came under my own observation. This variety of pulse is generally described as a form produced by aortic stenosis, but occurring much more rarely than the more usual modification of the pulse found in this lesion—namely, the anacrotic pulse. Dr. Steell defines the pulsus bisferiens as follows: "Both percussion and tidal waves are well represented, but the latter, instead of being rounded and sustained, forms a sharp angle. Moreover, the second wave begins low and the two waves reach the same level, or nearly so. The dicrotic wave follows and its degree of development varies." He states that the object of his "communication is to raise doubt as to the true nature of the pulsus bisferiens being necessarily associated with aortic stenosis." In his case the peculiar pulse was present in the left radial only; there was a double aortic murmur with systolic thrill. In a subsequent communication<sup>3</sup> he gives the results of the necropsy. Mitral stenosis was present, and the left ventricle was greatly dilated and somewhat hypertrophied. The aortic cusps were extensively diseased, but from the nature of the lesions Dr. Steell concludes that "there would not be a very extreme degree of obstruction to the blood passing from the ventricle into the aorta." No explanation of the pulsus bisferiens being unilateral was discovered. The circumference of the aortic orifice was one inch and three-quarters. I will now relate my own case, which was under observation at different periods during several years, in which the pulsus bisferiens was present at one period of the case in great perfection and imperfectly at another, and in which the post-mortem examination showed the presence of free regurgitation, but no obstruction at the aortic valve.

A man twenty-one years of age came under observation as an out-patient at the General Hospital, Bristol, on Jan. 1st, 1889, complaining of shortness of breath and palpitation. He had had syphilis eighteen months previously, but never rheumatism or rheumatic fever. He worked at a manufactory, and his employment involved exposure to great heat from boilers and severe muscular exertion in shifting casks weighing from 16 to 17 cwt. There was no suspicion of lead poisoning, and the illness had come on gradually without assignable cause. There was capillary pulsation and also much pulsation of the arteries. The apex beat of the heart was felt in the sixth space one inch outside the nipple. The area of dulness began at the third rib above, and extended from midsternum to outside the nipple line. A long blowing, diastolic, aortic murmur was preceded by a fainter systolic one. Both were heard all over the cardiac area, but loudest at the base, and the former was conducted down the sternum, and also to the apex. The pulse was 84, tense during the wave, and water-hammer in character. A sphygmographic tracing showed a well-marked rise due to the tidal wave and a distinct dicrotic wave; after the administration of ten minims of tincture of digitalis three times a day for two weeks, showed tracing of pulsus bisferiens, but with tidal wave not rising very high. I did not see him again until Oct. 13th, 1893, when he was admitted into the hospital with a history of cardiac symptoms for twelve months previously; he had managed to do his work, however, until a week before admission. He was a very temperate man as regards alcohol, but drank tea four times a day. He was anæmic, but well nourished. There was pulsation in the arteries of the neck and in the upper and lower extremities, and a thrill was to be felt in the subclavian and innominate arteries. Capillary pulsation was noted. The apex beat of the heart was heard in the sixth space half an inch external to the nipple; the maximum impulse was heard in the fifth space internal to the nipple. The area of dulness

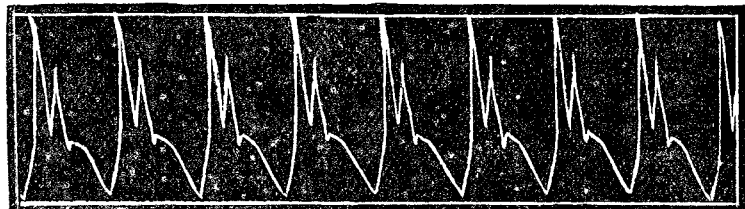
began above at the third rib, and transversely extended from the right border of the sternum to a point nearly one inch to the left beyond the position of the maximum impulse. A long, soft diastolic murmur in the aortic area entirely replaced the aortic second sound, and was conducted upwards but more distinctly downwards along the left border of the sternum, and was audible at the apex. The first sound at the apex was indistinct, and sometimes during his stay in hospital a systolic murmur appeared there. No systolic murmur was heard at the base. The pulmonary second sound was feeble. There was a little dulness at the base of the left lung, and the liver and spleen were both enlarged. There was no dropsy and the urine did not contain albumen. A cardiographic tracing at this time, taken over the seat of the maximum ventricular impulse, showed delay in the pulse wave (only an indication and not a good tracing of the brachial pulse was obtained); in the ventricular curve the rise due to the auricle was well marked, and a second notch near the top of the ascending line occurs, probably due to commencing contraction of the papillary muscles; both rise and fall are rapid and the line of the diastolic period varies, sometimes ascending rapidly and at other times being straight. On Nov. 7th, by which time he had much improved and was able to be up, the pulse was felt by the finger to give a double shock. Fig. 1 shows well the peculiar characters of the pulsus bisferiens; the dicrotic wave is plainly marked, following the tidal wave. On Nov. 14th the tracing still

FIG. 1.



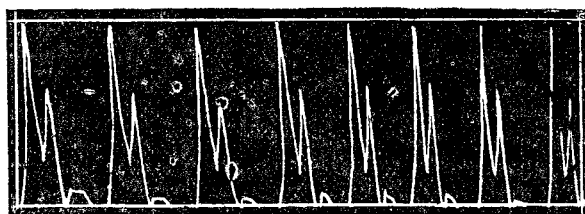
shows the double beat, but the percussion wave is now much greater than the tidal; the dicrotic wave is well marked and the pulse fairly full between the beats. (Fig. 2.) On Nov. 21st

FIG. 2.



the pulse showed more decided evidence of regurgitation, the artery being very empty between the beats, the rise of the lever during the percussion wave is higher than in previous tracings and the tidal wave smaller, both, however, being still separated by a distinct notch. (Fig. 3.) There was a distinct

FIG. 3.



interval between the carotid and radial pulses. The temperature was, as a rule, normal, but reached from 99° 5' to 99° 8' F. on a few occasions. He went out shortly afterwards, but was readmitted on March 16th, 1894. The pulsation in the vessels was now more marked than before. The apex beat of the heart was felt in the sixth space outside the nipple; dulness extended above to the second rib, as far out as the right border of the sternum at this level, and at the fourth rib from one inch beyond the right sternal border to half an inch beyond the nipple line to the left. There was some deep seated pulsation

<sup>1</sup> A paper read before the Bristol Medico-Chirurgical Society on Nov. 14th, 1894.

<sup>2</sup> Medical Chronicle, vol. xviii., pp. 229 and 313

<sup>3</sup> Loc. cit., p. 313.

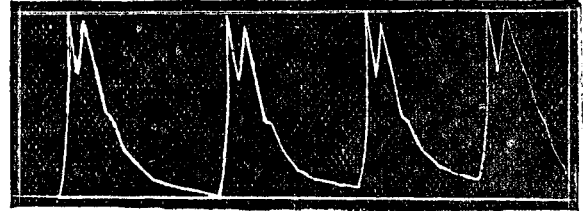
to the right border of the sternum in the second and third intercostal spaces (dilatation of aorta). Loud diastolic aortic and fainter systolic mitral murmurs were heard. The pulse-rate was 72. The sphygmograph showed the pulse of aortic regurgitation with notch of tidal wave plainly marked on down stroke and the dicrotic wave absent, no sign of pulsus bisferiens being present. The tracing taken from the carotid arteries with cardiograph (recording tambours and drum of Rothe's polygraph) showed a distinct notch about half way up the ascending line of trace. According to Landois and Stirling<sup>4</sup> in well-marked aortic insufficiency an anacrotic pulse wave occurs in the large vessels from the contraction of the auricle causing in the blood a wave which is at once propagated through the open mouth of the aorta into the large bloodvessels and is soon lost in the peripheral vessels. This I take to be the explanation of this notch in the carotid trace. After a short stay in the hospital he was relieved by rest and treatment and went to a convalescent home. He was admitted again very ill on Oct. 16th and died on the 22nd. He was too ill during this period to be disturbed by taking fresh tracings—in fact, he appeared to be dying on admission, but rallied a little for two days, then gradually sank, and died. The aortic diastolic murmur was extremely loud, with a marked systolic murmur at the apex, but none at the base. The arteries pulsated to an extraordinary degree, and on placing the chest-piece of the stethoscope over the bronchial or radial arteries a loud knock was produced by the pulse wave.

At the necropsy the lungs showed brown induration. Infarcts were found in the left lung and in the spleen. The pericardium was normal and contained no fluid. The heart was very large, extending upwards above the first rib, to the right nearly to the nipple line, and to the left to two inches beyond the nipple line. All the cavities were full of dark clot. It weighed, free from blood, thirty ounces. The right ventricle was dilated. The tricuspid valve admitted five fingers. The pulmonary arteries were competent, the segments of both being normal. The left ventricle was very large, dilatation being in excess of hypertrophy. The aortic valve was altogether incompetent, measuring three and a quarter inches in circumference (or 81 mm., 71 mm. being the normal according to Landois and Stirling). The valve segments were covered by masses of mostly fine, but in one or two places large, granulations. These extended freely from the margin and the ventricular surface of the cusps for a length of a quarter of an inch, in one part half an inch, into the blood stream. One cusp was almost entirely destroyed, being replaced by granulations, and the others were very much damaged; the sinuses of Valsalva were somewhat dilated, and the valve segments were pressed back against them, so that so far as could be ascertained they offered no resistance to the flow of blood. At one spot fine sago-grain-like granulations spread upwards on the aorta for a distance of half an inch, and downwards they extended to cover the ventricular surface of the anterior segment of the mitral valve, this flap and its chordæ tendinæ being thickly covered with them; the latter had only in two instances ruptured, although the granulations had run along them as far as the muscoli papillares. On the auricular surface of this anterior valve segment there were two rounded aneurysmal swellings with hard calcified walls. They opened freely into the ventricle, being formed by the giving way of diseased portions of the valve. One of them had also a pin-hole aperture into the auricle. The other mitral segment was also covered, though to a less extent, with fine granulations, especially abundant on its free margin. The mitral valve admitted five fingers. The aorta itself was only slightly dilated, and with the coronary arteries appeared normal.

The first point to notice is the appearance of the type of pulse of the pulsus bisferiens, imperfectly developed, however, after the administration of digitalis in January, 1889. I may say here that no digitalis had been given when this pulse in its perfect form was found in November, 1893, and that none of the other patients mentioned below had taken digitalis. All the tracings were taken at the same pressure of 3 oz. Next, in Tracings 1 and 2, in which this type of pulse is shown, the pulse is fuller between the beats—i.e., the pulse wave is better and longer sustained, the rate is slower, 72 and 76 to the minute respectively, as compared with Tracing 6 in which it was 90 to 84. As the pulsus bisferiens becomes less marked (Figs. 1, 2, and 3) it is the tidal wave which becomes weaker, while the percussion wave be-

comes more sudden (straighter upstroke) and higher. Further, cardiograms showed that there was delay in the transmission of the pulse wave. With regard to the question of the occurrence of the pulsus bisferiens in aortic stenosis or in regurgitation, I have given three tracings from other cases, in which there were, however, no post-mortem examinations. Fig. 4 was from a man aged seventy-five years, with enlarged

FIG. 4.



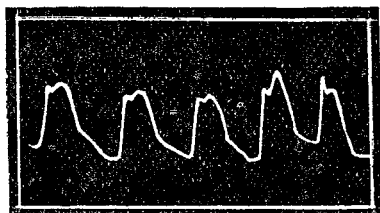
heart; the apex-beat was present in the sixth space, two inches outside the nipple; there were faint systolic, and long, loud blowing diastolic murmurs at the right base, conducted in the usual directions. There were capillary pulsation and marked throbbing of the arteries; the pulse was of a water-hammer character. The arteries were degenerated. A similar tracing was obtained from a man aged forty years. There was no history of rheumatic fever. The apex beat was heard in the fifth space outside the nipple, heaving in character; there was a soft blowing diastolic murmur at the second right costal cartilage conducted down the left border of the sternum and audible at the apex. The pulse was collapsing, 48 to the minute. No. 11 was from a man aged sixty years, with marked pulsation in the arteries. The pulse was strong, but collapsing. The apex beat was heard in the sixth space, one inch outside the nipple. Closure of the aortic valves could be plainly felt, and the second sound, loud and ringing, was followed by a faint whiffing diastolic murmur. The urine contained a trace of albumen. The arteries were somewhat degenerated. Though there was a systolic murmur in the case from which Fig. 4 was taken, the clinical signs pointed to predominance of regurgitation, and in the other two cases, there was no clinical evidence of aortic stenosis. These cases presented fair examples of the pulse under discussion. A marked tidal wave, with acute, not rounded summit, is often met with in tracings from aortic regurgitation. So far as clinical evidence is to be relied on, and I do not wish to lay too much stress on clinical signs unconfirmed by post-mortem examination in cases of this kind, they are in favour of the occurrence of this type of pulse in aortic regurgitation without at any rate marked stenosis. I think, however, that my first case affords conclusive evidence that the pulsus bisferiens may occur in simple aortic regurgitation.

In Dr. Steell's case the pulsus bisferiens was, curiously enough, only present in the left radial. There was no marked difference between the pulses on the two sides in my first case. I venture to suggest that the pulsus bisferiens of the type here described may be produced in deficiency of the aortic valves when the amount of regurgitation is not extremely great and the arterial tension is fairly well maintained, while at the same time the left ventricle is hypertrophied, and there is that delay in the transmission of the pulse wave which is often found in this disease. The percussion wave of the sphygmographic tracing is generally held, after the view of Mahomed, to be produced by the shock of the ventricular contraction, to be due to the inertia of the lever, and to be roughly a measure of the suddenness of the ventricular contraction. In the pulsus bisferiens of this type there is a great rise of the lever, due to the sudden contraction of the powerfully acting ventricle. Then, owing to the delay in the pulse wave, the lever has time to drop some distance. It is now raised by the pulse, or the tidal, wave, this again giving a high rise to the lever, but the angle traced out is an acute one, partly because the ventricular contraction is sharp and sudden, partly because regurgitation takes place. According to the amount of regurgitation the fall of the lever will be nearly to the base line, or if, as is the case in some of the above tracings, the regurgitation is not very considerable, it will take place more gradually. It should be noted that in the above case the pulsus bisferiens appeared the first time when the action of the heart had been improved by digitalis, and the second time when the patient had been for three weeks at rest in bed under

<sup>4</sup> Text-book of Human Physiology, vol. i., second edition, p. 159.

treatment in the hospital; in other words, it appeared when the physical signs and symptoms indicated improvement in the action of the heart and a lessened amount of regurgitation. When he came in the third time with the heart more dilated and all the symptoms and physical signs worse, the trace showed, not the pulsus bisferiens, but an ordinary one of aortic regurgitation. Further, in this case the peculiar pulse disappeared by the percussion wave becoming more marked, the tidal wave less so, and the artery more empty between the beats. It seems to me that the above type of pulsus bisferiens is of a totally different nature from that other one described as met with in aortic stenosis. I have given an example of this latter in Fig. 5, and the

FIG. 5.



same pulse is figured by Sir W. Broadbent in his description of the pulsus bisferiens.<sup>5</sup> Sir W. Broadbent associates this pulse with aortic stenosis, and attributes its second beat to "a reinforcement of a prolonged systole near its close." It is easy to see that such a pulse may easily be transformed into an anacrotic one; some of the beats in Fig. 5 and those in the upper of the two tracings given by Sir W. Broadbent are practically anacrotic beats. They are both found to occur in aortic stenosis, and the mechanism of production of this second form of the pulsus bisferiens given by Sir W. Broadbent corresponds with that held for the production of the anacrotic pulse—namely, that it is due to the prolonged effort of the left ventricle against obstruction. Hence I submit that two distinct varieties of the pulsus bisferiens must be recognised: (1) occurring in aortic regurgitation of the kind I have first described; (2) occurring in aortic stenosis, merely a modification of the anacrotic pulse. Dr. Graham Steell suggests that transition pulses between the pulsus bisferiens and the anacrotic exist. There is no difficulty in acknowledging their existence in the second variety of pulsus bisferiens. Of the first variety he says that "starting with a common type of simple aortic regurgitation sphygmogram, in which the tidal wave, while of considerable elevation, does not reach the level of the percussion wave and is unsustained and irregular. A probable result of narrowing of the aortic orifice might be interference with the percussion wave so that it became level with the tidal wave (pulsus bisferiens)"; and he suggests that in a still further degree, and with progression of the narrowing, the curve would be rendered anacrotic, the anacrotic is the expression of a great, the pulsus bisferiens of a minor, degree of obstruction. If the explanation of the first variety of the pulsus bisferiens that I have advanced above is correct, we should, on the contrary, expect that as the regurgitation increased and the arterial pressure fell the tidal wave would grow less and less marked and the pulse tracing become one of ordinary aortic insufficiency type. This happened in the fatal case I have described above, and would be the ordinary course of events in aortic insufficiency. In a case of mixed aortic obstruction and insufficiency the existence of the first variety of pulsus bisferiens would show the preponderance of regurgitation over obstruction, and that of the second variety the reverse. The appearance late in the case of the second type of pulsus bisferiens passing on into the anacrotic pulse would indicate increasing aortic obstruction. It might possibly happen that a disease of the aortic valves which at first allowed free regurgitation might in the further course of its development cause rather obstruction than permit regurgitation, and a pulsus bisferiens of the first type pass through the second type into the anacrotic pulse. Clinical experience would lead one to believe, however, that such a course of events in aortic disease is infinitely rare, if it ever occurs, and in such a case the mechanism of the production of the second type of the pulsus bisferiens and then the anacrotic pulse would be the same as in the ordinary cases of simple aortic stenosis, and not be, strictly speaking, a modification of the first type, but rather an entire change in the conditions

present. This case may, therefore, practically be left out of consideration.

Since writing the above I have read Dr. Graham Steell's paper,<sup>6</sup> in which he comes to the conclusion that his cases "seem to show that neither the anacrotic nor pulsus bisferiens possesses pathognomonic value as a sign of aortic stenosis."

Clifton, Bristol.

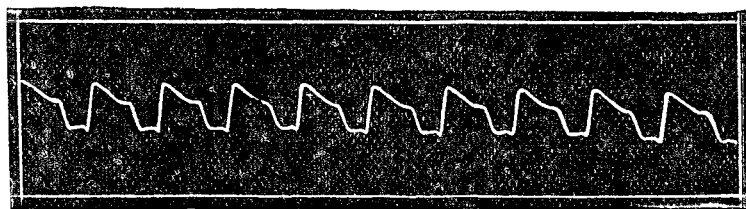
## ON AN AVOIDABLE ERROR IN SPHYGMOGRAPHY.

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I HAVE read with pleasure and instruction Dr. Graham Steell's article on the Pulse in Aortic Stenosis.<sup>1</sup> Those who know the careful character of Dr. Steell's work in this department of medicine will not lightly criticise it. It is upon the latter portion of his article that I wish shortly to comment. He remarks that a very striking feature of the pulse in two cases of double aortic disease which he relates was that there was an "inequality of the development of the peculiar characters on the two sides." He states also that after careful clinical and pathological examination of the arteries no explanation of this phenomenon was forthcoming. While I am not in a position to state that the explanation I am about to suggest applies to his cases, I am disposed to think that it is not unworthy of consideration. I the more readily make it in a spirit which I trust will not be misunderstood—namely, as one desirous of seeing the greatly extended use of the graphic method in the investigation of clinical problems affecting the circulation. I can most conveniently do so by relating a recent experience. On Nov. 29th, 1894, I had an opportunity of seeing, with Dr. Cecil Morgan, a young woman twenty-one years of age, who had suffered from rheumatic fever and was the subject of aortic valvular obstruction and regurgitation, with much the same physical signs as those related by Dr. Steell in the cases in question. I took a sphygmogram of both radial pulses. Fig. 1 is that of the right and Figs. 2 and 3 are those of the left radial artery. The

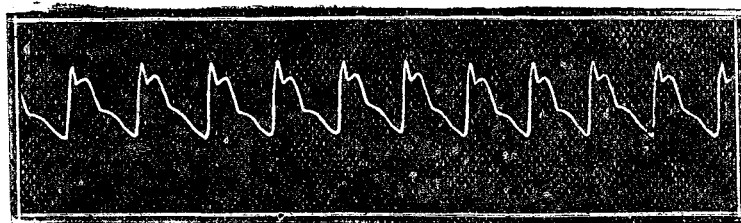
FIG. 1.



Sphygmogram of right radial artery, somewhat tense. Nov. 29th.

patient was in bed, on her back, and close to the edge of the bed. As she was in a very weak state, not wishing to disturb her more than possible, I took the sphygmograms as she lay, with the right forearm projecting somewhat outside the bed

FIG. 2.



Sphygmogram of left radial artery, unstretched. Nov. 29th.

and supported by Dr. Morgan. In this position there was some tension of the brachial structures generally. The instrument was used on the left arm, while it was completely

<sup>5</sup> The Pulse, p. 201, Fig. 47. Cassell and Co., 1890.

<sup>6</sup> THE LANCET, Nov. 24th, 1894.

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