THE VENTRICULAR RATE IN COMPLETE HEART-BLOCK.

By A. E. NAISH, M.A., M.B., B.C.CANTAB.,
M.R.C.P.LOND.,
ASSISTANT PHYSICIAN, SHEFFIELD ROYAL HOSPITAL.

In the great majority of cases published so far, when complete heart-block was present, the spontaneous ventricular rhythm has been between 30 and 40 per minute; most commonly in the neighbourhood of 32. Windle¹ has published a case in which the rate was usually 50, and at times rose to over 60 per minute. Lewis² has put on record a case in which, while the auricles were fibrillating, the ventricles beat regularly at about 90 for a few days, and Mackenzie³ mentions a similar case with a ventricular rate of 70; presumably complete heart-block was temporarily present in these two instances. In spite of these 3 cases it is very generally held that the idio-ventricular rate in man is a very constant one, varying but little from 32 per minute.

During the past year I have come across 2 cases in which the rate was not infrequently over 50 per minute, and another one in which the rate was as low as 17. I subjoin a short record of the two former, since it may well turn out that a comparatively rapid idio-ventricular rate is less uncommon than usually supposed, cases remaining undetected owing to there being no remarkably slow pulse-rate to call attention to the condition. As a matter of fact, one of the two had been for some time under the observation of a medical man without special attention having been drawn to his heart.

Erlanger and Blackman,⁴ having succeeded in producing chronic heart-block in 5 dogs, found that even under normal conditions there was a considerable difference between the lowest and highest ventricular rate. In 1 dog there was a variation of 40 beats per minute (maximum 74, minimum 34), and the average variation for the 5 dogs was nearly 25 beats per minute. Under abnormal conditions—for example, nervous states, running exercise, ether, etc.—the maximum rate was usually much higher. Individual dogs also varied from one another, the lowest under normal conditions averaging 35 beats per minute and the highest 50.

The first case, which I have reported more fully elsewhere,⁵ came under observation on April 26th, 1912, and from then up to June 10th showed complete heart-block with the exception of five days in May, when the rhythm was 2:1. During these six weeks the ventricular rate varied from 32 to 52 per minute. The slower rate was only seen after a shower of extra-systoles, which were usually produced by exercise. When resting the rate was most usually about 48, but was sometimes approximately 52 for considerable periods.

In the short stretch of tracing seen in Fig. 1 the average ventricular rate is 50 per minute. It will be seen that the rhythm of the ventricles is not perfectly regular, and in other parts of the tracing the irregularity was even more marked. In Dr. Windle's case irregularity was also noted.

In the second case, a man aged 66, who has shown complete heart-block for the past three months, the ventricular rate has varied between 38 and 54, being most frequently between 40 and 50. Fig. 2 shows the ventricles beating at approximately 52, and the auricles at nearly 65 per minute, a very low auriculo-ventricular ratio.

Apart from the possibility of such cases being overlooked altogether, the faster ventricular rate is more likely to give to a simulation of partial block. With the ventricles beating at 32 per minute the auricles have to beat as slow as 64 or as fast as 96 before there can be an appearance of partial block, and these are both rather unusual for the auricles under ordinary conditions. With the ventricles, on the other hand, beating in the neighbour-

hood of 40 per minute, it is obvious that a partial block must be much more frequently simulated, since the human heart commonly varies between 70 and 90 under conditions of rest or mild exercise. Wardrop Griffith has recorded 2 cases in which the difficulty of distinguishing between complete and partial block was considerable. Erlanger,⁶ in his classical experiments, sometimes found the same difficulty. He says: "Occasionally the ventricular rate in complete block may be an aliquot part of the auricular rate over long intervals of time, and in such instances the ventricular beats may so follow the auricular beats as to appear to be caused by them."

Fig. 1.

Fig. 2.

REFERENCES.

- ¹ *Heart*, ii, p. 102. ² *Heart*, iii, p. 279. ³ *BRITISH MEDICAL JOURNAL*, 1911, ii, p. 872. ⁴ *Heart*, i, p. 177. ⁵ *Quart. Journ. Med.*, vi, p. 196. ⁶ *Amer. Journ. Phys.*, xvi, p. 160.

THE AURICLE IN MITRAL STENOSIS.

BY
D. W. SAMWAYS, D.Sc., M.D., M.R.C.P.,
MENTONE.

THE somewhat complex series of murmurs audible during the course of a case of mitral stenosis seem to me to find an easy and natural explanation if it be granted that the contraction of the auricle may not cease when that of the ventricle commences, but may continue for a longer or shorter period to overlap it.

Such an assumption is by no means unreasonable.

The late Professor Potain always maintained, basing his conclusions on repeated observations, that the contraction of the auricle continued normally till the time of opening of the aortic valves.¹ Dr. Barclay wrote: "The two cavities (auricle and ventricle) do not contract and dilate alternately, but the contraction of the one overlaps to some extent that of the other."² Roy and Adami gave a series of simultaneous curves, showing the relation in time of the principal elements which united constitute the heart-beat. Those curves show the contraction of the auricle maintained in full force into the time of the out-flow of blood from the ventricle into the aorta.³ Dr. Galabin, commenting on his heart curves, and on those of Professor Marey, wrote: "From a comparison with Marey's tracings we have come to the conclusion that the beginning of the ventricular rise coincides with the first increase of the intraventricular pressure, whereas the auriculo-ventricular valves appear in general to close when the half or the third of the ventricular rise is accomplished."⁴

I am, therefore, not astonished that Dr. James Mackenzie says: "The curves of auricular pressure by different physiologists are very perplexing, some getting a rise of varying duration during ventricular systole."⁵

Now in mitral stenosis a tendency for the auricle to prolong its contraction still further is to be expected, as it would take longer to expel its contents through a stenosed than through a widely open orifice, and considerable evidence has accumulated to indicate that this is so.

If, then, the auricle in mitral stenosis can prolong its

contraction into ventriculo-systolic time, the following results would occur:

1. The presystolic murmur would go up to and into the first sound.

It would not necessarily stop with the advent of the first sound, for, as I showed in my article in the *BRITISH MEDICAL JOURNAL* of January 11th, 1913, the pressure on its contents which a contracting auricle can exert varies, roughly, inversely as the cube of its diameter.

The auricle having expelled most of its contents into the ventricle before the contraction of the latter commences, it could contract on the residue of its contents with far greater effect, and exert a far greater pressure; than the ventricle could on the full total of its contents. An auricle when small is more than a match for a ventricle when large; the auricle, in fact, is in somewhat the same position as the small piston of a Bramah press, which can overcome and force up the larger in virtue of its being the smaller. Blood would therefore flow from the contracted auricle into the large contracting ventricle, and the murmur, like the blood, would go into ventriculo-systolic time. The murmur would be crescendo in character, in harmony with the increasing pressure under which, as it contracts, the auricle can expel its residual contents into the ventricle. (In my communication "On the Genesis of the Venous Pulse"⁷ I pointed out why regurgitation from the auricle, in the absence of valves at the great veins, could not occur.)

2. A systolic murmur might be absent for years, though the auriculo-ventricular valve was incompetent.

Patients with mitral stenosis often live for long periods without any signs of regurgitation occurring, yet the auriculo-ventricular valve suggests from its appearance after death that it has been incompetent through, perhaps, most of the time. Why has this incompetent valve not leaked? or why has it leaked only towards the end of the illness? The rounded, hypertrophied auricle one often finds in these cases⁶ has for a long period prevented regurgitation by prolonging its contraction into ventriculo-systolic time. It has nearly emptied itself and become small—say, half its initial diameter—by the time the ventricle starts contracting, and it continues to try and drive its residual contents into the ventricle. It could now exert greater pressure on its contents, urging them forwards, than the ventricle could oppose to them, for the ventricle can never raise a pressure greater than that in the aorta without its contents escaping into it.

The auricle, therefore, would allow no regurgitation from the ventricle so long as the auricular contraction continued, and consequently the incompetent stenosed mitral valve would not leak. In fact, any leak would, so to speak, take place forwards from the auricle to the ventricle during the early part of ventricular contraction in cases of mitral stenosis accompanied with an auriculo-systolic murmur.

Incidentally it is interesting, as showing what auricles may do, to recall the case observed by Allan Burns where the ventricles were calcified, and the auricles had accomplished for a long time the whole work of the circulation.⁸

3. A systolic regurgitant murmur would replace the presystolic crescendo murmur when the auricle failed to contract to a sufficiently small chamber before the oncome of the ventricular systole.

If the auricle does not get small in time, because of the difficulty of expelling its contents quickly enough into the ventricle, the oncome of the ventricular systole would catch it before it had the physical advantage of being small enough to exert sufficient pressure on its residual contents to match or master the ventricle. Regurgitation into the auricle would then occur, a systolic murmur appear, the auricle would speedily dilate, and cardiac breakdown take place.

Rest or bleeding might temporarily give the auricle a chance of recovering its function, and becoming small in time to oppose the ventricular contraction. In that case the systolic murmur would again disappear, and be replaced by the presystolic. In time the breakdown would occur again in the same way, and probably be permanent, the auricle remaining dilated, and allowing regurgitation from the ventricle.

As soon as it is recognized that the auricle may be to the ventricle what David was to Goliath, simply because it starts first, the auricle will "come into its own" and be

respected as it deserves. Not recognizing this has misled and wronged many a good observer. Dr. Barclay wrote: "The more powerful contraction of the ventricle must overcome that of the auricle if there exists anything like antagonism between them." This false conclusion must have made it very difficult for him to accept the evidence of his own senses and affirm so correctly that "the essential character of this (presystolic) murmur is that it continues up to the first sound, during which it ceases."⁹

Dr. Dickinson, on the other hand, because the auriculo-systolic murmur was heard by him during the apex beat (as also it was by Professor Potain) concluded that it must be ventriculo-systolic in origin and regurgitant.¹⁰ He was forced to a false interpretation of a true observation through imagining relative muscle was everything and relative size was nothing in any struggle between the auricle and ventricle.

REFERENCES.

¹ Dr. Potain, *Clinique médicale de la charité*. ² Dr. Barclay, *Lancet*, March 16th, 1872. ³ Professors Roy and Adami, *Practitioner*, vol. xlv, p. 251. ⁴ Dr. A. L. Galabin, On the Interpretation of Cardiographic Tracings, *Guy's Hospital Reports*, vol. xx, 1875. ⁵ Dr. James Mackenzie, *Diseases of the Heart*, second edition, p. 109. ⁶ D. W. Samways, Mitral Stenosis: A Statistical Inquiry, *BRITISH MEDICAL JOURNAL*, February 5th, 1898. ⁷ *BRITISH MEDICAL JOURNAL*, April 13th, 1912. ⁸ Cited by Dr. A. Gérard, Sur l'oreillette gauche dans le rétrécissement mitral, *Thèse de Paris*, 1894. ⁹ Dr. Barclay, *Lancet*, March 16th, 1872. ¹⁰ Dr. Dickinson, *Lancet*, 1887.

The Goulstonian Lectures

ON

DEATH BY ELECTRIC CURRENTS AND BY LIGHTNING.

DELIVERED BEFORE THE ROYAL COLLEGE OF PHYSICIANS
OF LONDON.

By A. J. JEX-BLAKE, M.A., M.B., B.Ch. Oxon.,
F.R.C.P. Lond.,

ASSISTANT PHYSICIAN TO ST. GEORGE'S HOSPITAL AND TO THE
HOSPITAL FOR CONSUMPTION, BROMPTON.

LECTURE II.—DEATH BY ELECTRIC CURRENTS (concluded).

SOME excellent experiments further illustrating the importance, so far as life is concerned, of the quantity of current traversing the thorax in electric accidents were published in 1911 by Weiss and by Zacon. These were made on dogs anaesthetized with chloral; the electrodes were placed either in the mouth and rectum or on the head and leg, and the amperage, voltage, and the duration of the current received by the animal in each case were noted. Tracings of the femoral blood pressure and the respiratory movements are given. These authors concluded that the resistance of the dog's tissues to alternating currents (12 to 75 periods per second) was the same as to continuous currents, and that chloral anaesthesia gave no protection against electric shocks. With alternating currents given for a few seconds, death would occur when about 70 to 100 milliamperes traversed the thorax with the heart *en route*; with continuous currents, death was not caused unless the current was as large as 300 milliamperes, roughly speaking. If, however, smaller electric currents were administered for long periods, it was found possible to produce death by tetanus and asphyxia; thus currents of 35 to 45 milliamperes were too small to produce cardiac fibrillation, but after about ten minutes' application caused death by continued inability to breathe, and slow asphyxiation. It was observed that large currents—400 milliamperes or more—could be passed through the head with comparative impunity, the heart continuing to beat though rather irregularly, and the respiration stopping only for so long (35 and 58 seconds in two instances) as the current passed. With still large currents, however, thus passed through the head and central nervous system, respiration might fail to begin again when the current was turned off, and death from asphyxia would then occur. The resistance of the animals generally diminished about 10 per cent. during the passage of the current.