of the bundle was entire, for the part played by the haemorrhages is not fully entered upon. Yet we are distinctly given to understand by the authors that the nature of the lesion precluded conduction through the bundle six weeks previously. Given that such was the case, we have to fall back on the explanation already noticed in the case of Imchanitsky's experiments. It is possible in this instance that although the main bundle was destroyed, yet other small muscular paths of conduction were present, and that these were sufficient to enable the maintenance of co-ordinate contraction.

In concluding the evidence of heart-block in man as the result of a lesion in the bundle, there is a statement by Hering which cannot be overlooked. It occurs in a footnote added to the paper in which he deals with the experimental production of heart-block in the dog, and it is unfortunate that the facts are somewhat ambiguously expressed. Nevertheless, they appear to bear only one interpretation. The statement by Hering is to the effect that he has succeeded in reviving, by perfusion, the heart of a man who died some hours previously, and he affirms that he observed nothing in his experiments on this heart which he had not already noticed in his experiments upon the heart of the dog.

Surveying the evidence as a whole, we may say that, knowing that heart block can occur in man, and that it may be associated with lesions of a bundle which has been shown to be essential to conduction in the lower animals, we are justified in concluding that heart-block in man may arise as the result of such a lesion. We are not justified at present in assuming that it will invariably arise from such a lesion, or that it is invariably dependent upon such a lesion.

In associating a composite of clinical symptoms exclusively with a particular lesion, the ideal evidence required is twofold. First, the lesion must be invariably found when searched for in cases succumbing to the affection; and, secondly, the lesion must be found to give rise invariably to the clinical symptoms which characterize the disease. But such a collection of evidence is frequently not forthcoming in its entirety, and we have to be content with evidence which is less complete, and consequently with conclusions of a less positive nature. So it is in the case of heart-block; the evidence goes to show that heart-block is frequently, and, indeed, so far as we know, invariably, associated with a bundle affection or with a lesion from the nature of which a bundle affection cannot be excluded. On the other hand, further attention has yet to be given to the histology of the bundle in other conditions, and this the more as already lesions of the bundle, not unlike those found in some cases of incomplete heart-block, have been reported, where heart-block of any degree could not be demonstrated. This statement refers to the preliminary results of the examination of hearts dying with the heart irregularity known as "nodal rhythm," an irregularity, be it observed, in which conduction of impulses from auricle to ventricle cannot be shown.

In describing heart-block in certain instances as a result of a definite pathological lesion of the auriculo-ventricular bundle, we must ever bear in mind that such a conclusion is open to limitation. It must be acknowledged that in man alone such a proposition is barely demonstrated, and that it is only when we survey the facts derived from all sources, and more particularly from animal experiment, that it becomes, at the least, highly probable that such a proposition is justified, and that we know that there is accumulated evidence which points in the clearest manner in this direction, carrying the proposition almost, if not quite, beyond the limits of hypothesis.

Two further cases reported by Professor G. A. Gibson (loc. cit.) have now received complete investigation, and details kindly sent by the author allow the statement that in both a definite lesion of the bundle was present. Tracings and histological evidence have been obtained in both instances. Another case has been observed by Dr. Byrom Bramwell.

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RETARDATION OF THE PULSE WAVE IN AORTIC REGURGITATION.

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THE fact that there has been so much difference of opinion as to the retardation of the pulse wave in aortic regurgitation has made it a matter of interest to use Mackenzie's polygraph to take a simultaneous record of the apex beat and pulse in all the cases of aortic regurgitation coming under my observation in the last two years, numbering thirty in all, and for comparison twenty-five records have been made from other cases.

Though there is no time marker on Mackenzie's instrument, which consists of a recording tambour and receiver attached to a Dudgeon's sphygmograph, it is quite easy to measure the time if the pulse-rate be taken, for the length of the pulse wave on the record corresponds to one-seventy-second or one-eightieth of sixty seconds if the pulse-rate be 72 or 80, and so on.

In taking the records the clockwork should be stopped before the tracing paper has completely passed through, so as to get a vertical line from the sphygmograph and the cardiograph. With a pair of calipers the distance is measured from the commencement of a pulse wave to the sphygmograph vertical line, and then from the commencement of the rise of the apex beat to the cardiograph vertical line. In a few cases there is a sucking-in at the apex beat instead of the outward thrust; this should be watched for, as the cardiogram is then upside down. The difference between these two distances is marked on paper, and, with care, by means of the calipers it can be found how often this goes into the length of a pulse wave, and, the time taken by that being known, the delay is easily worked out. For instance, if the pulse-rate be 80, and the difference between the two distances be found to go four times into the length of a pulse wave, the delay between the apex beat and the pulse is one-quarter of one-eightieth of sixty seconds, which is three-sixteenths of a second, approximately the normal one-fifth. For simplicity's sake, the lengths of delay in these cases have been given as one-sixth, one-fifth, one-fourth, one-third, according to which is nearest to the more complicated fractions.

The cases have been divided into two groups: (1) Those in which the amount of delay between the apex beat and the pulse is normal or below; (2) those in which it is above normal.

On studying these groups, it is at once apparent that there is very frequently considerable increase of the interval between the apex beat and the pulse wave in severe aortic regurgitation, while in slight rheumatic cases and in the majority of the atheromatous and syphilitic cases the interval is the normal one-fifth or less; of course, in these latter there is usually not much regurgitation.

When simultaneous tracings of the carotid and the pulse were taken in cases in which the pulse delay was considerable, it was found that the interval between the carotid and the pulse was only slightly greater than the normal, so that the main delay evidently took place between the apex beat and the aorta. It occurred to me then that this delay might be due to the increased size of the heart, so I measured the distance from the apex to the aortic cartilage in subsequent cases; but, as will be seen from the accompanying tables, there is no relationship between the size of the heart and the time of arrival of the pulse at the wrist, some of the largest hearts showing no delay. The systolic blood pressure also seems to have nothing to do with the amount of delay.

There is a rough correspondence between the extent of the aortic regurgitation and the amount of the delay, the greater the one the greater the other, and I think there may be found to be some relation to the compensation of the valvular lesion, there being increased pulse delay with failing compensation in some of these cases; of the seven cases with delay of one-third of a second I know that three are already dead.

The tracings taken from other than aortic cases show that increased pulse delay is not peculiar to aortic regurgitation. There was delay in two cases, showing signs of adherent pericardium. Both had an apex beat hardly moving on rolling the patient from one side to the other, diminished respiratory movement in the epigastrium,

TABLE I.—Delay One-fifth of a Second or Less.

	Age.	Disease.	Length of Heart.	Delay.
M. M.	66	Slight atheromatous aortic regurgitation; blood pressure, 190	in. 8	Under one-fifth.
G. F.	59	Slight atheromatous aortic regurgitation; blood pressure, 170	8½	One-fifth.
J. B.	57	Slight atheromatous aortic regurgitation; blood pressure, 134	8	One-fifth.
I. B.	56	Slight aortic regurgitation; syphilitic? Blood pressure, 145	8½	One-fifth.
E. D.	60	Atheromatous aortic regurgitation	—	One-fifth.
J. B.	78	Atheromatous aortic regurgitation	8½	One-fifth.
J. V.	51	Aortic regurgitation; syphilitic?	8	One-sixth.
A. C.	60	Ditto	—	One-sixth.
R. W.	45	Slight syphilitic aortic regurgitation, and ataxy	—	One-fifth.
W. P.	35	Slight aortic regurgitation	6½	One-fifth.
F. S.	18	Ditto	—	One-fifth.
E. R.	16	Moderate aortic regurgitation	7½	One-fifth.
A. W.	25	Severe aortic regurgitation	—	One-fifth.
D. B.	15	Slight aortic, much mitral regurgitation	8	One-sixth.
E. S.	18	Aortic and mitral regurgitation, dropsy	9	One-sixth.
A. G.	12	Mitral regurgitation	6	Under one-fifth.
E. T.	49	Fusiform aneurysm of aorta; blood pressure, 165	9	One-sixth.
H. W.	50	Aneurysm of second part of aorta; blood pressure, 145	7½	One-fifth.
W. W.	56	Dilated heart from high tension	9	One-fifth.
G. W.	60	Chronic Bright's disease	—	One-sixth.
T. P.	—	Ditto	—	One-fifth.
J. M.	25	Acute Bright's disease	6½	One-fifth.
A. B.	30	Palpitation	—	One-fifth.
B. W.	21	Ditto	6½	One-sixth.
G. T.	25	Ditto	6½	One-sixth.
A. P.	53	Old Graves's disease; blood pressure, 135	7½	One-fifth.
G. S.	68	Paralysis agitans; blood pressure, 130	5½	One-fifth.
M. P.	41	Intermittent heart	—	One-sixth.
A. H.	20	High tension	7	One-fifth.
P. W.	44	Ditto	7½	One-fifth.
G. W.	—	High tension; blood pressure, 170	7½	One-fifth.
A. A.	—	Normal	6½	One-sixth.
E. B.	30	Ditto	6½	One-fifth.

TABLE II.—Delay of Over One-fifth of a Second.

	Age.	Disease.	Length of Heart.	Delay.
J. B.	78	Atheromatous aortic regurgitation	in. 8½	Over one-fifth.
G. R.	23	Slight aortic regurgitation	8	One-fourth.
D. R.	20	Ditto	7	Two-fifths.
F. H.	46	Dilated aorta and regurgitation	—	One-fourth.
R. L.	63	Rheumatic aortic regurgitation and stenosis	9½	One-fourth.
R. K.	—	Rheumatic aortic regurgitation and stenosis	8½	One-fifth and one-fourth.
F. W.	39	Severe aortic regurgitation, syphilitic	8	One-fourth and one-third.
A. W.	30	Aortic regurgitation	—	One-fourth.
W. S.	23	Ditto	—	One-third.
G. B.	—	Ditto	—	One-third.
E. R.	16	Severe aortic regurgitation; blood pressure, 125	8½	One-third.
G. S.	37	Severe aortic regurgitation; blood pressure, 125	9	One-third and one-half.
A. W.	—	Severe aortic regurgitation; blood pressure, 125	—	One-third.
R. M.	34	Severe aortic regurgitation; blood pressure, 125	—	One-third.
D. G.	15	Acute aortic endocarditis	—	One-third.
W. C.	—	Mitral regurgitation and adherent pericardium	11	One-fourth.
A. M.	14	Mitral regurgitation and adherent pericardium	7	One-third.
J. L.	30	Congenital heart; pulmonary stenosis?	12½	One-third.
A. E.	23	High tension; blood pressure, 135	6½	One-fourth.
F. F.	19	Ditto	7	One-fourth.
A. A.	56	High tension; blood pressure, 140 to 160	7½	One-fourth.
P. N.	60	Atheroma	—	One-fourth.
J. L.	61	Chronic Bright's disease	7	One-fourth.

increased cardiac dullness, systolic retraction of the eleventh and twelfth ribs in the back, and a heart more embarrassed than the murmurs led one to expect, and both had histories of pericarditis.

There was also delay in a case of congenital heart disease. The patient was a woman of 30, dusky coloured; but not actually blue; her apex beat was in the posterior axillary line in the ninth intercostal space, and the distance

to the aortic cartilage was 12½ in., a phenomenally large heart, which possibly may account for the delay. There was a loud systolic murmur everywhere, of greatest intensity in the second and third left intercostal spaces.

The other cases in which there was abnormal delay had high tension, and sometimes atheromatous pulses; there were, however, similar cases showing only normal delay, but I found no low tension case with increased delay.