the heart should have suddenly stopped in diastole, but it is interesting to note the greater calcium content in the left ventricle than in the right.

[The treatment of mitral stenosis was also dealt with, but as this address was very long Sir James Barr has reserved that portion of it for publication on a future occasion.]

A Lecture

ON THE

OCCURRENCE OF HEART-BLOCK IN MAN AND ITS CAUSATION.

DELIVERED AT THE LONDON HOSPITAL MEDICAL SCHOOL
ON NOVEMBER 13TH, 1908,

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[In the opening remarks it was assumed that the audience was aware of the evidence for the myogenic theory in the lower and cold-blooded vertebrates, that the heart beats have their origin near the mouths of the great veins and that the contraction passes as a conducted wave from sinus to auricle and auricle to ventricle.]

We may proceed at the present time to a consideration of certain general propositions which have an intimate bearing upon clinical work, and to a discussion of the experimental and clinical evidence upon which the modern view is based, that in mammals the auriculo-ventricular bundle is the path of impulse conduction between auricle and ventricle. It will be obvious that, in demonstrating that the auriculo-ventricular bundle is the means of stimulus transmission, we shall not only prove that this bundle, already shown to be morphologically identical with the corresponding bundle in the cold-blooded heart, has also identical physiological functions with these structures, but shall at the same time strengthen the position of the myogenic theory as a whole.

THE EVIDENCE FOR CONDUCTION BY MUSCULAR CONTINUITY IN MAMMALIA.

The fact that dissociation of the usual co-ordinate sequence of contraction in the chambers of the mammalian heart can occur has been known for many centuries, though the cause of it remained for a long while obscure. Harvey, in his essay on the movements of the heart, stated that he observed the prolongation of the interval between auricular and ventricular systole which occurs in the dying heart, and noted an occasional ventricular response following upon several auricular contractions under similar circumstances.

Two years after the publication in which His described the auriculo-ventricular bundle, the same observer attempted, in conjunction with Graufner, to destroy the connexion between auricle and ventricle in the heart of a rabbit, and apparently with success. In a preliminary communication he records the fact that complete dissociation of the auricular and ventricular rhythm was produced, though neither detail nor tracing is given.

In 1894 Humblet made further observations. He operated on the heart *in situ*, opening the right auricle, injuring the region of the bundle, and then reclosing the wound. The experiments were performed on the dog and yielded no satisfactory results. The method was discarded for that of perfusion. Using Locke's fluid and perfusing the dog's heart, he recorded the fact that cuts or cautery wounds in the neighbourhood of the bundle have no effect, but that when the bundle itself is interfered with an arrhythmia is produced. In the single tracing which is given complete dissociation appears to be demonstrated.*

The first important series of experiments were performed more than ten years later by Hering upon the perfused dog's heart. He showed that sections traversing the

bundle produced complete heart-block, and illustrated his communication by excellent tracings. He further found that no other incisions would produce a similar result. Almost simultaneously Erlanger published his first observations. In the first instance, an attempt was made to pass a ligature through the heart walls so as to include the bundle. In seven experiments performed, heart-block was obtained on one occasion only. An auriculo-ventricular clamp was then tried, a method similar to that employed by Gaskell for the cold-blooded heart. In seven experiments heart-block was obtained in two instances. The third method introduced yielded results of far greater value, and was finally adopted permanently owing to the uncertainty and unsatisfactory nature of the earlier attempts.

Erlanger devised a special clamp, one blade of which was introduced into the left ventricle and driven underneath the bundle as it lay on the septum. The clamp could be introduced and tightened without interfering with the remaining heart musculature to any extent.

The results obtained in this way have been very striking, and are in the main a complete confirmation of the similar clamp experiments of Gaskell. The following is an abstract of Erlanger's conclusions:

By compression of this bundle all stages of heart block may be obtained. These include:

- (a) An increase of the intersystolic pause.
- (b) An occasional ventricular silence.
- (c) Regularly occurring ventricular silences—for example, one silence in ten, nine, eight, seven, six, five, four, three, and two auricular beats.
- (d) A 2:1 rhythm.
- (e) A 3:1 rhythm.
- (f) Complete heart-block.

The compression of the bundle was subsequently confirmed in Erlanger's experiments by careful autopsies and histological examinations carried out by Retzer. The protocols of the seventeen experiments given in the first communication show that a successful result was obtained in sixteen cases, and that these were the experiments in which the bundle had been damaged. The work has been extended and amplified by the same writer in conjunction with Hirschfelder in researches in which operations of a similar nature were performed with similar results. Erlanger,† in conjunction with Blackman and Cullen, has also been able to damage the bundle in seven dogs, the animals surviving the operation for a varying time, and presenting various grades of partial block or permanent and complete block. The experiments are of importance in that they demonstrate the permanence of the result, and suggest the absence of other paths of conduction, which might only respond to a special call (Erlanger). They also show the possibility of producing syncope, attacks and epileptiform seizures experimentally by this means. Heart-block has been similarly produced by damage to the bundle by Tabora. Biggs has recently produced it in the rabbit by incisions.

The strict conclusion which the experiments so far cited justify is that in the particular animals investigated the conduction of impulses depends upon the integrity of a very limited tract of tissue, and upon the integrity of this tract alone. Further, this tract includes a strand of specialized tissue which has been microscopically demonstrated as being in continuity with auricular and ventricular musculature. Accordingly, we are justified in assuming that it was by this specialized bundle of fibres, and by this bundle alone, that impulses were carried from one chamber to the other in these experiments.

But are we justified in concluding that the auriculo-ventricular bundle is the sole path of conduction in all species of mammalia, and in all individuals of such species? It appears that we must regard the bundle as one of several remains of the primitive tissue of which the embryonic heart is composed, and that during the phylogenetic, if not during the ontogenetic, development of the heart, the connexion between auricles and ventricles was at one time around the whole ring. The amphibian and reptilian heart still shows such a junction. Now, it would be a very surprising fact, if such a fact should be eventually demonstrated, that complete segregation of the joining fibres has occurred in mammalia, and that such segre-

* Humblet (*Arch. Internat. d. Physiol.*, 1905-6, iii, p. 330) reports seven successful experiments upon eight dogs. *Post-mortem* confirmation of the lesions was obtained.

† In Erlanger's hands the experimental production of heart-block has become a part of the preliminary technique, a fact of very great significance, for it demonstrates the certainty of his method.

gation is universally present in mammalia. Individual variation in the same species, and the lesser grades of developmental anomaly, are ever-present phenomena, and are compatible with undisturbed physiological function. In fact variations of the sort must be regarded within certain limits as normal. Variation in structure from species to species may be wide, and concomitant variation of function may be of too subordinate a character to attract or fix our attention.

It must be remembered that the demonstration of the isolation of the bundle is a far from easy task in any particular case. It requires a laborious and studious examination of numerous serial sections. It is a far more easy matter to demonstrate that tissue continuity is present than that continuity is absent; and most of the evidence which we have, while certainly pointing in the direction of the isolation of the connecting fibres, is limited to a few species and to a few individuals in each of these species. It must also not be forgotten that it was a long time before careful search revealed the presence of the bundle, and further, that Kent believed that he saw other connexions. The view that there may be variation is strengthened by the statement of Biggs that his experiments go to show that in rabbits the bundle occasionally runs a somewhat abnormal course.

The experiments so far considered all point in one direction, but there remain some observations of a contradictory nature, which, though meagre by comparison, and therefore to be accordingly judged, yet cannot be entirely neglected. In a recent note Imchanitsky states that the heart may show co-ordinate contraction subsequent to complete destruction of the bundle. The completeness of the section was confirmed subsequently by histological examination. Kronecker has made somewhat similar observations on the rabbit. Biggs found also in the same animal that section of the two main branches of the bundle is without appreciable effect, but that in all cases the last section, high up and completely dividing the septum, produced instant stoppage of the ventricles. In regard to such observations there are several important considerations. Some of these have been already noted. It has been customary, since the work of Tawara on the anatomy of the bundle and its connexions, to hold that the main bundle throws off no branches until it divides for the supply of the respective ventricles. But this is a proposition which it is extremely difficult to prove satisfactorily, and it may be that in certain instances, more especially in the rabbit, small divisions of it are sent out at a higher level than has hitherto been suspected.

It may be said that while the majority of workers have accumulated evidence for the particular animals investigated, which allows of no other conclusion than that in these animals there is segregation of the conducting fibres, yet it cannot at present be absolutely stated that this bundle is throughout its whole length the sole conducting path in all mammalia.

The primary importance of the discovery of the bundle lies in the fact that it allows us to accept the evidence of Gaskell for the dependence of conduction upon muscular continuity, and permits us to apply the theory of myogenic conductivity to the mammalian heart. So long as the connexion between the mammalian auricle and ventricle remained undiscovered this was impossible. The junction was predicted and sought from this special point of view, and finally it was found.

Without entering more fully into the minor difficulties and discrepancies of the experimental work, we may finally conclude that as a rule the auriculo-ventricular bundle and its branches is the sole path of impulse transmission between the auricle and ventricle, but that there is every possibility that it is a structure which, like all other animal structures, is open to variation in its position and arrangement, the extent of which variation it is at present impossible to estimate with certainty.

The expression *muscular continuity* has been used advisedly in preference to the expression *continuity of contraction*. When, as in the case of the amphibian and reptilian heart and as in the case of many mammalian hearts, the structural continuity is by tissue bearing histological features closely resembling those of the remaining cardiac tissue, we have every right to assume that conduction is not only by structural continuity,

but also by continuity of contraction. But when, as in the case of the ox and the sheep, continuity is established by a highly specialized tissue, which lacks richness in cross striation, and consists essentially of homogeneous protoplasm, such an assumption must be made with greater care. For while we are aware that cross striation is not essential to contraction, as exemplified by involuntary muscle and the tissue of which jelly fish are composed, yet the direct demonstration of contraction in Purkinje substance, or in tissue closely allied to it, is still wanting, and it is quite conceivable that the passage of the impulse occurs, not as a result of the actual contraction, but in virtue of associated chemical or physical changes, which may or may not be accompanied by actual or perceptible contraction. The conduction of impulses through auricular tissue, in the absence of contraction sufficiently prominent to be registered, is well known. At the same time, it should be understood that the modern conception, to be strictly harmonious, distinctly involves the idea of continuity of contraction.

The opinion is not infrequently expressed that the auriculo-ventricular bundle, having a sectional area in man of a little more than 2 sq. mm., is small, and that it is not conceivable that a strand so minute should supply so large a mass of muscular substance as that contained in the ventricular walls. The doubt which is entertained is based on false ideas. There is no reason to compare the supply of a muscle by its nerve and the supply of the ventricles by the bundles—a comparison which is distinctly implied. There is every reason to believe that the conditions are entirely different. Again, such a comparison is not in accord with the facts of the case. The size of the ventricles is immaterial. It is sufficient that a very limited part of the ventricular wall should contract for the whole to follow suit. There is no reason to assume the supply to each separate fibre or bundle of ventricular fibres; there is only reason to assume the supply of a sufficient number of fibres at different points of the ventricular walls, which may by their contraction pass on the impulse in such a way that a co-ordinate contraction of the whole mass may result. In the case of muscle and nerve we know that there is representation. That is to say, that certain ganglion cells have axons which run from them to particular parts of the muscle supplied, and to these parts only. In the case of the heart muscle and its supply by the bundle the position is entirely different. Biggs states that section of either the right or left branches of the main bundle, or even section of both, does not appreciably affect the ventricular systole. Moreover, he finds that the bundle itself may be reduced to a very narrow thread and the impulses still pass. There may be, as the result of such interference, a temporary block, complete or partial, but recovery takes place, and the remaining strands take up the function of the whole, so long as these remaining strands are not so far damaged by the section as to be incapable of recovery. A similar interpretation is to be placed on Gaskell's experiment, in which an isolated strip of muscle was taken from the apex of the ventricle of the tortoise. The act of isolation damaged the conducting powers of the whole strip, but recovery was frequently demonstrated. When the auriculo-ventricular junction is seized in the jaws of a clamp, it is observed that the degree of heart-block produced is in proportion to the tightening of the clamp. It is the whole thickness of the bundle which is damaged, and recovery is not so marked. In brief, there is no evidence which allows us to rationally ascribe the results of a local and limited damage to the bundle to a limitation and consequent increased resistance of the path. The auriculo-ventricular bundle is an intimate network which supplies the ventricles through the medium of a still more complex network. Histologically intercommunication is free in every direction; why, then, assume the impossibility of functional interchange?

The connexion between auricle and ventricle may be likened in many respects to a train of gunpowder laid between two magazines—light the one and you explode the other. Destroy half the thickness of the train by pouring water on it and the explosion travels equally well through the other half; but if in destroying the first half you damp the second half, then the conduction of the

explosion will be delayed, and the flash will only pass unimpeded when the remaining powder dries.

In 1885 Chauveau described a case of heart affection, and was able from tracings taken from the radial pulse, apex beat, and carotid to recognize that the auricle and ventricle were beating at different and entirely independent rates.

In 1899 Wenckebach and His both described cases of heart-block in man, and suggested as its cause a lesion of the auriculo-ventricular bundle. Since this time very numerous cases have been placed on record, and the main facts in regard to the condition are rapidly receiving elucidation. The condition now known as Adams-Stokes syndrome may be defined as one in which the symptom complex, bradycardia and epileptic fits, occurs; it has been recognized for over eighty years, but it is only quite recently that evidence has so far accumulated as to point to the condition as being due in certain instances to a pathological lesion in the auriculo-ventricular bundle. For the time being we may deal with simple propositions, and inquire, first, for the proofs of heart-block in man, and, secondly, for the evidence which refers it to a bundle lesion.

THE EVIDENCE OF HEART-BLOCK IN MAN.

In the first place we have the evidence from animal experiment, which has shown the possibility of heart-block. In the second place we have the evidence derived from clinical observation. The last does not consist alone of the findings of venous pulse tracings. It depends also upon cardiographic and oesophageal curves, and upon actual observation with the fluorescent screen, the evidence of the stethoscope, and upon the electro-cardiogram.

In certain cases of bradycardia or slow pulse, the fact that the auricles and ventricles are beating at different rates may be both recorded and observed. The radial pulse is slow, beating usually at a rate of about 30 per minute. The beats are large and forcible, each systole is prolonged, and the pause before the next beat is greatly increased. During the long pause of diastole the blood-pressure falls considerably, as is indicated by the fact that the line of the pulse beats in diastole is almost horizontal.

At the apex beat there is no difficulty in recognizing the waves due to ventricular systole, and auscultation reveals a first and second sound with each. In the jugular curve the pulsation due to ventricular systole is also observed, each beat producing a "c" and "v" wave.

But in addition to the events which are obviously of ventricular origin, physical signs of different origin may appear at either one of the points mentioned.

In the jugular tracing there are numerous waves arranged at regular intervals. They are small and regular, and may be twice, three or four times (etc.) as numerous as the waves attributable to the ventricle. In cases of partial heart-block one of these waves from each group will precede each ventricular wave, and is consequently presystolic in time. The rate of the waves may be sometimes identified with the rate of contraction of the auricle by means of the fluorescent screen, and they are due to the contraction of that chamber. The left auricular contractions may be recorded from the oesophagus, at the apex beat and occasionally they may also be indicated upon the radial pulse. In each situation they maintain the same relationship to the ventricular beats. The contraction of the right auricle may, and often does, leave its impression on a curve taken from the epigastrium. Such records show the contractions at similar intervals. Lastly, faint sounds may be audible over the base of the heart which can only be attributed to the same source.

With these physical signs there can be little doubt in a typical case that the rate at which perceptible contractions of auricle or ventricle are occurring is fully recognizable, and little hesitation is experienced in asserting it as a case of heart-block.

Perhaps the most perfect example of a record of heart-block is that given by Einthoven, a curve which represents the electrical changes of potential in the limbs in a case of Adams-Stokes syndrome. The waves attributed to auricular and ventricular contraction are perfectly regular and clean; when they fall together they are accurately superimposed, and as a whole present incontestable evidence of the complete dissociation of auricular and ventricular rhythms.

By means of simultaneous tracings it has been shown that all those grades of heart-block which have been observed by Gaskell in amphibia and reptilia, and which have also been demonstrated by Erlanger in mammalia, also occur in man. Further, numerous records have been obtained of the transition stages, or the passage of a condition of slightly diminished conductivity into one of graver impairment of that function, or complete dissociation of the rhythm of the two chambers.

The earliest evidence of impaired conductivity in man presents itself, as in animals, by an increase in the *a-c* interval, an interval which, as we have seen, is given by the venous record, and which normally is extremely constant, and does not exceed one-fifth of a second. The interval in the pathological state may increase to one-half second or more before the next stage is reached. This consists in the dropping out of an occasional ventricular response, and as a rule there is a gradual gradation of the *a-c* period up to the point of the failure of the ventricular response in any given instance of it.* Later, the failure of response becomes more frequent, a ventricular systole drops out at regular intervals of several auricular beats. Still later the rhythm of the auricle to that of the ventricle may be as 2:1, 3:1, 4:1, etc., till finally all relation between the rhythms vanishes, and they can be demonstrated as totally distinct.

The establishment of complete dissociation involves the development of an entirely new rhythm—namely, that of the ventricle—and the events which are associated with its establishment are not as yet fully understood. Suffice it at present to say that a ventricular rhythm in man as a result of disease has been repeatedly and conclusively demonstrated, and like that of the lower vertebrates, is slower than the rhythm of the corresponding sinus. In man it appears to be surprisingly constant, and is usually within a few units of 30 beats per second.

THE EVIDENCE FOR ASSOCIATING HEART-BLOCK WITH DAMAGE TO THE AURICULO-VENTRICULAR BUNDLE.

Before discussing this second proposition it may be well to point out that just as the syndrome, bradycardia and epileptic fits, is no absolute criterion of heart-block, so we cannot as yet regard heart-block as of necessity a criterion of a macroscopic or microscopic lesion of the bundle. Heart-block may be produced in animals by other means, notably by vagal stimulation, and the possibility of a similar causation in man must always be borne in mind.† The necessity for caution will be at once apparent when the following considerations are attended to. The evidence subdivides itself naturally into several classes.

1. Numerous records are to be found in the literature of the subject of cases of Adams-Stokes syndrome, in which *post-mortem* examination demonstrated a lesion in the interventricular septum at or about the situation at which the auriculo-ventricular bundle is now known to exist. In a few of the later cases more detailed examination allows of the statement that the bundle was definitely involved. In this class of case no records of pulsations are given, and while the presumptive evidence of heart-block in many of them is strong, and in some almost certain, the absolute evidence is absent.

2. There is a very large collection of cases presenting Adams-Stokes syndrome, in which no *post-mortem* examination was made, but in which tracings show very definite evidence of the presence of heart-block, and in some few of which there was reason for believing the patients to be subjects of syphilitic lesions, lesions by no means infrequently found occurring in the heart muscle. By itself this group is of little value, but, taken in conjunction with the last, it is not to be neglected.

3. Finally, there is a group of cases, showing the same symptoms, in which tracings were taken sufficient to prove the presence of heart-block, and in which subsequent *post-mortem* examination left no doubt of the affection of the auriculo-ventricular bundle.

This third class of case is by far the most important and conclusive, and will receive special attention. For while the three classes, taken together, present evidence of undeniably greater strength, yet it is the absolute character

* The falling out of a ventricular beat cannot necessarily be ascribed to diminished conductivity.

† It is almost inconceivable that a constant and prolonged heart-block should result from such a cause, and quite inconceivable when it lasts days, weeks, months, or years.

of the third class which renders it distinct, and points it out as the evidence upon which conclusions must ultimately rest. The necessarily complex nature of the evidence as a whole is difficult for those to understand who are unfamiliar with the disease, and the abundant obstacles met with in following up cases clinically and in obtaining the final data.

It will be of advantage, and it will suffice, if we examine the following records of cases of Adams-Stokes syndrome, in which lesions are described in the neighbourhood of the bundle. An examination of these 27 cases shows that in 17 instances histological evidence has been obtained, and damage to the bundle clearly demonstrated. In 10 of these 17 cases tracings were taken, but in only 7 are those which are published satisfactory. Consequently the necessary evidence is forthcoming in these instances only. They are the cases of Hay and Moore, Barr, Gibson, Ashton Norris and Lavenson, Jellinek and Cooper, Heineke Müller and Hösslin, and lastly, Turrell and Gibson. They constitute at present the best evidence available.

There are in all 27 reported cases of Adams-Stokes syndrome, in which lesions are described in the neighbourhood of the bundle. An examination of these 27 cases shows that in 17 instances histological evidence has been obtained, and damage to the bundle clearly demonstrated. In 10 of these 17 cases tracings were taken, but in only 7 are those which are published satisfactory. Consequently the necessary evidence is forthcoming in these instances only. They are the cases of Hay and Moore, Barr, Gibson, Ashton Norris and Lavenson, Jellinek and Cooper, Heineke Müller and Hösslin, and lastly, Turrell and Gibson. They constitute at present the best evidence available.

If we inquire as to the relation between the extent of the lesion and the degree of heart-block, in the hope of

finding accordance between experimental and clinical facts, the evidence, already meagre, dwindles almost to vanishing point. The most that can be said is that the clinical facts allow of the following conclusions:

1. No case of complete heart-block has been reported in which there was not grave reason to suspect complete functional discontinuity of the bundle.

2. With one exception, no case of incomplete heart-block has been reported in which the bundle was demonstrated as completely destroyed at any single point at its course.

The exception requires further examination; it is the case described by Heineke Müller and Hösslin. The relevant facts are as follows: An elderly patient had suffered for many years from symptoms which might be attributed to an incomplete heart-block. On May 23rd, 1907, a tracing was taken with the pulse-rate at 60, and it showed unequivocally that ventricle was responding regularly to auricle. The patient died on July 5th, or six weeks later, and in the interval several tracings were taken, though the dates of them are not given, and the majority of these appear to show complete block. In brief, the tracing of May 23rd is the first and last dated tracing in which conduction is shown. At the autopsy the bundle was found to be completely destroyed in the greater part of its course, being involved in an old-standing lesion, fibrous, calcareous, and haemorrhagic. There is no definite evidence to show that during the last six weeks of life the block was at any time incomplete, neither is there conclusive evidence to show that on May 23rd the destruction

Author.	Lesion.	Histo-logical Confirmation.	Tracings.	Tracings Satisfactory.	Nature of Block and Remarks.
1. Sendler ...	Hard, grating tumour in ventricular septum 1 cm. under pulmonary orifice	Fibroma			? Pulse 22 to 40.
2. Rendu ...	Gumma of septum (high up)				? Pulse 36 to 40.
3. Luce ...	Round-celled sarcoma of septum and wall of ventricle	Sarcoma			? Pulse 28 to 48.
4. Handford ...	Ill-defined thickened mass of ventricle invading <i>a-v</i> ring	Gumma			Probably incomplete.
5. Hay ...	Stretching of <i>a-v</i> bundle with partial obliteration		+		At times complete. Pulse-rate occasionally up to 80.
6. Hay and Moore ...	Almost complete occlusion of right coronary artery; central body atheromatous	+	+		Incomplete.
7. Stengel ...	Endocardial lesion in upper part of left ventricle and septum said to involve bundle		+	Poor	Probably incomplete.
8. Schmoll ...	Sclerosis causing atrophy of <i>a-v</i> fibres	+			Uncertain whether complete or incomplete.
9. Keith and Flack ...	Gumma of septum, <i>a-v</i> fibres involved	+			?
10. Barr ...	Bundle two-thirds obliterated	+	+	+	Complete. Tracings suffered by reduction
11. Keith, Miller, and Chapman ...	Upper half of bundle completely destroyed	+			?
12. G. A. Gibson ...	Sclerosis and separation of muscle fibres	+	+	Fair	Probably incomplete.
13. Aschoff ...	Severe sclerosis of arteries traversing node and principal part of bundle				No details.
14. Aschoff ...	Marked fatty deposit at node				No details.
15. Vaquez and Esmein ...	Gummatous sclerosis affecting bundle	+	+	Very poor	Probably usually incomplete.
16. Fahr ...	Tumour penetrating ventricular septum and completely destroying bundle (gumma)	+			? (Probably Luce's case.)
17. Fahr ...	Sclerosis at division of bundle. Left branch gone; right partially destroyed	+			?
18. A. G. Gibson ...	Partial obliteration of bundle by fibrosis and fat deposit	+			
19. Ashton, Norris, and Lavenson ...	Gumma interrupting bundle completely	+	+	Fair	Complete.
20. Jellinek and Cooper ...	Anaemic necrosis of muscle in neighbourhood of bundle	+	+	+	Probably complete.
21. Heineke, Müller, and Hösslin ...	Gummatous infiltration of bundle	+	+	Fair	Incomplete; possibly complete later.
22. Heineke, Müller, and Hösslin ...	Complete destruction of bundle by sclerosis, calcification, and haemorrhage	+	+	Fair	Incomplete 6 weeks before death; possibly complete later.
23. Butler ...	Bundle showed fatty infiltration and degeneration, and atrophy to one-fifth size	+			? Pulse 6 to 42 per minute.
24. James* ...	Deep ulcer invading bundle region in left side of ventricular septum		+	+	Usually complete.
25. Jagic ...	Numerous gummata in ventricular septum				? Pulse 28 to 60.
26. Turrell and Gibson†	The bundle represented by a few isolated strands	+	+	Fair	? Incomplete.
27. Gerhardt†	Acute inflammation of bundle	+	+	Poor	Incomplete.

* A further case reported by Dock is cited by James.

† BRITISH MEDICAL JOURNAL, 1908, vol. ii, p. 1486.

‡ *Deutsch. Archiv f. klin. Med.*, 1908, Bd. 93, S. 485.

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.