Further observations on heart-block / by G. A. Gibson and W. T. Ritchie.

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By clinical observation and experimental investigation, it has now been conclusively proved that disturbances of both the rate and rhythm of the heart may be the manifestation of impairment of its conductivity. In the heart of the tortoise and of the frog, we know that the stimuli to contraction, which normally originate in the sinus venosus, and pass thence to the auricular and the ventricular fibres, may be blocked at various points, but, in particular, at the junction of the sinus and auricle, and again at the auriculo-ventricular boundary. Defective conduction of a stimulus may be held as proved, whenever the period, elapsing between the contraction of the sinus and the auricle, or of the auricle and the ventricle, is unduly lengthened.

The study of the conductivity of the mammalian heart has, however, been almost entirely restricted to that of the fibres of the auriculo-ventricular bundle, which constitute the functional bond of union between the auricles and the ventricles. That there must be a muscular connection between the auricles and the ventricles has been stoutly maintained ever since Gaskell[1] recorded his observations on the heart of the tortoise, but although our knowledge of the site, distribution, and structure of the connecting bundle—the auriculo-ventricular bundle—was advanced by the observations of Stanley Kent,[2] His junior,[3] Retzer,[4] and Braeunig,[5] it is only by the recent observations of Tawara,[6] which have been

[1] *Journal of Physiology*, 1883, Vol. IV., p. 43.
[2] *Journal of Physiology*, 1893, Vol. XIV., p. 233.
[3] *Arbeiten aus der med. Klinik zu Leipzig*, 1893, S. 14–49; *Wiener med. Blätter*, 1894, XVII., S. 653; *Centralbl. f. Physiol.*, 1896, Bd. IX., S. 469.
[4] *Archiv f. Anatomie u. Physiologie*, Anatomische Abteilung, 1904, S. 1.
[5] *Archiv f. Anatomie u. Physiologie*, Physiologische Abteilung, 1904, Supplement-Band, S. 1.
[6] "*Das Reizleitungssystem des Säugetierherzens*," 1906.

verified by Keith and Flack,[1] that an accurate conception of its arrangement and distribution has been formed. Tawara has shown that, within the auricular walls, there exists a network of special muscle fibres with a nodal point in the inter-auricular septum just above the attachment of the mesial segment of the tricuspid valve. From the nodal point, a bundle of muscle fibres passes downwards through the fibrous auriculo-ventricular ring, to divide in the inter-ventricular septum into two branches, one for each ventricle. The fibres of each branch subsequently pass as Purkinje's fibres in the subendothelial tissue, and ultimately terminate in the ordinary cardiac muscle fibres in the walls of the ventricles, and in their papillary muscles. Those, who uphold the myogenetic theory of the rhythmic action of the heart, believe that the fibres of the auriculo-ventricular bundle are mainly concerned with the conduction of stimuli to contraction, but it should not be forgotten that the truth of the myogenetic theory has recently been called in question by Hering,[2] and also by Carlson,[3] who has made a most careful study of the action of the heart in *Limulus*. It is admitted, on all hands, that the auriculo-ventricular bundle contains not only nerve fibres but ganglia, as Tawara[4] has clearly shown. That the conduction is by muscle is, as yet, therefore unproved.

After section of the main stem of the auriculo-ventricular bundle, as performed experimentally by His junior,[5] Humblet,[6] and Hering,[7] no stimuli to contraction can be transmitted onwards from the auricles to the ventricles, nor can retrograde extra-systoles pass from the ventricles to the auricles, and while the rhythm and rate of the auricular contractions continue unchanged, the ventricles beat less frequently than the auricles, and with independent rhythm. Section of the bundle, therefore, results in complete dissociation of the auriculo-ventricular rhythm—complete heart-block.

[1] *Lancet*, 1906, I., p. 623; *Ibid.*, 1906, II., p. 359.

[2] *Zeitschrift für experimentelle Pathologie und Therapie*, 1906, Band III., S. 511.

[3] *American Journal of Physiology*, 1905-06, Vol. XV., p. 113.

[4] *Zentralblatt für Physiologie*, 1906, Bd. XIX., S. 300.

[5] *Wiener med. Blätter*, 1894, Bd. XVII., S. 653; *Centralbl. f. Physiologie*, 1896, Bd. IX., S. 469.

[6] *Arch. intern. de Physiologie*, 1906, tome III., p. 330.

[7] *Archiv f. d. gesammte Physiologie*, 1905, Bd. CVIII., S. 267; *Ibid.*, 1906, Bd. CXI., S. 298.

The results of experimental clamping of the auriculo ventricular bundle have been studied by Erlanger,[1] by that observer and Hirschfelder,[2] and by von Tabora.[3] By gradually increasing the pressure of the clamp, the conductivity of the fibres of the bundle can be gradually lessened, so that only every alternate, and, subsequently, only every third, fourth, or fifth stimulus is transmitted onwards from the auricle to the ventricles. Thus the ratio between the auricular contractions and the ventricular may be 2 : 1, 3 : 1, 4 : 1, etc., and the condition, which then prevails, is one of partial heart-block. When the pressure of the clamp is still further increased, no stimuli can pass from the auricles to the ventricles, the block which was previously partial becomes complete, and there is dissociation of the auriculo-ventricular rhythm.

It is now well established that both partial and complete block at the auriculo-ventricular bundle may be met with in the human heart. In former papers[4, 5] we have recorded such cases, which we studied by graphic methods, the fluorescent screen, and the capillary electrometer, and we have described the effect of atropine administration upon the rate of the auricles and of the ventricles in complete auriculo-ventricular heart-block. Within the last two years, the subject of auriculo-ventricular heart-block has engaged the attention of many other observers, and tracings demonstrating that condition have been recorded by Mackenzie,[6] Rihl,[7] Belski,[8] Hay,[9] Bönninger,[10] Erlanger,[11] Brouardel and Villaret,[12] Leuchtweis,[13] Finkelnburg,[14]

[1] *Journal of Experimental Medicine*, 1906, Vol. VIII., p. 8.
[2] *American Journal of Physiology*, 1906, Vol. XIV., p. 153.
[3] *Zeitschrift f. experimentelle Pathologie u. Therapie*, 1906, Bd. III., S. 499.
[4] "*The Nervous Affections of the Heart*," 1904, p. 61; *Edinburgh Medical Journal*, 1905, Vol. XVIII., New Series, p. 9; *British Medical Journal*, 1906, II., p. 22; *Ibid.*, 1906, II., p. 1113.
[5] *Proceedings of the Royal Society of Edinburgh*, 1905, Vol. XXV., p. 1085.
[6] *British Medical Journal*, 1905, I., p. 521; *Ibid.*, 1906, II., p. 1109.
[7] *Zeitschrift f. experimentelle Pathologie u. Therapie*, 1905, Bd. II., S. 83.
[8] *Zeitschrift f. klinische Medizin*, 1905, Bd. LVII., S. 529.
[9] *British Medical Journal*, 1905, II., p. 1034; *Lancet*, 1906, I., p. 139; *Medical Chronicle*, 1906, Vol. 44, p. 371.
[10] *Zeitschrift f. experimentelle Pathologie u. Therapie*, 1905, Bd. I., S. 663.
[11] *Journal of Experimental Medicine*, 1905, Vol. VII., p. 676.
[12] *Archives de médecine expérimentale et d'anatomie pathologique*, 1906, tome XVIII., p. 230.
[13] *Deutsches Archiv f. klinische Medicin*, 1906, Bd., LXXXVI., S. 456.
[14] *Ibid.*, 1905, Bd. LXXXII., S. 586; 1906, Bd. LXXXVI., S. 462.

Magee Finny,[1] Roos,[2] Hay and Moore,[3] Bard,[4] Lichtheim,[5] Wenckebach,[6] Goteling Vinnis,[7] Schmoll,[8] Joachim,[9] and Ashton, Norris, and Lavenson.[10] The case recorded, in 1900, by Votruba [11] was not referred to in our previous papers, but may now be accepted as a heart-block, whereas the exact nature of the cases recorded by Perugia [12] and Livierato [13] must remain somewhat uncertain in the absence of graphic records of the jugular pulsations and ventricular contractions. The only other method, whereby heart-block has been graphically demonstrated, is that recently described by Einthoven,[14] who shows that, by the method which he has devised for graphically recording the electro-motive changes of the heart, a dissociation of the auriculo-ventricular rhythm can be as accurately studied by the electro-cardiogram as by tracings of the jugulo-carotid pulsations.

Although a lesion of the auriculo-ventricular bundle has been experimentally shown to produce heart-block, and, although morbid changes have been described in that bundle of the human heart by Stengel,[15] one of us,[16] Schmoll,[17] Jellick, Cooper, and Ophuls,[18] and Keith and Miller,[19] it must be borne in mind that a partial, or possibly even a complete block at

[1] *British Medical Journal*, 1906, I., p. 967 ; *Transactions of the Royal Academy of Medicine in Ireland*, 1906, Vol. XXIV., p. 99.

[2] *Zeitschrift f. klinische Medizin*, 1906, Bd. LIX., S. 197.

[3] *Lancet*, 1906, II., p. 1271.

[4] *Journal de Physiologie et de Pathologie général*, 1906, tome VIII., p. 466, and p. 473.

[5] *Deutsches Archiv f. klinische Medicin*, 1905, Bd. LXXXV., S. 360.

[6] *Archiv f. Anatomie u. Physiologie*, Physiologische Abteilung, 1906, S. 297.

[7] De aanhoudende verdubbeling van den hartslag (hart-bigeminie), Leiden. *Proefschrift*, 1905.

[8] *Deutsches Archiv f. klinische Medicin*, 1906, Bd. LXXXVII., S. 554.

[9] *Ibid.*, 1906, Bd. LXXXV., S. 373 ; 1907, Bd., LXXXVIII., S. 574.

[10] *American Journal of the Medical Sciences*, 1907, Vol. CXXXIII., p. 28.

[11] *Sbor Praci*, 1900, p. 123.

[12] *Gazetta degli Ospedale e delle Cliniche*, 1905, XXVI., p. 859.

[13] *La Clinica Medica Italiana*, 1906, XLV., p. 71.

[14] *Archives Néerlandaises des Sciences exactes et naturelles*, Série II., tome XI., 1906, p. 239.

[15] *American Journal of the Medical Sciences*, 1905, Vol. CXXX., New Series, p. 1083.

[16] *British Medical Journal*, 1906, II., p. 1118.

[17] *Deutsches Archiv f. klinische Medicin*, 1906, Bd. LXXXVII., S. 554.

[18] *Journal of the American Medical Association*, 1906, Vol. XLVI., p. 955.

[19] *Lancet*, 1906, II., p. 1429.

the auriculo-ventricular bundle may be induced by vagus stimulation, as was observed by Knoll[1] after the administration of helleborein, and by Cushny[2] and Mackenzie[3] after the employment of digitalin. By means of calycanthine, Cushny[4] has also been able to cause heart-block in a pithed frog, but, in this experiment, the block was almost certainly due to a direct action upon the heart muscle, the inhibitory mechanism not being involved. Von Tabora[5] has recently investigated, by experimental methods, the auriculo-ventricular block induced by digitalin, and has shown that it is due partly to the effect of the drug on the vagus, and also to a direct effect upon the auriculo-ventricular bundle. When the bundle is intact, digitalis may of itself undoubtedly cause a partial block at that point, but probably never a complete auriculo-ventricular dissociation, whereas, when the conductivity of the fibres of the bundle is already impaired by structural change, the administration of digitalis may convert a pre-existing partial block into one that is complete. That stimulation of the vagus has an effect upon the conductivity of the fibres of the auriculo-ventricular bundle has also been proved by Mackenzie[6] in a patient in whom, although there was no pre-existing block, the conductivity of the heart was impaired. In that patient, the act of deglutition induced a transient block at the auriculo-ventricular bundle.

Whereas the conductivity of the mammalian heart, as we have already stated, has been studied almost exclusively at the auriculo-ventricular bundle, we are now able to record a case in which there was a block at that bundle, together with a marked depression of conductivity at the junction of the superior vena cava with the right auricle.

A cabman, *æt.* 66, came under our observation in July 1906, complaining of weakness and shortness of breath. For thirty years, he has indulged in occasional drinking-bouts, but has taken almost no alcohol during the last eight years. He

[1] *Sitzungsberichte d. k. Akad. d. Wiss. in Wien*, 1894, Bd. CIII., Abt. iii. (Math. Naturioiess. Classe), S. 298.

[2] *Journal of Experimental Medicine*, 1897, Vol. II., p. 232.

[3] *British Medical Journal*, 1905, I., p. 587.

[4] *Archives internationales de Pharmacodynamie et de Therapie* 1905, tome XV., p. 487.

[5] *Zeitschrift f. experimentelle Pathologie u. Therapie*, 1906, Bd. III., S. 499.

[6] *British Medical Journal*, 1906, II., p. 1109.

acquired syphilis nine years ago ; treatment was not continued for more than three weeks. Six years ago, he suffered from a severe attack of acute rheumatism, and, for the last six years, he has suffered from chronic bronchitis, being often confined to bed for 6–8 weeks during the winter time. He gives a history of occasional syncopal attacks. Their onset is sudden, although they are ushered in by a choking sensation in the throat, and "and a terrible buzzing noise in the ears." During an attack, he has, on several occasions, dropped on to the floor or on the street, the period of unconsciousness lasting from a few seconds to a couple of minutes. It does not appear that there were ever any epileptiform convulsions during an attack.

The patient is not cyanosed or dropsical. The cardiac impulse, which is visible only in the 5th left interspace, 1 inch external to the left mammary line, and 5 inches from the mid-sternal line, is somewhat feeble, but rhythmical ; the rate when the patient was first seen was 35 per minute. The borders of the heart lie 2 inches to the right and 5 inches to the left of the mid-sternal line. On auscultation at the mitral area, there is a soft blowing systolic murmur, while a soft diastolic murmur follows the second sound, but no pre-systolic murmur is audible. At the other areas, both the first and second sounds are closed ; the aortic second sound is accentuated. On auscultating over the inner end of the 2nd and 3rd left interspaces, one or more faint additional sounds are distinctly heard during the long interval elapsing between the second sound and the subsequent systolic sound. Those additional sounds are to be regarded as caused by auricular systoles. When the patient is examined with the fluorescent screen, the auricular pulsations are seen to be at least twice as numerous as those of the ventricles.

The radial arteries are thickened, but are not obviously tortuous. The rate of the pulse, when the patient first came under observation, was 35 per minute, and it was quite regular. The arterial blood pressure is high. Faint yet distinct pulsations are visible in the jugular veins at the root of the neck. Both these features will be considered in a later part of this paper.

The thorax is enlarged in its antero-posterior diameter,

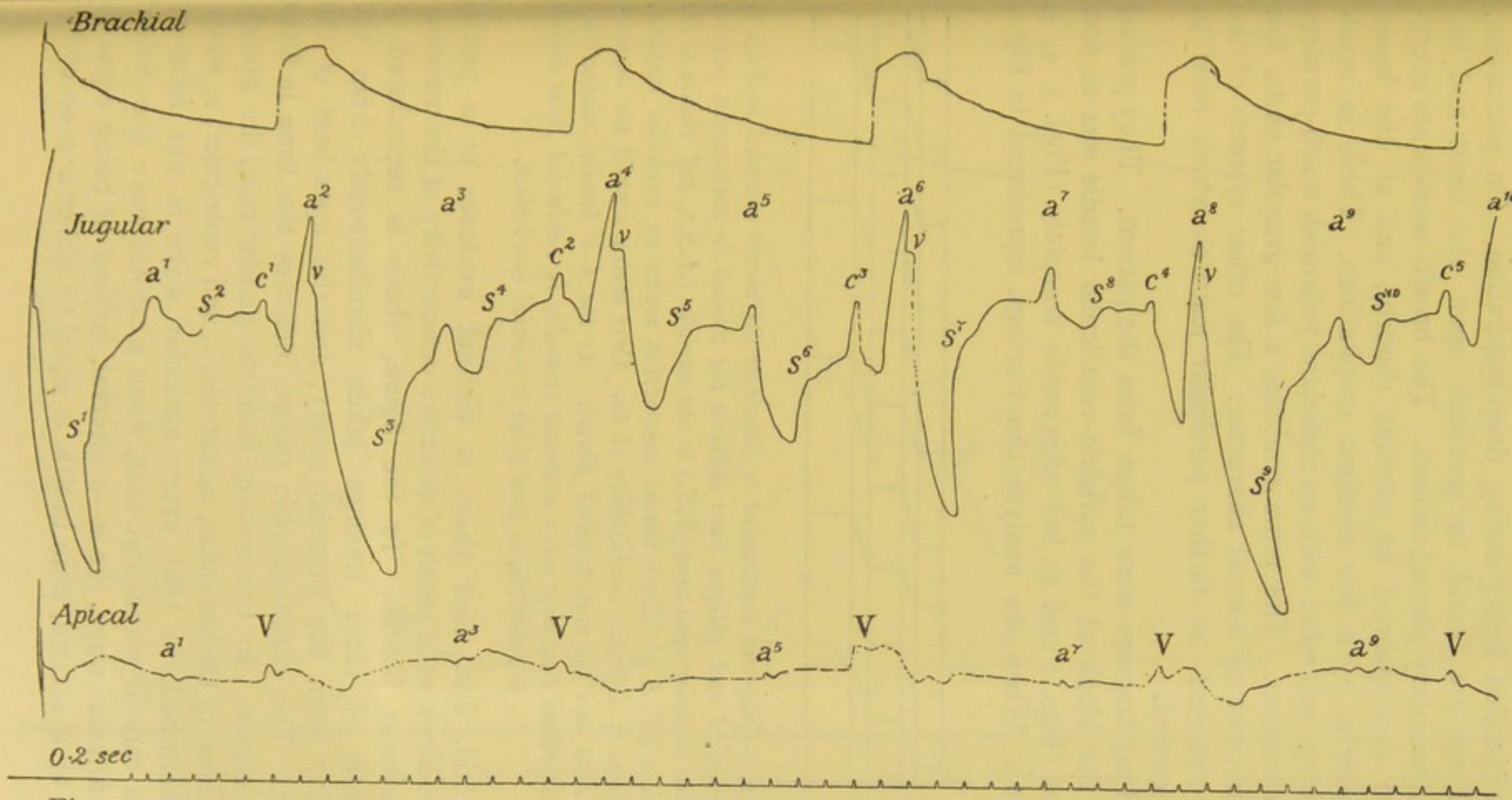


Fig. 1.—*Partial auriculo-ventricular heart-block, and defective conductivity at the junction of the superior vena cava and right auricle.*

and expanded feebly during deep inspiration. On percussion, the lungs are found to encroach upon the heart and the complementary pleural sinuses. The breath sounds are everywhere accompanied by sibilant rhonchi, and at the bases, posteriorly, by a few medium crepitations. There is much coughing, attended with an abundant yellowish muco-purulent expectoration. The urine contains a few granular casts, but is otherwise of normal character. The other systems of the body present no further particular features which need to be recorded.

Many tracings were taken from the patient. They proved that the block at the auriculo-ventricular bundle was at first partial (Figs. 1 and 2), but afterwards complete (Figs. 3, 4, 5, and 6). When we analyse the tracings, reproduced in Fig 1,

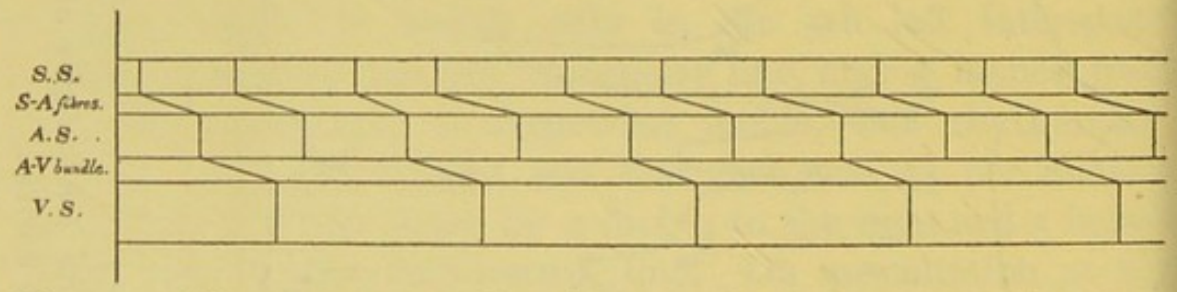


Fig. 2.—*Diagram constructed to illustrate the events recorded in Fig.* 1. *The vertical and oblique lines indicate the stimuli to contraction passing from the superior vena cava* (*S.S.*) *to the auricles* (*A.S.*), *and thence to the ventricles* (*V. S.*). *Each venous contraction evokes an auricular response, but there is defective conductivity at the fibres which connect the superior vena cava and the auricle* (*S-A fibres*). *At the A-V bundle, conductivity is so depressed that only every alternate stimulus is conducted from auricle to ventricle, so that there is partial heart-block.*

we find, first, that there is distinct evidence of a partial heart-block as a result of defective conductivity at the auriculo-ventricular bundle. In the figure, there is reproduced a portion of three tracings taken simultaneously from the jugular vein, the brachial artery, and the apex-beat of the heart. The chronographic record indicates the time in fifths of seconds. In the tracing from the jugular vein, the normal sequence of the auricular, *a*, carotid, *c*, and ventricular, *v*, waves is disturbed, in that every auricular wave is not followed by a carotid wave, and that, when a carotid wave does follow an auricular wave, the *a—c* interval, instead of being equal to 0·2 of a second, as in health, is equal to either 0·7 or 0·8 of

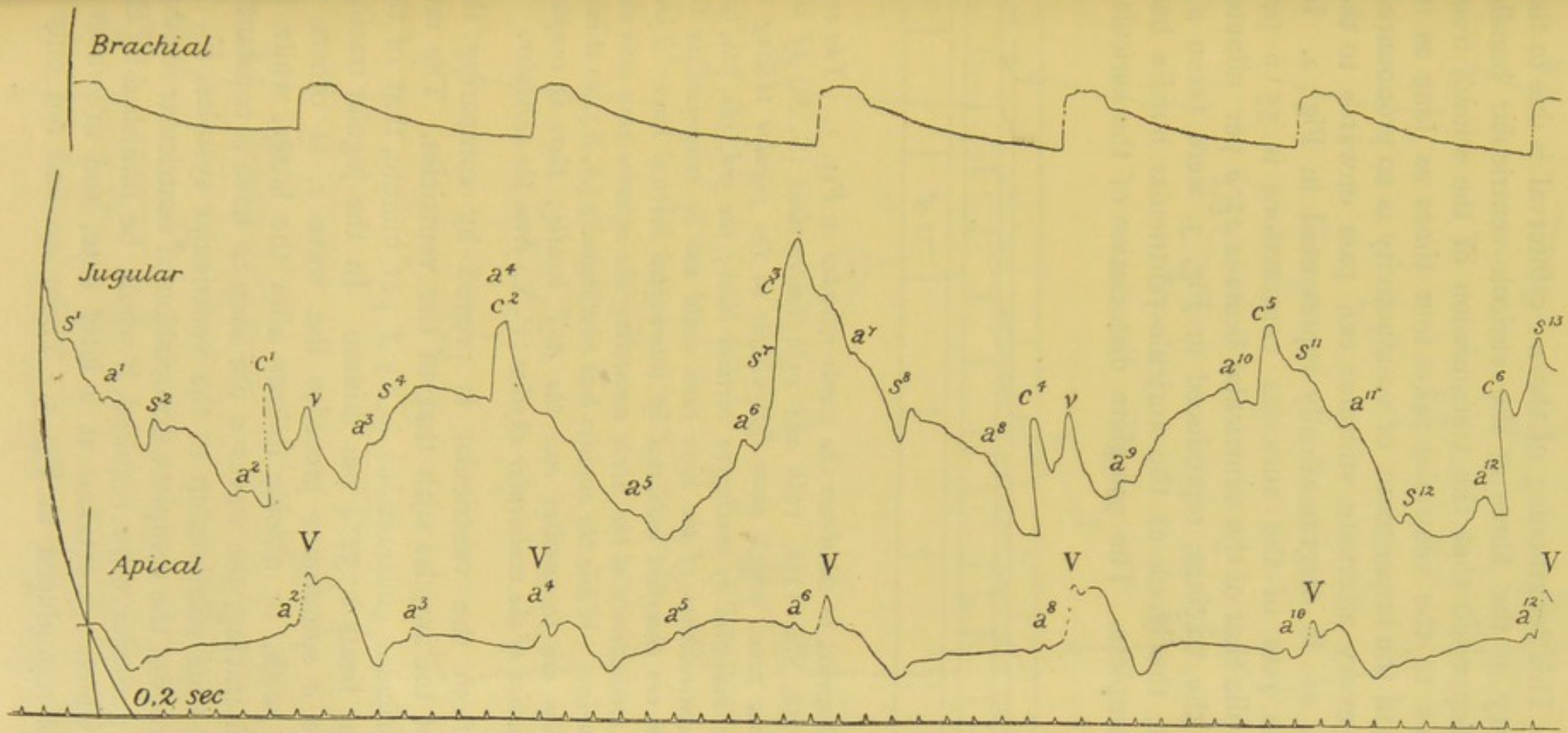


Fig. 3.—*Complete auriculo-ventricular heart-block, and defective conductivity at the junction of the superior vena cava and right auricle.*

a second. This lengthening of the *a—c* interval is due to the conductivity of the fibres of the auriculo-ventricular bundle being so depressed that the transmission of the stimuli from the auricles to the ventricles takes four times as long as in health ; and the impairment of conductivity is so pronounced that only every alternate stimulus can pass onwards to the ventricles, as is diagramatically represented in Fig. 2. In Fig. 1 the rate of the auricular contractions is 55·6 per minute, while that of the ventricular beats is 27·8 per minute.

When the tracings, reproduced in Fig. 3, were taken six days later, the block at the auriculo-ventricular bundle had become complete. The complete dissociation of the auricular

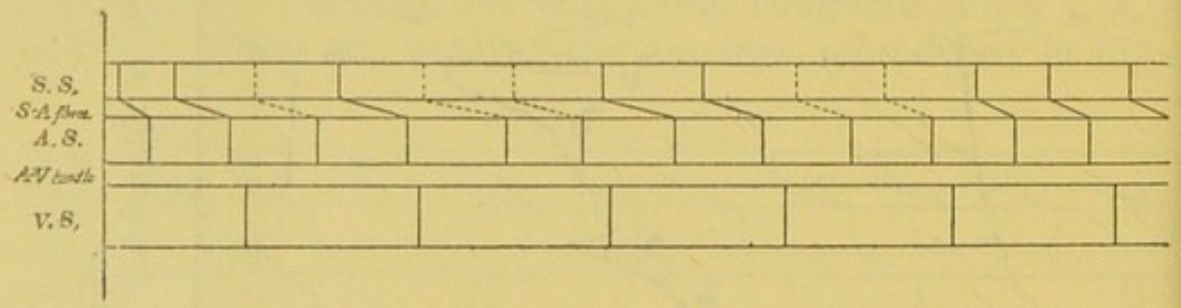


Fig. 4.—*Diagram to illustrate the events recorded in Fig.* 3. *The* 1*st*, 2*nd*, 4*th*, 7*th*, 8*th*, 11*th*, 12*th and* 13*th contractions* (*S. S.*) *of the superior vena cava, which were recognisable in the jugular tracing of Fig.* 3, *are indicated by continuous vertical lines ; the* 3*rd*, 5*th*, 6*th*, 9*th and* 10*th contractions of the vena cava could not be recognised in the tracing, and are therefore indicated by interrupted vertical lines. There is defective conductivity at the fibres connecting the superior vena cava and the right auricle. While the auricles beat rhythmically* (*A. S.*), *no stimuli to contraction are transmitted over the A-V bundle ; there is complete dissociation of the ventricular rhythm* (*V. S.*) *from the auricular.*

rhythm from the ventricular is proved by comparing the rhythm of the auricles with that of the ventricles. The rate of the auricular contractions is 65·2 per minute, that of the ventricular beats is 32·4 per minute. In the jugular tracing, the wave *a* sometimes precedes the wave *c*, at others, it coincides with, or quickly follows after the latter, while, in the apical tracing, the waves *a* are seen to have a constantly changing time relationship to the ventricular systoles, *V*. If further proof of the complete dissociation of ventricular rhythm from the auricular were required, it would be furnished by the tracings which were taken at a later date, and of which a portion is reproduced in Fig. 5. There we find not only a

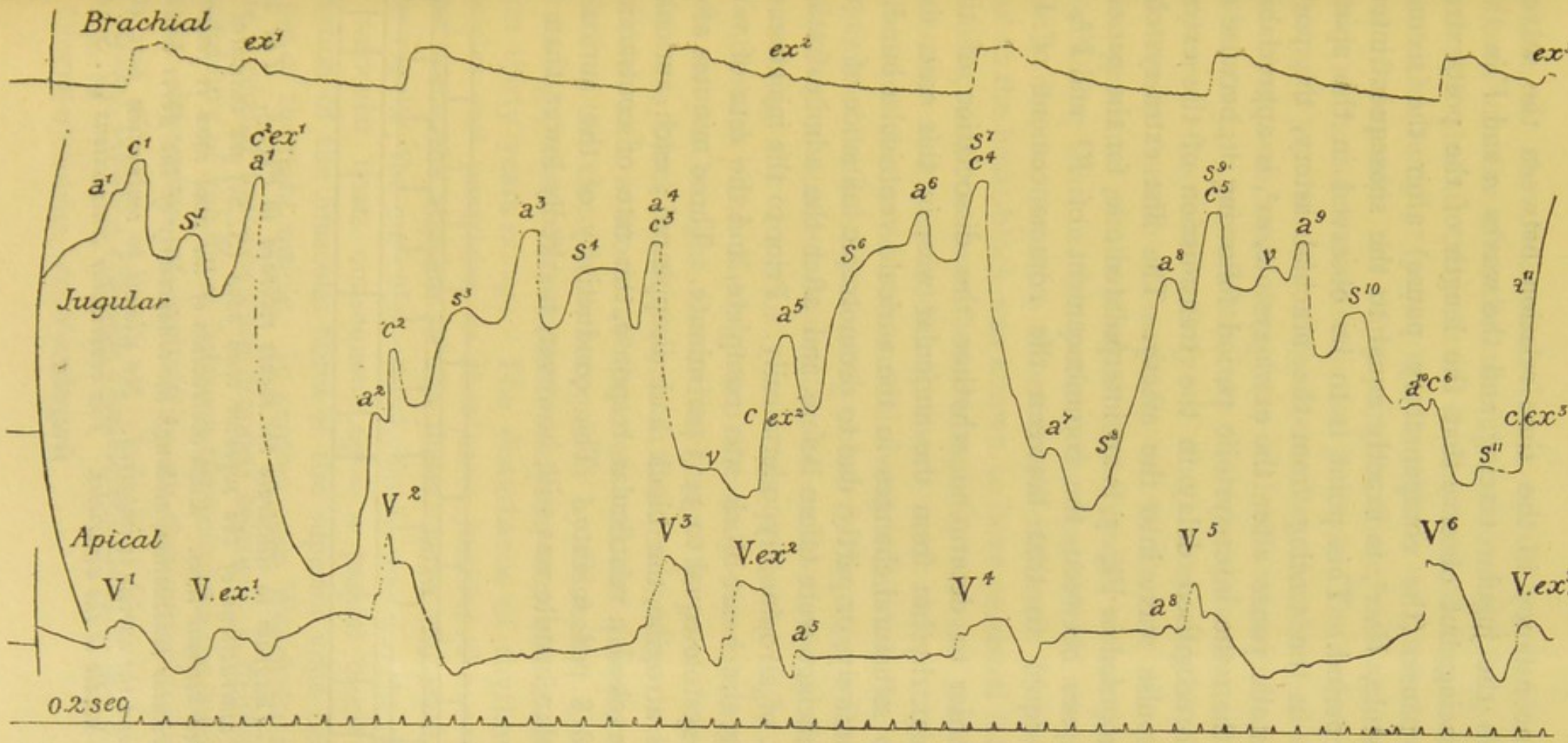


Fig. 5.—*Complete auriculo-ventricular heart-block, and also defective conductivity at the junction of the superior vena cava and right auricle, with ventricular extra-systoles.*

similar inconstancy in the time relation between the waves *a* and *c* in the jugular tracing, and the waves *a* and *V* in the apical tracing, but observe that the length of the post-extra-systolic pause (the compensatory pause) after the second extra-systole, $V\,ex^2$, is exactly equal to the subsequent inter-systolic period. This point is to be observed in the apical tracing; in the tracing from the brachial artery, the post-extra-systolic pause after the extra-systole, ex^2, is appreciably shorter than the inter-systolic period following it, because of the well-recognised delay in the transmission of the extra-systolic pulse wave into the artery. The first extra-systole, $V\,ex^1$, recorded in Fig. 5, is an interpolated one, for the period, that elapses between the commencement of V^1 and V^2, is exactly equal to that between the commencement of V^2 and V^3.

In order to determine whether the dissociation of the ventricular rhythm from the auricular was, in this case, due solely to structural changes in the auriculo-ventricular bundle, or was entirely, or partly, due to dromotropic influence of vagus origin, tracings were taken before and after the administration of $\frac{1}{60}$ gr. of atropine, hypodermically. Prior to the injection of this drug, the heart-block was complete, and the rate of ventricular systole equal to 34·3 per minute. Three minutes after $\frac{1}{60}$ gr. of atropine, the block had disappeared; each auricular systole evoked a ventricular response, the rate of contraction being 58·8 per minute. The conductivity of the auriculo-ventricular bundle was still, however, markedly lower than in

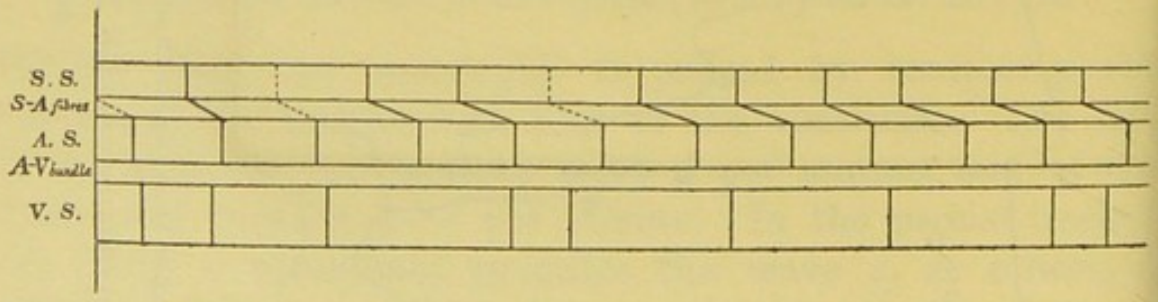


Fig. 6.—*Diagram to illustrate the events recorded in Fig. 5. The 2nd and 5th contractions of the superior vena cava (S. S.) are indicated by interrupted vertical lines. Each contraction of the vena cava is followed by an auricular contraction, although the conductivity of the fibres between the vein and the auricle is impaired. No stimuli to contraction pass over the A-V bundle to the ventricles. The ventricular contractions (V. S.) are irregular.*

health, for the *a—c* interval was equal to 0·6 of a second. Fifteen minutes after the administration of the drug, the block was still absent, the rate of contraction of the auricles and ventricles being 37·5 per minute; the *a—c* interval was only 0·4 of a second. In thirty minutes, there was again a partial block at the auriculo-ventricular junction; the ventricular rate was 34·7 per minute. One hour after the dose of atropine, the block was again complete, and the ventricular rate had fallen to 32·7 per minute. As the administration of atropine improved the conductivity of the fibres of the auriculo-ventricular bundle so markedly as to lead to the transient disappearance of the block at that bundle, we are justified in concluding that, in this patient, the heart-block was at least partly due to negative dromotropic influence of the vagus. But as the heart-block was known to have persisted for several months, and to have been complete, we are confident in asserting that, in addition, there must have been some structural change in the auriculo-ventricular bundle.

One of the most interesting points in the case, is the graphic record of a marked depression of conductivity in the fibres connecting the superior vena cava with the right auricle. Wenckebach[1] and Hering[2] are the only two observers who have as yet brought forward evidence to prove that there may be a block at that point in the mammalian heart comparable with a block at the sino-auricular junction of the heart of the tortoise, as recognised by Gaskell.[3] One series of tracings, which Wenckebach analyses, was obtained from a man thirty years of age. The disturbance of rhythm of the auricles and ventricles, in that case, justifies the conclusion that, while the rhythm of the fibres above the right auricle was regular, the auricular allorrhythmia was due to dropping of auricular beats consequent upon a partial block at the junction of the muscular fibres of the superior vena cava with those of the right auricle. In another case, Wenckebach shows that auricular systoles were dropped because of depression of excitability of the fibres between the vein and the auricle. A similar depression of excitability at the auriculo-ventricular

[1] *Archiv f. Anatomie u. Physiologie*, Physiologische Abteilung, 1906, S. 318.
[2] *Loc. cit.*
[3] *Journal of Physiology*, 1883, Vol. IV., p. 43.

fibres, leading to dropping of ventricular beats, has already been recorded by one of us,[1] as well as by Hay,[2] and by Wenckebach.[3]

In 1900, Hering[4] observed that, in rabbits, when the rate of the exposed heart decreased on cooling, the auricular pulsations were seen to distinctly follow those of the great veins, that, subsequently, an auricular contraction would occur only after several pulsations of the veins, and that, eventually, the latter pulsated alone without any accompanying auricular beats. The same observer has recently[5] obtained tracings from a rabbit and a dog, both presenting a similar disturbance of cardiac rhythm, which can only be explained by the assumption of a transient defect of conductivity of fibres between the site, at which the stimuli to contraction originate, and the auricles. These are the sole contributions, so far as we are aware, which have hitherto been made to either prove or disprove the possibility that affections of conductivity may be met with at the junction between the venæ cavæ and the right auricle of the mammalian heart.

In the jugular tracings of Figs. 1, 3, and 5, we find, in addition to the waves *a*, *c*, and *v*, yet another wave, *s*, more or less well marked throughout the tracings. In Fig. 1 the wave *s* precedes the auricular wave, *a*, by 0·6—0·8 of a second. The wave *s* is not an accidental one, for it is recognisable in all the tracings taken from the patient. It is not due to a ventricular extra-systole, for neither in the cardiogram nor in the sphygmogram is there evidence of such an event. Nor is the wave *s* due to an auricular systole, for whereas the auricular waves of the jugular tracing (*see* Fig. 1), with the exception of those which occur at a time when the ventricles are in systole, are also represented by distinct waves, *a*, in the cardiogram, the waves *s* are not represented in the latter. The wave *s* is sometimes followed immediately by an auricular wave, for example, s^{12} and a^{12} in Fig. 3, also s^{3} and a^{3} in Fig. 5. At other times, a carotid wave is interposed between them, as between s^{4} and a,[4] and between s^{8} and a^{8} in Fig. 1; and when there is

[1] *British Medical Journal*, 1906, II., p. 1113.
[2] *Lancet*, 1906, I., p. 139.
[3] *Archiv f. Anatomie u. Physiologie*, Physiologische Abteilung, 1906, S. 328.
[4] *Archiv f. die gesammte Physiologie*, 1900, Bd. LXXXII., S. 21.
[5] *Zeitschrift f. experimentelle Pathologie u. Therapie*, 1906, Bd. III., S. 511.

complete dissociation of the rhythm of the auricles and of the ventricles, the wave *s* may coincide with the wave *v*, or with the carotid wave. The latter event is observed in the broad topped waves s^7 c^4, and s^9 c^5 in Fig. 5.

The wave *s* is, we believe, the expression of the rise in pressure in the jugular vein induced by the rhythmical contraction of the muscular fibres of the superior vena cava. That the venæ cavæ pulsate was known by Haller,[1] and Johannes Müller,[2] but the fact had been forgotten until Lauder Brunton, and Fayrer[3] again drew attention to the rhythmical contractile power of the pulmonary veins and of the vena cava.

The possibility that the contraction of the muscular fibres of the superior vena cava may give rise to an appreciable wave in the jugular vein is admitted by Erlanger,[4] who says that the auricular systole may be preceded by a distinct positive wave in the veins of the neck. In normal circumstances, this wave cannot be observed, but Erlanger believes it is possible that, when the pressure in the great veins is high, the effect of their contraction may become evident. Wenckebach[5] also admits that such a wave may be recognisable in tracings. He says, "I cannot but believe that the difficulty so frequently experienced in determining precisely the commencement of the *a* wave is due to a slight elevation preceding it." In one of his cases, Wenckebach draws attention to the fact that the auricular waves were broader than normal, that some of those broad waves really consisted of two component waves, the first a small wave, the second a larger one, and that the interval between the latter and the succeeding carotid wave was exactly equal to the *a*—*c* interval in other parts of the tracing. He, therefore, concludes that the second and larger elevation of the compound wave represented the true auricular wave, and that the smaller elevation, which preceded it, could only be ascribed to the contraction of the muscular fibres of the superior vena cava.

In Figs. 1, 3, and 5, as well as in the diagrammatic schemes constructed therefrom (Figs. 2, 4, and 6), the rhythmical

[1] "*Elementa Physiologiae Corporis humani*," 1757, tomus 1, pp. 399 and 410.
[2] "*Elements of Physiology*," trans. by W. Baly, 1838, Vol. I., p. 172.
[3] *Proc. Roy. Soc.*, London, June, 1876, Vol. 25, p. 172.
[4] *Jour. of Exper. Med.*, Vol. VII., 1905, pp. 690, 691.
[5] *Archiv f. Anat. u. Phys.*, Phys. Abth., 1906, S. 331.

occurrence of the wave *s* is readily observed. A point, to which we would particularly draw attention, as it is one which has not been hitherto demonstrated in the human heart, is the excessive length of the interval between the contraction of the fibres of the superior vena cava and the succeeding auricular contraction. This interval, the *s—a* interval, is of almost constantly uniform duration throughout the tracings, being equal to 0·6 of a second, though sometimes slightly shorter or longer. The excessive length of the *s—a* interval can only be explained by a depression of conductivity of the fibres passing from the superior vena cava to the right auricle. We are confirmed in this belief by our analysis of tracings taken both before and after the administration of $\frac{1}{60}$ gr. of atropine. Whereas the *s—a* interval was equal to 0·6 of a second before that drug was given, 11 minutes after its administration the interval was only 0·4 of a second, half-an-hour after the atropine, the *s—a* interval was 0·52 of a second, and, after one hour, that interval was again equal to 0·6 of a second. Comparing these results with those of the conductivity of the auriculo-ventricular bundle of the same case, we find that the improvement in the conductivity of the fibres between the vena cava and the auricle on the one hand, and of those of the auriculo-ventricular bundle on the other, was induced by atropine simultaneously, and that as the transient benefit, occasioned by that drug, passed off, the conductivity of both sets of fibres fell gradually and simultaneously to the former level.

The condition of the blood-pressure in cases of heart-block has not as yet received much attention. About two years ago, certain facts bearing upon this aspect of the condition were mentioned by one of us,[1] and the statement was amplified last year.[2] During the careful observations which were made on the patient, whose case is fully detailed in the present paper, the blood-pressure was repeatedly observed. The highest systolic pressure recorded at any time was 190 mm. Hg., and, with this maximum systolic pressure, the mean diastolic pressure was 80 mm. Hg. At other times, with a systolic pressure of 175, the diastolic pressure was 75 mm. Hg. Here, therefore, as was stated in the contributions already

[1] *Edin. Med. Journ.*, 1905, Vol. XVIII., New Series, p. 9.
[2] *Brit. Med. Journ.*, 1906, Vol. II., p. 1113.

Fig. 7.—*Tracing taken from the patient with Erlanger's instrument, and slow release of air.*

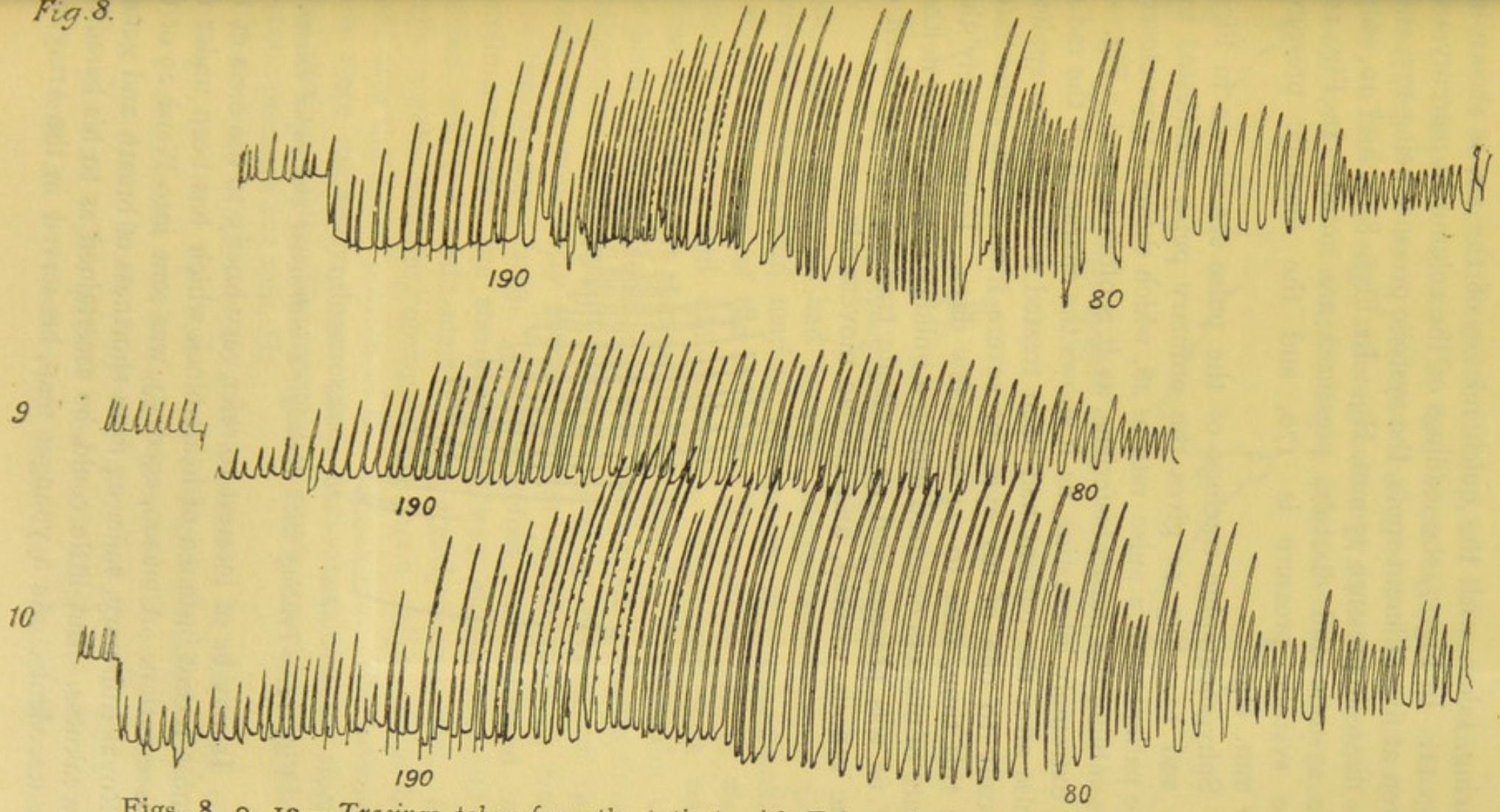
referred to, the important point lies in the enormous variation between systolic and diastolic pressure. In one patient, who has been long under the observation of one of us, the systolic pressure is sometimes as high as 270, and the diastolic pressure only 80 mm. Hg. It is obvious that the long pauses, occurring between the contractions of the ventricles, must allow a great fall of pressure to take place in the intervals between the contractions.

As a rule, the estimations of the blood-pressure were made by means of the Riva Rocci, or the Erlanger sphygmomanometer. For ordinary daily observations, the former instrument is amply sufficient, as it is at once easy of application and accurate in results ; but when a permanent record is required, the instrument devised by Erlanger is essential. A description of the instrument has been published by Erlanger,[1] and a capital account, both of the instrument and of its method of employment, is to be found in the work of Janeway.[2] The mechanism resembles that of most of the mercurial sphygmomanometers with circular compression of the arm. The air contained in the pad and the tube leading to the column of mercury is, by a most ingenious valve, brought into communication with a tambour, whose lever inscribes its movements upon a revolving cylinder. The instrument is employed, in the same manner as the Riva Rocci and other syhygmomanometers, by increasing the pressure in the circular pad until the radial pulse is entirely obliterated, and then allowing the air to escape. It is found that, just at the instant of time when the radial pulse reappears at the wrist, the lever begins to manifest its largest excursions. The maximum systolic pressure is, therefore, easily determined. The diastolic pressure is not so definite, but it is believed that, just at the moment when the largest amplitude of the excursions begins to diminish, the diastolic pressure may be considered as present. The air may be allowed to escape slowly or quickly, the former giving the more accurate results. Four tracings, obtained from the patient whose case has been described, with the slow release of the air, are represented in Figs. 7, 8, 9, and 10, and another

[1] *The Johns Hopkins Hospital Reports*, 1904, Vol. XII., p. 53.

[2] *The Clinical Study of Blood-pressure : a Guide to the Use of the Sphygmomanometer*, 1904, p. 93.

Fig. 8.



Figs. 8, 9, 10.—*Tracings taken from the patient with Erlanger's instrument, and slow release of air.*

tracing, taken with the quick release of the air, is shown in Fig. 11. In Fig. 7, the readings of the column of mercury are given at five different spots, the systolic pressure being 175, and the diastolic pressure 75 mm. Hg. In Figs. 8, 9, and 10, only the systolic and diastolic pressures are noted. In Fig. 11, the systolic pressure is 170, and the diastolic pressure 70 mm. Hg.

Sphygmographic tracings of the pulse are shown in Figs. 12 and 13. Fig. 12 gives the ordinary pulse manifested by the patient with a pulse rate of 28, which shows a type closely resembling the pulsus bisferiens, as it is often termed. Fig. 13 is extremely interesting, as it shows the character of the radial pulse after the patient had been treated with atropine applied locally to one of his eyes for severe iritis. The pulse rate, at this time, rose to 74 for a few days. Unfortunately this state of matters occurred during a holiday season, when neither of us was present, and no multiple tracings were obtained of the venous, arterial, and cardiac movements.

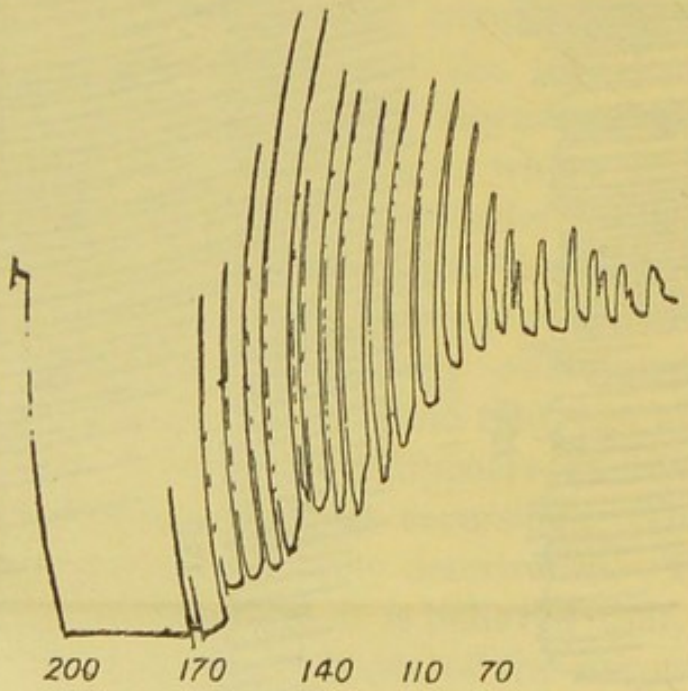


Fig. 11.—*Tracing with Erlanger's instrument and rapid release.*

It may be of interest to refer very briefly to the facts of the most recent instance of heart-block which has been under our observation. A mason, aged 53, was sent into Ward 29 of the Royal Infirmary, suffering from shortness of breath and general weakness. But little could be ascertained as to his hereditary antecedents. As a younger man, he served in the Army, and

he used to be much addicted to alcohol, but for some years had been a temperate man. He had been exposed to climatic vicissitudes of the most pronounced degree, but he had never really, to his knowledge, suffered from any serious illness. The present condition of matters began about five years ago, and,

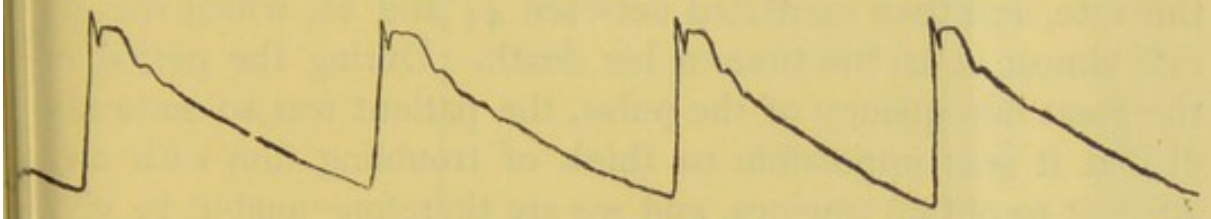


Fig. 12.—*Sphygmographic tracing. Ordinary rate.*

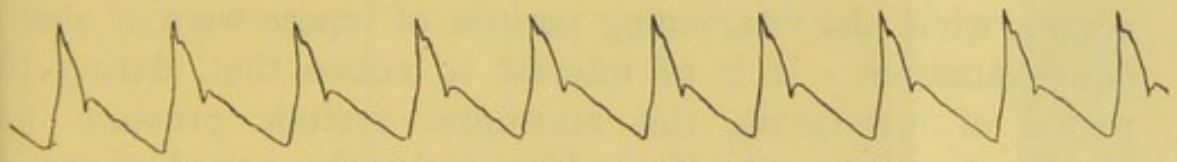


Fig. 13.—*Sphygmographic tracing after atropine.*

with slight variations, had become progressively worse. On admission, he showed marked orthopnœa and considerable cyanosis. The rate of the pulse was from 80 to 90. It was perfectly regular, and almost equal. The apex beat was diffuse, and the heart somewhat enlarged, the right border of the heart being 2 inches, and the left border 5 inches, from midsternum. On auscultation, a galloping rhythm of the sounds was constantly present in the mitral area as well as in the tricuspid area. The first sound was followed by systolic murmurs; the second sound was almost invariably doubled, and the aortic element was greatly accentuated. A rough systolic murmur, obviously of aortic origin, could be heard over the upper portion of the manubrium. The arterial walls were markedly degenerated, distinct calcareous rings being distinctly felt. The systolic pressure was 195, and the diastolic pressure 125 mm. Hg.

There were some evidences of hyperæmia at the bases of the lungs; the liver was somewhat enlarged, but there was no ascites; albuminuria was constantly present. The erythrocytes reached 5,100,000, the leucocytes 11,700, and the hæmoglobin was 80 per cent. The patient was under observation for four months, and, in spite of every method of treatment which

could be thought of, he became progressively worse. During the last three weeks of his life, the rate of the pulse, which had almost invariably been above the normal, showed some tendencies to become subnormal, and, during the last week, it fell to 36 per minute. It remained for two days at, or about, this rate, and then oscillated between 44 and 68, which was its rate almost up to the time of his death. During the period of the great infrequency of the pulse, the patient was so seriously ill that it was impossible to think of troubling him with any attempt to obtain tracings, and we are therefore unable to give any graphic records of his condition. The patient frequently had attacks of Cheyne-Stokes breathing, most of which were characterised by periods of respiration of 30 seconds or thereabouts, while the intervening periods of repose were of almost equal duration. It is of interest to notice that, during the period of respiration, the maximum systolic pressure was usually about 200 mm. Hg., while, during the period of repose, it was usually about 195 mm. Hg. The diastolic pressure always seemed to be 125 in both phases, but it must be remembered that the criterion for this is not so definite as in the case of the systolic pressure.

After death, it was found that there were no lesions of the cusps, but simple dilatation of the mitral and tricuspid orifices with dilatation also of the aorta. The most interesting point lies in the fact that, on careful dissection of the auriculo-ventricular band, it was found to pass into a mass of fibrous tissue. A full microscopic investigation of the condition will be undertaken in the future, and we need only mention, at present, that this case adds another fact to the rapidly accumulating body of evidence in favour of disturbance of cardiac rhythm by means of a lesion in this region.

For much help in the clinical investigation of these two cases, we desire to thank Dr. J. C. D. Allan, who was untiring in his observations, and we also wish to express our obligations to Dr. E. B. Jamieson for his kindness in making a beautiful dissection of the heart in the second case.