NEW METHODS OF STUDYING AFFECTIONS OF THE HEART.

By JAMES MACKENZIE, M.D.,
Burnley.

I.—AFFECTIONS OF THE FUNCTION OF CONDUCTIVITY.

I PROPOSE to endeavour to apply some of the results of recent experimental and clinical discoveries to the explanation of certain phenomena observed in the clinical examination of the human heart. Although, in the main, these papers are explanatory and indicate how certain signs may be recognized, I have accumulated sufficient evidence to show how these signs may profoundly modify our conception of many affections of the heart, and how the views here set forth may have a very practical bearing on the treatment of a great variety of cases of heart disease.

1. THE FUNCTIONS OF THE HEART MUSCLE FIBRES.

Gaskell¹ describes the functions of the muscular fibres of the heart as rhythmicity, excitability, contractility, conductivity, and tonic—*that is to say*, the muscular fibres of the heart possess the power of rhythmically creating a stimulus, of being able to receive a stimulus, of responding to the stimulus by contracting, of conveying the stimulus from muscle fibre to muscle fibre, and of maintaining a certain ill-defined condition called "tone." By virtue of these inherent functions the heart is able to beat and to keep up the circulation of the blood independent of any nervous assistance. In the ordinary sense of the term the heart possesses no motor nerves, the vagus and sympathetic nerves having only a moderating influence upon these varied functions of the heart muscle fibres.

2. ARRHYTHMIAS DUE TO AFFECTIONS OF THESE FUNCTIONS.

While the nature of these functions has been abundantly demonstrated experimentally, little has been done so far to apply the "myogenic" doctrine to the study of heart affections in man. The difficulty has lain in the absence of any facts showing how modifications of these functions would exhibit themselves in the movements of the heart, or in modifying the blood flow in arteries and veins. Engelmann,² in a minute and detailed inquiry into the nature of these functions, demonstrated, among other things, that the first four of these functions may each be affected independently of the others. Wenckebach,³ who had made himself familiar with the most minute details of Engelmann's experiments, conceived the idea that where one of these functions became depressed an arrhythmia peculiar to that function would be manifested. He was able to detect such arrhythmias in the experiments of Engelmann and Hoffman, and afterwards was able to recognize similar types of arrhythmia in certain forms of irregularity in the human heart. His method consisted mainly in taking sphygmographic tracings of the radial pulse and minutely analysing the variations in rhythm. As Engelmann's results were obtained from the dying heart of the frog, it seemed rather a far cry to compare these with those of a fairly healthy human heart. On these grounds objections were plentifully raised against Wenckebach's views, and as his observations depended on inferences only, his position seemed somewhat untenable, particularly as arrhythmias bearing a superficial resemblance to some of his special types could be referred with certainty to other conditions. As I had pursued the study of heart irregularities by a different and more complicated method whereby I could frequently demonstrate the parts played by both auricles and ventricles, I was able to demonstrate the correctness of Wenckebach's interpretations in many of the most striking arrhythmias.

3. NATURE AND FUNCTIONS OF THE MUSCLE FIBRES CONNECTING AURICLE AND VENTRICLE.

While the functions that have been cited are inherent in all muscle fibres, some of these functions are more highly developed in certain portions of the heart muscle than in other portions. The muscle fibres at the mouth of the great veins and adjoining portion of the right auricle (remains of the sinus venosus) possess the greatest power of automatically creating the stimulus for contraction, and because of this the wave of contraction normally starts here, and passes in a peristaltic manner over the rest of the heart "with varying speed, quickest over the tissue which has become modified

so as to approach more nearly in its properties to ordinary striated muscle—namely, the reticulated bulged portion of the auricle and ventricle—more slowly over those parts which retain a more embryonic character—namely, the auriculo-ventricular muscular ring and the bulbus arteriosus."

Gaskell further points out that the muscular tissue joining the auricle and ventricle (canalis auricularis) shows a less-developed type of muscle fibre, being less striated, a condition which has been verified to exist in the mammalian heart by Kent, His, jun., and others. This muscle tissue is distinct in its functions from the more striated muscle fibres in that the wave of conductivity passes more slowly over its fibres, and it possesses a greater power of automatically creating a stimulus for contraction. In the human subject these peculiarities can be recognized, and their recognition explains satisfactorily a great number of obscure actions of the heart some of which have hitherto escaped attention, while others have been completely misunderstood. In this paper I shall deal only with the evidence of the first of these peculiarities—that is, the nature of the conducting power, whereby the stimulus is conveyed from auricle to ventricle, leaving for a subsequent communication the description of the results of over-excitability of these fibres.

4. METHOD OF ESTIMATING THE CONDITION OF THE CONDUCTIVITY.

This function is recognized by the time that elapses between the auricular systole and the ventricular. The method of studying the movements of the heart by taking simultaneous tracings of the radial and jugular pulses affords an exceedingly simple and readily demonstrable method for observing the conductivity in man. To obtain a tracing of the jugular pulse a shallow, open cup is placed over the lower end of the relaxed sterno-mastoid muscle. This cup (a receiver) is connected by means of a tube with a tambour, which is fitted on a Dudgeon or von Jaquet sphygmograph in such a manner that the lever of the tambour can be made to write on the same paper and at the same time as the sphygmograph lever inscribes the movements of the radial pulse. It is frequently impossible to get tracings of the jugular pulse without some movement due to the carotid. With the radial pulse as a standard one can with certainty assign the part of the tracing due to the carotid and the part due to the jugular. It often happens that the only influence exerted by the carotid is a jerk of the lever due to the sudden tension of the carotid at the beginning of the ventricular systole and manifested in the jugular tracing by a short, sharp wave (c in all the tracings), as in Fig. 1.

Fig. 1.—The space E represents the time of the ventricular systole. The wave a is due to the right auricle, and the wave c to the carotid impact. The space A represents the a-c interval. The time marked in all these tracings equals one-fifth of a second.

At other times the jugular pulse is so faint that only a slight elevation is obtained, and the tracing from the neck is otherwise entirely due to the carotid as in Fig. 2.

Fig. 2.—The upper tracing is from the neck and shows a small wave (a) from the jugular vein, and due to the right auricle, preceding the large carotid wave.

For the special purposes of studying affections of conductivity this interference by the carotid is a fortunate circumstance, inasmuch as it provides a readily recognizable point

in the cardiac cycle from which to gauge the interval between the auricular and ventricular systoles.

5. THE INTERSYSTOLIC PERIOD (*a-c interval*).

This interval is occupied by three events—namely (1) the systole of the auricle; (2) the transmission of the stimulus from auricle to ventricle; (3) a minute portion of time taken up by the interval during which the ventricular pressure is rising before opening the semilunar valves (*Anspannungszeit* or presphygmic interval). As 1 and 3 are practically constant, they may for the purpose of this inquiry be ignored, any variation in the length of the *a-c* interval being due to variation of the rate of stimulus conduction.

In normal hearts I have found that the *a-c* interval is fairly constant, lasting usually one-fifth of a second (Fig. 1). Sometimes it appears a little shorter. The rapidity with which the tracing paper moves does not separate the events widely enough to enable me to measure the changes with sufficient minuteness. Still the differences can be appreciated readily by the eye.

6. DEPRESSION OF CONDUCTIVITY WITHOUT ARRHYTHMIA.

While the *a-c* interval may be considered normal when it does not exceed one-fifth of a second, as in Fig. 1, considerable increase of this interval may take place with no manifestation in the rhythm of the heart. The recognition of this

Fig. 3.—The *a-c* interval (space A) is twice the normal period, lasting two-fifths of a second in place of one-fifth as in Fig. 1, indicating a delay in the stimulus passing from auricle to ventricle (taken in 1904).

Fig. 4.—The *a-c* interval (space A) equals two-fifths of a second. (Taken in 1892 from the same patient as gave Fig. 3.)

depression of conductivity can only be afforded when we have evidence of the contraction of the auricles.

In Fig. 3 there is a tracing of the jugular and the carotid pulses taken at the same time as the radial pulse. The radial shows a perfectly regular rhythm, while the neck tracing shows a great increase in the *a-c* interval equal to two-fifths of a second (space A). The heart may continue to beat perfectly regularly for years with the conductivity affected to this extent. Thus Fig. 4 was taken from the same patient as gave Fig. 3 in 1892, and the jugular tracing shows a like increase of the *a-c* interval to two-fifths of a second. Except for a short period in 1898 (Figs. 8 and 9) this patient's pulse was quite regular up to 1904.

7. INFLUENCE OF REST UPON CONDUCTIVITY.

With each contraction all the functions of the muscle fibres are for the time being abolished. During diastole these functions are gradually restored. Normally their recovery is simultaneous, so that when the rhythmical stimulus arises at the auricle or great veins it passes over the whole heart, and the muscular fibres respond at a uniform time. When one of these functions is depressed, its recovery does not take place

as speedily as the others, therefore the relationship of the action of these functions is not uniform, and an interference with the regular sequence of events results. Certain variations in the rate of conduction can frequently be detected, not only when conductivity is depressed, but also when it is presumably normal, such variations apparently depending on the time required for recovery after the previous exhaustion.

Fig. 5.—The carotid and jugular tracing shows a shortening of the *a-c* interval (space A₂) after the long diastolic periods and a lengthening (space A₁) after the short diastolic periods of the cardiac cycle.

One has little difficulty in illustrating this in a variety of ways in certain forms of heart irregularity. In a patient aged 24, and pregnant with her first child, suffering from mitral disease, I had noted an increase in the *a-c* interval for some years. During labour it frequently happens that the pulse becomes irregular, the irregularity often being of a respiratory type. The radial tracing in Fig. 5 shows such an irregularity.

The tracing of the carotid and jugular shows a rise and a fall due to the movements of respiration—the rise in the tracing corresponding with expiration, and the fall during inspiration. It will be noted that there is a distinct lengthening of the radial pulse periods at the beginning of inspiration. It will be seen that the *a-c* interval during the short pulse period (space A₁) is nearly double the duration of the *a-c* interval during the long pulse period (space A₂). The reason for this variation in the *a-c* interval is that the conductivity is continually depressed, but when the heart beats at a slower rate the conductivity has time to recover, so that the stimulus is conveyed from auricle to ventricle at about the normal rate (A₂). Here the arrhythmia is not originally due to the depressed conductivity, but evidently starts at the auricle, for the auricular wave, *a*, appears at irregular intervals. The modifying effect on the ventricular rhythm of the variable condition of the conductivity can be illustrated in a variety of ways. If we examine minutely the incidents where an occasional premature or extra systole occurs, sometimes very curious variations in conductivity can be detected.

When a stimulus arises prematurely in the auricle, the conductivity has not yet been completely restored, therefore the stimulus takes a longer time in passing to the ventricle, and as a consequence there is an increase of the *a-c* interval.

The patient from whom Fig. 6 was taken always showed a slight but distinct depression of the conductivity, as can be observed by the slight and varying length of the *a-c* interval (spaces A₁ and A₂). The radial pulse shows two premature pulse beats (x x) with corresponding premature beats (c') in the jugular tracing. These premature beats are due to extra systoles of auricular origin, for it will be observed that in the jugular tracing the waves *a'* occur too soon. The interval (A₃) between *a'* and *c'* is greater than the average *a-c* interval (A₂) and is very markedly greater than the *a-c* interval (A₁) following the premature contraction. The shortening of the

Fig. 6.—Shows two premature or extra systoles of auricular origin (x). The waves *c'* in the jugular tracing occur at the same time as the small premature beats (x) in the radial tracing, and are therefore due to the carotid. These are preceded by premature waves *a'* due to the auricle. The interval A₃ between *a'* and *c'* is greater than the average *a-c* interval (A₂), and is much greater than the following *a-c* interval (A₄).

$a-c$ interval (A_1) is due to the more complete restoration of the function of conductivity in consequence of the longer pause after the auricular systole a' . Good illustrations of the shortening of the $a-c$ interval after periods of longer rest is to be seen in Figs. 11 and 12.

It may seem an unnecessary refinement to dwell upon such minute changes, but I trust to show later that the recognition of these facts affords the evidence for determining with certainty some changes of the utmost significance in a large class of cases of heart disease.

8. ARRHYTHMIA DUE TO DEPRESSION OF CONDUCTIVITY.

Slight arrhythmia may be due to variations of conductivity. Thus the short space o in the radial in Fig. 6 is seen to be due to an increase of the preceding $a-c$ interval (space A_1), that is, to a delay in the ventricular systole.

Arrhythmia of a more marked character arises when the conductivity is so grievously depressed that the stimulus occasionally or frequently fails to cross the auriculo-ventricular junction. How this occurs is well seen in Fig. 7.

Fig. 7.—Shows a gradual lengthening of the $a-c$ interval till a radial and carotid pulse beat drops out after the auricular wave a' . The following $a-c$ interval is shortened.

There is a constant delay in the conduction of the stimulus here, the $a-c$ interval being unduly prolonged. Before the intermission there is a slight but gradual increase of the $a-c$ interval. After the auricular wave a' there is no carotid wave (c) nor pulse beat in the radial. The reason for this is manifestly that the auricular systole a' occurred so soon after the previous ventricular systole (as evidenced by the carotid wave c immediately before a') that there was not sufficient time for the recovery of the function of stimulus conduction in these fibres, and hence no stimulus reached the ventricle and a beat dropped out. By this means a longer rest was procured for these fibres, and when the next stimulus comes down from the auricle the longer rest has so restored the function of conductivity that the $a-c$ interval following the pause is shorter than the average. In Fig. 8 there is a dropping out of ventricular beats at regular intervals.

Fig. 8 was taken in 1898 from the patient from whom Figs. 3 and 4 were taken. For many years tracings from this patient showed constant though slight depression of the conductivity. For some reason in 1893 this conductivity became further depressed so that at regular intervals a ventricular

Fig. 8.—Regularly intermittent pulse due to depression of conductivity.

beat dropped out (Fig. 8). The true nature of this arrhythmia is shown in the jugular tracing (Fig. 9), where the auricle is shown to contract regularly (wave a), while the ventricle fails to respond to every third auricular systole. To demonstrate this more clearly I reconstruct Fig. 9 in the form of a diagram

Fig. 9.—Taken at the same visit as Fig. 8, and shows the venous pulse during the arrhythmia. The wave a is quite regular in its appearance.

(Fig. 10), after the manner of Wenckebach, modifying it so as to show the actual site of delay at the fibres joining auricle and ventricle. The down strokes in the upper division re-

Fig. 10.—Diagram constructed to show that the arrhythmia in Figs. 8 and 9 is due to a blocking of the conductivity at the fibres joining auricle and ventricle (A-Vf.).

present the auricular systoles (A_s). The down strokes in the lower division represent the ventricular systoles (V_s), and correspond with the radial and carotid pulses in Figs. 8 and 9. The thick, slanting lines represent the conduction at the fibres joining auricle and ventricle (A-Vf). It will be seen that a ventricular beat drops out regularly after every third auricular systole.

This form of arrhythmia has been described by Wenckebach and its cause inferred from the manner in which the radial pulse varied. His proof rested on similar variations in the movement of the frog's heart described by Engelmann, and it affords me much pleasure to be able to verify the interpretation of this shrewd observer.

9. BRADYCARDIA DUE TO DEPRESSION OF CONDUCTIVITY.

Gaskell states that on applying a screw clamp around the auriculo-ventricular groove of a frog's heart, "according to the tightness of the clamp the ventricle can be made to beat synchronously with the auricles, to respond to every second contraction of the auricle, to respond to every third, fourth,

Fig. 11.—From a slow irregular pulse after influenza. The jugular tracing shows that the bradycardia was due to the ventricle failing to respond to every auricular systole. Note that after the short pulse period in the radial the $a-c$ interval is much longer than after the other periods. Note also at x a slight depression in the radial tracing due to the action of the left auricle affecting the arterial column.

or other contraction, or to remain quiescent." All these varying results can be demonstrated to occur in the human heart.

In Fig. 11 there is one short pulse period and the $a-c$ interval here is much longer than the $a-c$ period after the longer periods, the lengthening being an evidence that the conducting power of the fibres at the auriculo-ventricular groove have not had time to recover as effectually as after the longer pulse periods. In Fig. 12 there are three auricular contractions to one ventricular, except during the last arterial pulse period, when there are but two auricular waves, and after the second of these the $a-c$ interval is longer than the other $a-c$ intervals in this tracing.

In an excellent tracing published recently by Gibson¹ there are four auricular beats to one ventricular. This blocking may be so extreme that the auricle may beat ten or twelve times and the ventricle stand still. When this occurs anaemia of the brain induces syncope and epileptiform seizures (Adams-Stokes syndrome). I have seen a few such cases, but unfortunately I was not able to take tracings during the attacks, but the classical case so remarkably well recorded by Webster² demonstrates these points most conclusively.

10. INDEPENDENT VENTRICULAR RHYTHM DUE TO HEART BLOCK.

In the tracings I have given so far it could be shown that when the ventricle does contract it is in response to a stimulus from the auricle. When a ligature is applied in the auriculo-ventricular groove of the frog's heart, so that the stimulus can no longer be conveyed from auricle to ventricle, after a time the ventricle will beat with a rhythm different from, and independent of, that of the auricle (heart block).

Fig. 12.—Shows a more extreme form of bradycardia, where the ventricle responds to every third auricular systole. The large wave (c) is due to the carotid. In the last period there are but two auricular waves and the a-c interval is longer than after the period with three auricular waves.

This independent rhythm can be demonstrated as the cause of certain forms of bradycardia in the human subject. Fig. 13 is a tracing of the radial taken at the same time as the pulsation in the neck due to the jugular and carotid. The small waves, *a*, are due to the right auricle, and there are two of these auricular waves to one carotid or radial pulse beat. But when one carefully analyses their relationship it is found that the two auricular periods are less than one ventricular period, so that the relationship of the auricular systole to the ventricular systole is a constantly varying one, sometimes at a distance, then gradually approaching till they are synchronous. Whatever relationship the *a* has to *c* no variation takes place in the rate. The same thing is shown in Fig. 14, where the apex beat is recorded at the same time as the radial. Here there is a small wave, *a*, due to the systole of the left auricle, and the same maintenance of the independent rhythms is manifest

Fig. 13.—Shows in the tracing from the neck that the auricle, *a*, beats regularly and at more than twice the ventricular rate. The varying length of the a-c intervals showed that the ventricle pursued a rhythm of its own independent of the auricle. Note the small dips, x, in the radial tracing, due to the movement of the left auricle affecting the arterial column.

administration of chloroform showed the same uniform pulse-rate. Professor Wenckebach informs me that in a similar case a glass of port wine considerably quickened the rate of the auricular contractions, while the ventricular rate was unaffected. To demonstrate the evidence of affections of the functions of the

heart muscle fibres I will describe in some further papers the effects of digitalis on the human heart, as this drug has a remarkable effect on the individual functions.

REFERENCES.

- 1 Gaskell, W. H., article, The Contraction of Cardiac Muscle, *Schäfer's Textbook of Physiology*.
- 2 Engelmann, Th. W., *Myogene Theorie und Innervation des Herzens, Sonderabdruck aus die Deutsche Klinik*, 1903.
- 3 Wenckebach, K. F., *Arrhythmia of the Heart*, translated by T. Snowball. Green and Sons 1904.
- 4 Gibson, G. A., *The Nervous Affections of the Heart*, Fig. 23. Pentland, 1904.
- 5 Webster, A., *Cardiac Arrhythmia in Relation to Cerebral Anaemia and Epileptiform Crises*, *Glasgow Hospital Reports*, 1901.

Fig. 14.—From the same case as Fig. 13. Shows the influence of the left auricle on the apex tracing (waves *a*). In the radial tracing the dips x due to left auricle are found to occur immediately after the auricular waves in the apex tracing.

SOME PRACTICAL ASPECTS OF CONJUNCTIVAL BACTERIOLOGY.

By FREELAND FERGUS, M.D., F.R.S.E.,
Surgeon to the Glasgow Eye Infirmary.

I DESIRE to say something as to the necessity and utility of daily bacteriological work in the modern eye clinic. The science of pathology and the science of therapeutics are sisters. The problem to be solved is the treatment of disease. The object of pathology is to explain the origin of disease, and the ideal object of rational therapeutics is to apply the teachings of pathology to its cure or to its alleviation. In all its departments medicine is in many respects more of an art than a science, and probably to a large extent must remain so; but, unquestionably, recent pathological advances have done a great deal to illumine the work of the practitioner, and to bring his efforts more into line with the biological sciences.

Thanks to the pathologists, we now have clearer ideas as to

the nature of the many forms of inflammation of the conjunctiva, which in days gone by were all classed together as catarrhal ophthalmia, and were for the most part put down to exposure to cold, or to other indefinite causes. Some considerable number of years ago it came under my notice that a number of persons, who all lived in the same tenement in High Street, Glasgow, were suffering from acute catarrhal conjunctivitis.

I investigated the matter at the time, and found that no fewer than forty persons were thus affected in this one tenement. It seemed to me, then, that we here had to deal with something which was epidemic, and to some extent analogous to one of the specific fevers. Some time later I read a very interesting paper by Dr. Weeks, which seemed to afford a complete explanation of such occurrences. In that paper he described his bacillus and the contagious catarrh to which it gives rise.

Many changes have taken place since then, and thanks to the labours of several distinguished pathologists we now know a considerable amount about the bacteriology of the conjunc