A Clinical Lecture

ON

SOME FORMS OF CARDIAC ARRHYTHMIA FROM THE POINT OF VIEW OF THE MYOGENIC THEORY.

ONE OF A COURSE OF LECTURES AND DEMONSTRATIONS GIVEN BY THE STAFF OF THE HOSPITAL FOR DISEASES OF THE CHEST, BROMPTON.

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THE early physiologists regarded the beat of the heart as due to the direct action of the blood in its chambers; but when it was shown that the heart of certain vertebrates, such as the frog, could beat when removed from the body and in the absence of a blood flow, the original view was discarded for the one which regarded the heart beat as the result of an inherent function of heart tissue, which was then said to possess rhythmical properties.

With the later discoveries of Remak, Bidder, and Ludwig of ganglion cells in the heart this conception was again modified. Analogy with the nervous origin of the contraction of other striped muscle and also with the phenomena of respiration led to the neurogenic conception of the cardiac movement, according to which the origin of the beat is to be looked for in stimuli arising in the intracardiac ganglia which initiate the contraction of the various parts of the organ, the function of the vagus and sympathetic fibres being to modify the action of the primary intracardiac system according to the needs of the moment.

This theory held the field until Gaskell showed, in 1883, that the action of the nervous system upon the heart is different from that upon skeletal muscles, and that the heart is an automatic organ whose functions are inherent in the muscle and primarily independent of the nervous system. This may be said to have been the origin of the myogenic theory. The myogenic theory states that the stimulus to the heart's contraction is formed in the heart muscle cells themselves, and is not conducted from the nervous system. This contraction stimulus also is not conducted from the place of its origin to the rest of the organ by nerve pathways, but is directly conveyed by muscle cells, evoking in its passage the orderly contraction of the heart's chambers.

These properties of stimulus formation and conduction of irritability and contractility are fundamental attributes of cardiac muscle, which, whilst having certain relationships to each other, are yet capable of independent variation within comparatively wide limits. The co-operation of these properties of the muscle cells produces the rhythmical movement of the heart, but its adaptation to the varying needs of the body is brought about by the action of the nervous system upon one or other of these functions of cardiac muscle.

The heart has been compared to a regularly trotting horse, which, endowed with automatic powers of movement, is yet rendered more useful to the needs of daily life by the control exercised by its rider, which in this instance is represented by the nervous system.

The properties of stimulus formation and conduction, of irritability and contractility, are possessed by the muscle of the entire heart, but not in equal degree.

Briefly and for the sake of clearness it may be said that the cardiac muscle is divided morphologically into two great systems, the one larger and more prominent, which subserves the function of propulsion, in which stimulus production has, in the course of development, fallen more or less into disuse, whilst the other functions, more especially contractility, have become increased. The second system, which subserves stimulus production and conduction, exists in special positions only as a more primitive type of tissue. In order to make myself clear I must direct your attention for a few moments to certain anatomical and physiological aspects of the heart.

The primitive organ consists of a tube, endowed with a capacity to initiate rhythmical contractions, which, starting at one end, travel as a peristaltic wave to the other, and this remarkable property exists also in the embryos of many vertebrates long before the most rudimentary nervous system has been developed either locally in the heart or in any way brought into relation with the organ from afar.

In the course of development this primitive tube has undergone various modifications, chief of which are a folding upon itself and an accession of muscle fibres in such positions as will enhance the value of the organ as a force-pump; at the same time a portion of the primitive tissue remains, and with it the property of initiating contractions at the venous end and of conducting them to the arterial end without calling in the aid of nervous elements—that is to say, whilst the propulsive system increased and largely covered up and obscured the more primitive, there still remains sufficient less specialized tissue to subserve stimulus production and conduction.

In the hearts of the lower vertebrates, for example, the frog, the great veins open into a common chamber, the sinus venosus, and this again communicates directly with the right auricle. In higher forms the sinus no longer exists as a separate compartment, but in the course of development has been included in the right auricle at the point of entrance of the venae cavae, and is thus directly continuous with the auricular wall. In the frog, similarly, there is direct muscular continuity between the auricle and common ventricle, but in man no muscular connexion between the auricles and ventricles was known until Kent and His, jun., in 1893 demonstrated the existence of a muscular connexion in the form of a narrow band of peculiar muscle fibres, which, taking origin near the orifice of the coronary sinus in the right auricle in a small swelling, the auriculo-ventricular node, crosses the auriculo-ventricular junction, and having divided into two branches, one for either ventricle, subsequently passes in the subendothelial tissue as the Purkinje fibres, to be distributed to the muscle cells of the ventricles and musculi papillares. This band has been named the "auriculo-ventricular bundle," and forms the sole muscular pathway between auricle and ventricle.

More recently Keith and Flack have demonstrated the existence, at the junction of the superior vena cava and right auricle, of a ring of peculiar muscle tissue which they name the "sino-auricular node," and which has a structure similar to that of the auriculo-ventricular bundle and node, and is furnished with a separate blood supply from both coronary arteries. The connexion between this ring of tissue and the auriculo-ventricular node is still uncertain, but appears to be by means of the auricular muscle fibres.

I must now remind you of two properties of cardiac muscle which are of the highest importance for a right understanding of the subject. I refer to the law of maximal contraction, according to which every stimulus that is sufficient to produce a contraction evokes the best of which the heart is capable at the moment—that is to say, the force of the contraction is independent of the strength of the stimulus; it is a case of "all or none."

The second property of cardiac muscle is the occurrence of what is called "the refractory phase," which means that during systole the heart cannot be stimulated to further contraction; it has become refractory, and all its functions are for the time being abolished, to be gradually restored at different rates after the contraction is over; from this it will be obvious that the heart cannot be tetanized, but will respond by rhythmical contractions to a continuous stimulus.

The refractory period begins shortly before the occurrence of contraction, and lasts until shortly after its cessation, after which the excitability is only gradually restored, reaching its maximum just before the time of the next contraction.

In considering the functions of stimulus production and conduction, irritability and contractility, and their relations to cardiac arrhythmia, it may at once be said that of the nature of stimulus production little that is really definite is known. That both auricles and ventricles possess the power in different degree of originating a contraction stimulus is clear, both from clinical and experimental observation. To the clinical aspect I shall

have to refer later on, but experimentally it has been found that if a ligature be placed round the junction of sinus and auricle in the frog's heart (Stannius ligature No. 1), the auricles and ventricle stop beating, whilst the sinus maintains a normal rhythm. After a period the auricles and ventricle commence to beat, but with a rhythm that is slower than that of the sinus. A state of things comparable to this occurs if the ligature be placed around the auriculo-ventricular junction (Stannius ligature No. 2); here the auricle maintains its rhythm, whilst the ventricle, after a period of standstill, takes on an independent rhythm of its own, which is, however, always slower than that of the auricle. It is thus evident that the stimulus to contraction reaches the point of effectiveness first at the sinus, or, in man, at the great veins entering the right auricle; also that the stimulus to contraction may originate either in the auricle or ventricle, but that, since the point of effectiveness is reached first at the sinus, the rate of the heart beat is set from this point. The generally accepted view is that, normally, stimulus matter is in constant formation, but reaches the effective point first at the sino-auricular node—that is, the superior vena cava—and evokes a contraction there which extends to the auricle, and from here, by way of the auriculo-ventricular bundle, where conduction is slower, to the ventricle; but the occurrence of a contraction not only destroys for the time being irritability, contractility, and conductivity, but the whole of the stimulus matter as well, which, therefore, has to be re-formed after every systole, and the time required to do this, other things being equal, determines the rate of the heart beat. This periodicity is also in some way under the control of the nervous system.

The abolition of these functions and their gradual restoration is of great importance in preserving the regularity of the cardiac rhythm. The efficiency of the properties of the heart muscle appears to bear a definite relationship to the length of the intervals of time which elapse between their being called into activity. For instance, if, whilst the heart is acting regularly, an extra auricular contraction occurs interpolated between the regular beats, in the absence of this slow return to effectiveness, a ventricular contraction would take place after precisely the same period as occurs normally, but owing to the exhaustion of the auriculo-ventricular bundle, and the insufficient time allowed for its recovery after the previous normal contraction, the time occupied by the passage of the stimulus over it is lengthened, the ventricular beat is therefore delayed and its rhythm less disturbed than would otherwise be the case.

The diagram which I now show you illustrates this

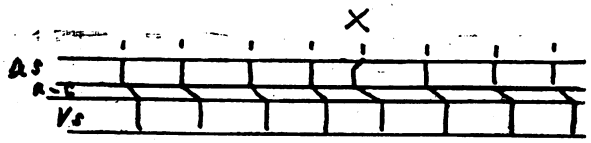

Fig. 1.—As, Auricular systole; Vs, ventricular systole; *a-c*, the passage of the stimulus over the auriculo-ventricular bundle. The short lines above the diagram represent the time of the stimulus from the great veins. Here it will be seen that an auricular extra-systole has occurred at the point ×, but that the period of time elapsing between the auricular and ventricular contractions is considerably longer than occurs at the normal beats.

delay very well; the upper vertical lines represent the beginning of the auricular contractions, and the lower the beginning of the ventricular contractions, whilst the oblique lines joining these two show the interval taken up by the passage of the stimulus over the auriculo-ventricular bundle—generally termed the *a—c* interval. I shall have to explain how this diagram is arrived at later, but for the present it will be seen that while the oblique lines are for the most part of fairly constant length, the one joining the interpolated auricular and ventricular beats is considerably longer than the rest, showing that the time occupied by the stimulus in passing over the auriculo-ventricular bundle is increased, and that consequently the ventricular rhythm is less disturbed.

Perhaps the simplest form of arrhythmia, and in many ways the most important, is the disturbance of rhythm which is termed the "extra-systole"; but before discussing this form of arrhythmia and its mechanism, it will be well to describe briefly the method adopted of graphically recording the pulse, and of measuring the times of the various events.

Dr. James Mackenzie has shown that it is possible to obtain records of the movements of all four chambers of the heart, but for our present purpose it is only needful to describe two, that is, those of the left ventricle and right auricle. The events taking place in the ventricle can be shown from a radial tracing obtained by means of a sphygmograph, or by recording the movements of the apex, but for various reasons the former is the one usually adopted. The events taking place in the right auricle are shown by applying a tambour over the jugular vein, in connexion with which a needle writes upon a smoked surface. It is so arranged that records of the radial and venous pulses are made simultaneously upon the same paper, and in this way the movements of the two chambers can be compared together, and by using the radial tracing as a standard of time their relations can be easily determined.

In interpreting the radial tracing obtained in this way it must be borne in mind that the opening of the aortic valve practically corresponds with the beginning of the rise of the pulse-wave, and its closure with the dicrotic notch, allowance being made for the delay of the pulse behind the heart beat, whilst the tricuspid valves open at a point somewhat before the occurrence of the dicrotic wave. Thus, by measuring the distance between the beginning of the rise and the dicrotic notch, the duration of the expulsive period from the ventricle can be made out.

When a tambour is applied to the great veins of the neck, and a record made upon a blackened paper, its characters are those shown upon the tracing which I pass round, and are said to be of the ordinary auricular type.

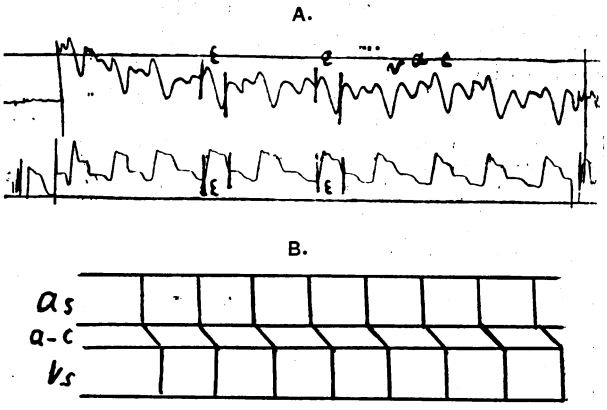

Fig. 2.

A.—*v*, Ventricular wave; *a*, auricular wave; *c*, carotid wave. The space *e* indicates the period during which the aortic valves are open.

B.—In this diagram As equals auricular, and Vs ventricular systole; *a-c*, the time occupied by the stimulus in passing over the auriculo-ventricular bundle.

It will be seen that there are several waves and depressions the interpretation of which, according to Mackenzie, is as follows: The rise marked *a* (Fig. 2) is due to contraction of the right auricle, which sends a wave back into the cervical veins; this, of course, takes place before ventricular systole, and is followed by a fall which is interrupted by a small wave *c* due to the impact upon the air in the receiver by the carotid pulse; this fall is caused by diastole of the right auricle, and is succeeded by a wave of medium size, marked *v*, the ascending limb of which is due to the filling of the auricle and great veins by the onflowing blood; at the apex of the wave the tricuspid valves open, and the fall which immediately follows is the result of the escape of the blood stored in the right auricle into the ventricle through the recently opened valve. The next rise is caused by the auricular contraction.

Now, if the interval measured in the corresponding radial tracing, between the beginning of the pulse wave and the dicrotic notch, be compared with the same period in the auricular tracing (marked *e* in both), the events taking place in the auricle during the expulsion period from the

ventricle can be shown, and the relation between the movements of the two chambers fairly easily made out.

The end of the period *e* does not, of course, correspond with the apex of the wave *v*, the brief interval between them being due to the time occupied by the ventricular relaxation before the intraventricular pressure is so far reduced as to allow the tricuspid valves to open.

From the venous tracing a diagram (Fig. 2 B) can be made, which is especially useful to demonstrate the various cardiac events in their relationship to one another, and which I have already shown you in connexion with the delay of the stimulus which, under certain circumstances, occurs in its passage across the auriculo-ventricular bundle. Short vertical lines are drawn corresponding to the beginning of the auricular waves; these lines are of equal length, and are joined top and bottom by two parallel lines. A third horizontal line is now drawn at a short distance below, from which a second series of vertical lines are dropped, corresponding in position to the beginning of the carotid wave; this set of lines being of equal length, their ends are joined by a fourth horizontal line. There are thus formed three horizontal spaces; in the upper one vertical lines represent the beginning of auricular contraction, and in the lower approximately the beginning of ventricular contraction. In the middle space, termed "the auricular-contraction interval," the upper and lower vertical lines corresponding to one another are joined obliquely; these lines correspond in length to the time occupied by three events—(*a*) auricular contraction, (*b*) the passage of the stimulus over the auriculo-ventricular bundle, and (*c*) the brief period which elapses between the commencement of ventricular contraction and the opening of the aortic valve. Since, however, the times occupied by the auricular and that part of the ventricular contraction mentioned are practically constant, the length of the oblique line will be proportionate to the time taken up by the stimulus in crossing the auriculo-ventricular bundle. This method of measuring the rate of passage of the stimulus over the auriculo-ventricular bundle gives, of course, only approximate results, but it is sufficient for the purpose.

It has been shown that in healthy, slowly-acting hearts the time so occupied is fairly constant at about one-fifth of a second, but decreases with an increase in the heart's rate. Bearing this in mind, it will be seen that if the interval between the beginning of auricular contraction and the opening of the aortic valve be measured, any alteration in its length will be due to delay in the passage of the stimulus over the auriculo-ventricular bundle; and therefore, in tracings of the jugular pulse, the relative times occupied by the passage of the stimulus over the auriculo-ventricular bundle will be obtained by measuring the interval between the beginning of the auricular and carotid waves.

You are familiar with the phenomena of pulse intermission as occurring in everyday practice: when examining an otherwise regular pulse a beat is occasionally omitted, or, if not entirely omitted, is found to have become exceedingly small and to follow the preceding beat after an interval that is obviously too short; the succeeding pause is generally too long, and the beat which terminates it full and large.

On auscultating over the cardiac apex in these cases it will be found that at the beginning of the intermission the heart appears to give two beats in quick succession; a normal first and second sound are heard, and after a short pause a first sound is again heard, which may or may not be succeeded by a second. There is then a long interval of silence, and the ordinary rhythm reappears. It will be readily understood that the reason for the intermission of the pulse here depends upon the ineffectiveness of the second heart beat of the pair just described to communicate a wave to the wrist. Indeed, as indicated by the occasional absence of a second sound, the contraction is not always sufficient to open the aortic valves. This weak beat is due to an extra-systole, and not, for reasons which will appear subsequently, to a premature physiological contraction.

Now it will be evident that as long as the intermission is only present at infrequent intervals an altogether unimportant degree of irregularity will ensue, and the rate of the pulse will not be appreciably altered; but if an extra-systole occurs more often, occasionally producing a small beat at the wrist, and occasionally none at all, or if a series of extra-systoles occurs at irregularly-disposed intervals, very marked forms of pulse arrhythmia may arise.

To put a not uncommon case: Where every second beat is insufficient to produce a radial wave it is evident that an apparent bradycardia of a very marked kind will follow. Since, however, the ventricular beat can always be shown to be present by auscultation or other method of examination, a false and not a true bradycardia exists. Again, it will be fresh in your minds that the extra beat takes place after an interval that is too short and precedes an interval that is too long, which is itself terminated by a beat unduly large. Now if this sequence of events takes place, as it may do, throughout a considerable period of time, a kind of "regular irregularity" will be produced, in which the apex beats and pulse waves will occur in pairs of alternating large and small beats, each pair separated by a long pause. There will be present the so-called "pulsus bigeminus," which, it should be noted, is not necessarily due to disturbance of the true cardiac rhythm, but to the interpolation of an extra beat. In the same way the beats may occur in groups of three, four, and so on.

The explanation of the extra-systole depends upon the two laws already stated which govern the action of cardiac muscle. It has been found experimentally that immediately after the refractory phase is over the ventricle becomes excitable for strong stimuli, and that, owing to the gradual return of its functions, it will respond to stimuli that are weaker as they are applied later in diastole. In this way an extra-systole can be produced experimentally and is always followed by a long pause. This pause has been called the "compensatory pause," from its having been regarded at one time as a period of rest or compensation for the extra work done by the heart. On measuring the interval between the beats in tracings obtained by this experiment, it is found that the period of time elapsing between the normal beat which precedes the extra-systole and the beat which terminates the compensatory pause, that is, the post-compensatory systole, is equal to two pulse periods. The true interpretation of the so-called compensatory pause has thus been shown to be that, as the result of an extra-systole, the ventricle is still so far refractory as to be incapable of responding to the next physiological stimulus. This stimulus is consequently of no effect, the normal contraction drops out, and none occurs until the physiological stimulus next after it reaches the ventricle. Thus the contraction which follows the compensatory pause takes place in the position which it would have occupied had there been no extra-systole at all.

Now, when a radial tracing is taken from a case showing pulse intermission precisely similar phenomena can be demonstrated. It will be found that the interval between the last normal and the post-compensatory waves is equal to two pulse periods, just as occurs in experiment where an extra-systole is artificially produced—that is to say, a normal beat is followed by a small, prematurely-occurring beat, and this again by a compensatory pause and large post-compensatory systole.

That this is a true extra-systole and not a premature physiological beat is shown by the occurrence of a compensatory pause, which, as previously explained, is the result of the dropping out of a normal contraction and the appearance of a physiological beat at the point which it would have occupied had the extra-systole not occurred.

It may be here noted that though the time between the preceding normal and post-compensatory systoles has been spoken of as equal to two normal pulse periods, it is, in fact, a little too short—a phenomenon explained by the longer rest that the heart muscle has during the compensatory pause, in consequence of which the activity of the muscle is increased and contraction follows sooner upon the appearance of the stimulus. A similar explanation holds in respect of the larger size of the post-compensatory wave; the longer diastolic period allows a more complete filling of the ventricle, a greater contractile energy, and a lower aortic pressure.

In the form of extra-systole just described it is assumed that the stimulus to contraction arises in the ventricle, whilst the auricle maintains an altogether unaltered

rhythm, as will be seen from the diagram taken by Dr. James Mackenzie from observations made simultaneously upon the radial and venous pulses (Fig. 3).

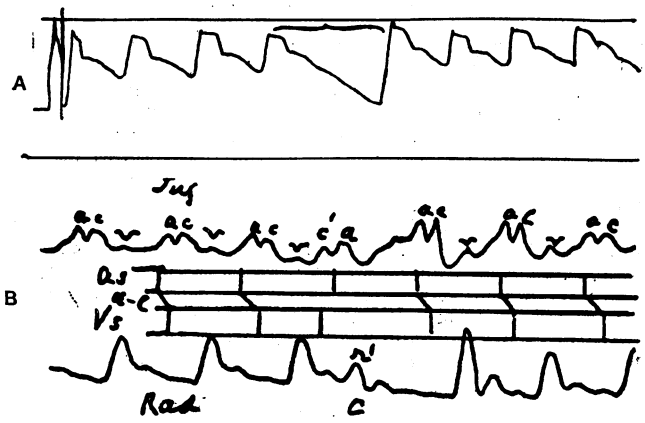

Fig. 3.

A.—Note the length of the intermission equals that of two pulse periods.

B.—The undisturbed auricular rhythm during occurrence of a ventricular extra-systole.

In a majority of cases of extra-systole of ventricular origin a compensatory pause is present, but there are instances in which this does not occur, as is shown in the following diagram:

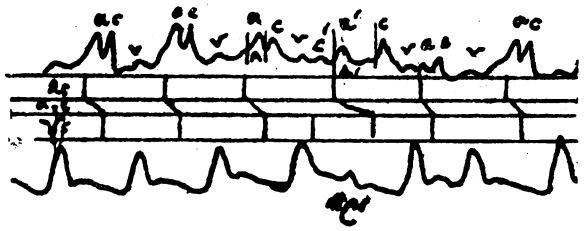

Fig. 4.

From this tracing it will be plainly seen that a ventricular extra-systole has occurred which is not followed by a compensatory pause. The inference to be drawn from this is that an extra ventricular beat has been interpolated at such a point in diastole that the refractory phase produced by it has had time to pass off before the next normally occurring stimulus from the auricle has appeared. The physiological beat is therefore not dropped, but has taken place as usual, though not in quite the normal position. It is really a little late, but there is no true compensatory pause. The delay is owing to the exhaustion and gradual return to full conductivity of the auriculo-ventricular bundle after a stimulus has passed over it, which results in a retardation in the passage of the next normal stimulus and a delay in the appearance of the corresponding ventricular contraction. The bearing of this upon the maintenance of the ventricular rhythm has already been referred to.

I have so far only spoken of extra-systoles of ventricular origin, but they may also arise in the auricle. In this case the compensatory period is generally too short, a phenomenon usually explained by assuming that since the primitive cardiac tissue is more or less fused with the auricle, the stimulus arising at some point in the auricular wall passes backwards and discharges from the great veins the stimulus material in process of formation but not yet at the point of effectiveness. The time occupied by its subsequent re-formation will, of course, be equal to a normal pulse period and the compensatory phase will consequently be absent (see Fig. 5).

The causation of the stimulus which gives rise to the interpolated systole is not definitely known, but it is held to arise in the heart muscle itself and not in the nervous system. With regard to the exact point of origin of the abnormal stimulus, there are two possibilities: either it is in some part of the remains of the primitive cardiac tube, as represented by the sino-auricular and auriculo-ventricular nodes and auriculo-ventricular bundle, or it may arise in any part of the cardiac muscle tissue as a whole.

Between these two possibilities it is difficult to decide, but experimentally it has been shown that, whereas the ventricle of the lower vertebrates when artificially stimulated responds by a single contraction, a series of contractions follows stimulation of the auriculo-ventricular ring, which in higher vertebrates remains as the auriculo-ventricular node and bundle, thus suggesting the greater excitability of these parts, and certain of Mackenzie's tracings are very suggestive of an origin in the primitive tissue. It has also been shown that the auriculo-ventricular bundle does not share in the atrophy or hypertrophy which may affect the rest of the cardiac tissue, the function of which appears to be chiefly contractile. In a case quoted by Mackenzie, in which heart-block had existed for eighteen years, the bundle below the seat of the lesion was in every way healthy, from which it may be inferred that this structure has some function other than that of mere conduction.

Moreover, similarity in histological formation, and the part played by the sinus node in the initiation of stimuli to contraction, strongly suggest the probability that such stimuli may arise, under certain conditions, in any part of the primitive tissue.

Whilst there is much to be said for the view that the origin of abnormal stimuli must be looked for in the auriculo-ventricular bundle or node, as well as in the sinus node, rather than in the heart walls as a whole, the question must remain for the present unsettled, and be left for further research to determine.

Clinically extra-systoles are met with under the most diverse conditions, and four classes of persons have been described by Wenckebach in whom this form of irregularity may be present.

The first class are those in whom nothing can be found in any way to account for the irregularity, and whose lives are in no way affected by their occurrence.

The second are those in whom the irregularity is due to lesions or conditions in other organs, and may be toxic in origin; for instance, diseases of the gastro-intestinal tract or the presence of parasites in the intestinal canal. They are found more commonly in slowly than in rapidly acting hearts, and generally disappear during fever. This may be regarded as a favourable sign. On the other hand, where the pulse irregularity persists or comes on for the first time during the febrile attack, the prognosis is serious, more especially in the young.

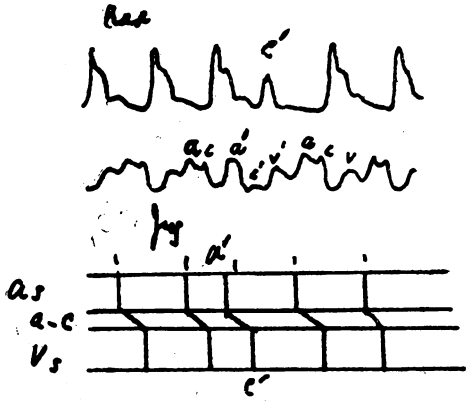

Fig. 5.—The auricular extra-systole is indicated at a' and the corresponding ventricular contraction at c'. The short lines above the lower diagram show the times of appearance of the normal stimulus at the great viens.

The third class comprises the aged. The cause is uncertain, but may be due to increasing irritability of the cardiac tissue with advancing life.

The fourth class are those who suffer from definite organic lesion. In these, again, the value of mere pulse irregularity is not certainly defined, and is a doubtful guide to the extent of the damage; conversely, where the damage is known to be great—as in the fatty degeneration of the organ in pernicious anaemia—irregularity does not necessarily occur. At present our knowledge of the clinical aspect of pulse irregularity is limited, and each case must be judged on its merits. The extra-systole is a method of explaining certain forms of arrhythmia, but its precise clinical significance is not fully determined,

and each case must be viewed from the widest possible standpoint, every factor being taken into consideration in arriving at a conclusion.

One value of the investigation of irregularity from the clinical point of view is the light it throws upon the action of drugs, and upon those cases of cardiac failure in which, *post mortem*, nothing at all commensurate with the clinical signs can be made out. Tawara, who examined the hearts in 112 cases dead of cardiac failure, found no microscopical change sufficient to account for the clinical signs observed. It is possible that in future defect in one or other of the four great properties of cardiac muscle may explain much that is at present obscure.

I must now pass on to consider defects in conductivity. These are best seen in cases of so-called Stokes-Adams disease, or, as it has been termed, "heart-block."

Experimental investigation by clamping the auriculo-ventricular bundle in the mammalian heart has demonstrated the possibility of so far varying the conductivity of the bundle by gradually increasing the pressure of the clamp, that only every third, fourth, or fifth stimulus can be transmitted from auricle to ventricle, and a ratio between the auricular and ventricular beats of 2 to 1, 3 to 1, 4 to 1, and so on may be formed in this way, and a condition of partial heart-block produced. When the pressure of the clamp is further increased, so as to destroy entirely the conductivity of the auriculo-ventricular bundle, no stimuli will pass, and a state of complete heart-block results, in which auricle and ventricle beat each with its own rhythm and in complete dissociation. You will perceive that here a state of things is experimentally induced which closely resembles the symptom-complex of Stokes-Adams disease met with in the human subject, so far as concerns its cardiac aspect. In this disease, of which the main symptoms are a persistent abnormal slowing of the heart's action with the occurrence of cerebral symptoms of a more or less epileptiform nature, it is comparatively easy to demonstrate, by simultaneous tracings from the jugular vein and apex or radial pulse, that either complete or partial heart-block is present, or that dissociation of the auriculo-ventricular rhythm has taken place.

Whilst the cerebral attacks seem to be associated with further paroxysmal slowing of the radial pulse, and an association between cerebral anaemia and epileptiform convulsions has been experimentally demonstrated, it must be remembered that heart-block, even with complete dissociation of auricles and ventricles, is not necessarily accompanied by the Stokes-Adams phenomena. In some cases disease of the cerebral vessels has been found which, it has been suggested, may wholly explain the symptoms, and to which the heart rhythm might be secondary. But against this the weighty objection has been urged that, if the bradycardia were a question of inhibition, the auricles should share in it, which tracings made simultaneously over the jugular veins and radial pulse and apex show not to be the case. Whilst the question cannot be regarded as settled, it is quite possible that the complete syndrome depends upon the combination of two factors:

1. Prolonged or permanent slowing of the heart beat with paroxysmal attacks of extreme bradycardia; and
2. Diseased cerebral arteries.

So long as the ventricle is able to deliver a sufficient blood supply to the brain symptoms may be absent, but failure to do this will more easily result where two factors, each tending in the same direction, are present. Alone either may be insufficient, but together moderate defect in the one is intensified by the condition of the other, and vice versa.

In most of the hearts of cases exhibiting this syndrome disease of the auriculo-ventricular bundle has been found of the nature of a sclerosis, gumma, or disturbance of vascular origin. In some cases no gross lesion has been present. Experimentally defects in conduction occur in states of asphyxia, and may be removed by the administration of oxygen.

With regard to the action of drugs in defects of conductivity, partial or complete block may be produced by digitalin, whilst atropine quickens the auricular rhythm but leaves that of the ventricle unchanged. It seems probable, however, that when the auriculo-ventricular bundle is intact, digitalis may cause a partial block but never produces a complete block, but where the bundle is defective digitalis may convert a partial block into a complete one.

In the following case the patient has been taking small doses of trinitrine with apparent benefit, in so far as his pulse-rate has gone up to and remains at 40, and no fits have occurred since this drug was administered.

J. L. came to my out-patient department on April 22nd, 1907, complaining of fits and dyspnoea.

History.

Up to two years ago, when the fits began, he was quite well. He first noticed giddiness and staggering on exertion, which would disappear on resting. Shortly afterwards he had a fit whilst undressing at night, and found himself lying on the floor, having lost consciousness completely while the fit lasted. Since then he has had a great number of fits—he says "hundreds"—which have always been of the same kind. Consciousness has always been lost, and at times he has hurt himself in falling. No tongue-biting or urination has been noticed. The fits are said to last about one minute only, and the fall "seems to bring him round." He has noticed shortness of breath on exertion for two years. There has never been noticeable swelling of the legs. There has been no pain, paroxysmal dyspnoea, or flatulence. During the fits no twitchings or convulsions occur. He states that the fits are liable to be brought on by exertion. The previous history is negative. Specific disease is denied, and there has been no gout or rheumatism. The family history is also negative.

Present Condition.

Pulse 24, regular, no extra-systoles. Wave well sustained. Tension between beats rather low. Vessel not markedly thickened, but slightly tortuous and inelastic. The beat ceases at 180 mm. of mercury.

Heart.—Apex beat in fifth space in nipple line, action weak; the first sound is replaced by a rough systolic murmur, which is well transmitted to the axilla. The apical second sound is reduplicated, but otherwise unimportant. The systolic murmur is well heard all over the precordium, but with greatest intensity at the apex. Over the cardiac base there is nothing noteworthy. Nothing suggestive of an auricular sound can be heard. There is a flickering venous pulsation to be seen in the neck, but its rhythm cannot be determined by the naked eye. Dullness begins about three fingerbreadths to the right of the sternum and extends to the nipple on the left.

The lungs show some emphysema and bronchitis only. Nothing abnormal is found in the abdomen. The urine is normal.

The tracings shown in Fig. 6 (p. 874) were obtained from this case. In the tracing A the upper figure is from the apex and the lower from the jugular vein. It will be seen that the auricular beats marked *a* are much more numerous than the ventricular systoles marked *v*, also that small waves *a* appear in the apical tracings during the diastolic periods, due to auricular impulses transmitted to the apex. They will be found to correspond in time exactly with the auricular pulsations in the lower figure.

In the tracing B the upper figure was obtained from the jugular vein and the lower from the radial artery. Here *a* is placed over the auricular beats, *c* over the carotid, and *r* over the radial pulse beats. This tracing shows clearly that the auricular pulsations are three to four times as numerous as the radial, and when the tracings A and B are compared with the tracing C it is equally clear that the slow pulse is due to a true bradycardia, and is not produced by the occurrence of weak ventricular systoles which have not reached the wrist. It is further worth noting that both auricular and ventricular contractions take place in a perfectly regular manner, and that there is well-marked dissociation between the auricular and ventricular pulsations.

The failure of the ventricle to respond to each stimulus from the auricle is explained by Erlanger in this way: Owing to defect in the auriculo-ventricular bundle the excitation stimulus is reduced to a point just insufficient to produce a ventricular contraction, but the following prolonged rest allows the irritability of the ventricle so far to increase that a stimulus of the same reduced strength is now sufficient to provoke a contraction, and thus the various rhythms can be explained.

As already mentioned, it is probable that the ventricles in mammals possess rhythmicity. When, therefore, the intervals between the efficient excitation waves become longer than the intervals between the spontaneous beats of the ventricle, the latter becomes more rhythmical than the efficient excitation waves, and consequently beats with its own independent rhythm; thus there results complete dissociation between auricle and ventricle, although the

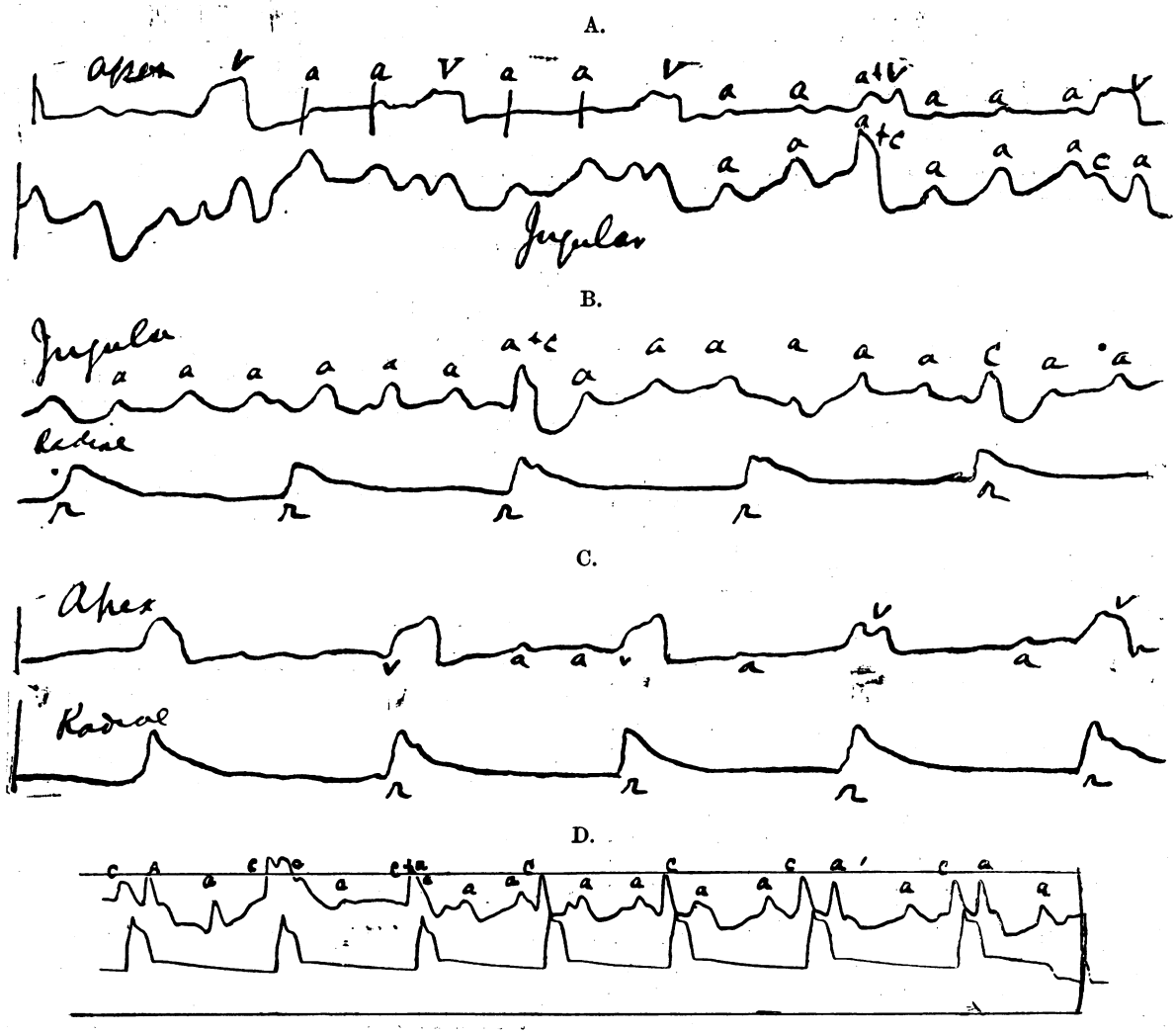

FIG. 6.

block to the passage of the auricular stimuli may not be absolutely complete.

DEFECTS OF CONTRACTILITY.

It can be shown by experiment that contractility may be made to vary independently of other functions of the heart muscle, and having in view the law of maximal contraction, according to which every beat of the heart is the best of which it is capable at the moment and is independent of the strength of the stimulus, it is a comparatively easy matter to measure the force of the contraction by noting its size. But when this method is applied to man it becomes a question how far it is to be relied upon. Systole abolishes all the functions of the heart, and among them that of contractility. The contractility increases during the diastolic pause, and consequently largely depends upon the duration of the diastolic interval, as has already been shown in connexion with the post-compensatory systole.

The best example of defect of contractility is, perhaps, that of the "pulsus alternans," in which large and small beats are found in alternation in the radial pulse. When the contractility is lowered the heart's contraction is made more quickly than normally—that is, it does not last so long, the consequence of which is that the duration of the pause following the smaller contraction is longer than that following the larger one. But a longer pause will be followed by a larger and, therefore, longer enduring contraction by reason of the length of the rest allowed by the longer pause, and, conversely, a large contraction by lasting longer will be followed by a smaller beat. If, therefore, when the contractility is lowered the duration of a pause should be lengthened from any cause, while the cardiac rhythm remains otherwise unchanged, a pulsus alternans will be the result.

This form of pulse must not be confused with the bigeminal pulse resulting from extra-systoles. In these there is generally a compensatory pause which does not occur in the pulsus alternans; moreover, the pulsus alternans may continue unchanged for long periods of time, whereas extra-systoles do not.

Concerning the functions of stimulus production and excitability, little that is really definite is known. Presumably, irregularity in stimulus production, or change in excitability, will be accompanied by forms of cardiac and pulse arrhythmia. Variations in the intensity of the stimulus might be neutralized by alteration in excitability—as, for instance, a feeble stimulus which normally was insufficient to evoke a contraction might become effective when the excitability was raised, and, conversely, defect in excitability would be overcome by increase in the strength of the stimulus; but for the present these possibilities are mainly in the speculative stage, and where two functions are so closely associated it is probable that great difficulty surrounds their elucidation.

Such, then, is in brief the bearing of the myogenic theory upon cardiac arrhythmia. The work upon the subject is recent, and much requires to be done before the many lacunae are filled up, and before it can be clinically applied with confidence; at the same time, it has explained much that was formerly obscure, and holds out still more promise for the future. In the light of this theory, and of the knowledge gained by investigation of the events taking place in a cardiac cycle by means of rightly understood graphic records of the venous and arterial pulses, much of the chapter upon cardiac pathology requires to be rewritten; when this has been done, it will be possible to apply with greater confidence and certainty such methods of treatment as may be available based upon more accurate knowledge.