Observations

ON

THE INCEPTION OF THE RHYTHM OF THE HEART BY THE VENTRICLE

AS THE CAUSE OF CONTINUOUS IRREGULARITY OF THE HEART.

BY JAMES MACKENZIE, M.D.,

Burnley.

THIS paper seeks to explain that most puzzling of all the forms of irregularity of the heart, where the heart is never regular in its action, where seldom or never two beats of the same character follow one another. Many names have been applied to this condition, such as delirium cordis, the mitral pulse, pulsus irregularis perpetuus, a heart irregular through loss of vagus control, etc. As the result of a study of a large number of cases where a jugular pulse was present I have been able to establish the fact that the cause of the irregularity is due to the rhythm of the heart proceeding from the ventricles, and not, as normally, from the great veins as they debouch into the auricles. I am also convinced that in all other cases of continued irregularity where there is no jugular pulse to explain matters (as in old people and others who suffer from attacks of palpitation with irregular action of the heart) the same cause is at work, not only because of similarity in type, but because in such people there is a great tendency to extra-systole of the ventricles—a condition which, as will be seen, often precedes the continuous irregularity.

INTERPRETATION OF A TRACING OF THE JUGULAR PULSE.

The interpretation of a tracing of an irregular pulse even with the aid of a simultaneous tracing of the apex beat is largely a matter of inference, depending on the resemblance of the tracing to the results obtained by experiment. Interpretation on such grounds is liable to error and satisfactory conclusions cannot always be reached. With the simultaneous record of the jugular pulse and arterial pulse the nature of many irregular pulses can be demonstrated. In a tracing of the pulsation in the jugular even where there is but a slight regurgitation back from the right heart, a series of waves is obtained whose significance, as a rule, can be easily recognized. In Fig. 1 the radial pulse and the jugular are taken together. The space *E* in this, as in all the other tracings, represents the time of ventricular systole during which the semilunar valves are open and when the effects of the ventricular systole would appear in any tracing, therefore a little later in the radial than in the jugular or carotid tracings.

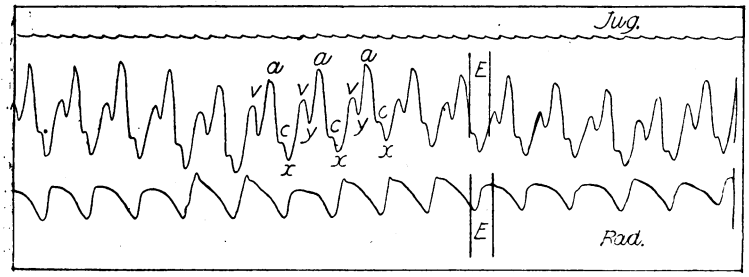

Fig. 1.

Immediately in front of this period (*E*) in Fig. 1 is a wave, *a*, which from its situation in the cardiac cycle can only be due to the systole of the auricle. Immediately after the auricular wave, *a*, is a great fall, *x*, which is due to the diastolic expansion of the auricle emptying the jugular of its contents. It is important to notice particularly this fall, *x*, as its presence indicates with certainty that the auricle must have contracted immediately before it. Sometimes it happens that the wave sent back by the auricle is so small, or that the vein is so distended, that the auricular wave cannot be detected in the tracing as a separate wave, and the systole of the auricle might be overlooked, were it not for the sudden emptying of the vein due to the auricular diastole, for, if the auricle dilates, it must have contracted. Further, when the rhythm of the pulse is altered, the presence of this fall is decisive in interpreting the nature of the changes that are taking place. Following on the fall *x* in Fig. 1, there is a rise in the tracing *v*, which is due to regurgitation by the last portion of the ventricular systole. Its appearance at this late period of the ventricular systole is due to the fact that the regurgitant blood sent back by the ventricle cannot appear in the jugular until the dilating auricle has been filled. If that has happened before the end of the ventricular systole, then the overflow appears in the jugular as a distinct wave *v*. It is to be borne in mind that the tricuspid orifice readily becomes incompetent with dilatation of the right heart. One can form an idea of the degree of engorgement from the earlier appearance in the period of ventricular systole (*E*) of the wave *v*. If the auricle does not completely empty itself, then it will fill the sooner, and the wave *v* appear the earlier. Thus Fig. 2 was taken from the same patient a week after I took Fig. 1. In this patient there was marked aortic and mitral regurgitation due to valvular disease, compensation gradually failed, and there was increased dilatation of the heart. The increased engorgement is reflected in the jugular tracing by the increase in the size of the wave *v*. The patient died the day after Fig. 2 was taken, and the necropsy verified the diagnosis, the auricles being enlarged and greatly distended with blood. If the patient had lived longer, the wave *a* and the depression *x* would have disappeared, and there would have been but one large wave synchronous with and due to the systole of the right ventricle, and one large fall *y*, synchronous with and due to the diastole of the right ventricle, as in Fig. 3. That is to say, the auricle would have ceased to contract (and therefore ceased to dilate), in fact, auricular paralysis from over-distension. I have observed such changes take place in the life of a patient, and for years the venous pulse would preserve the character of Fig. 3, and never show the slightest symptom of auricular contraction, even when the heart action became very slow as in Fig. 4. Fig. 4 is from the same patient as gave Fig. 3. The shading under Fig. 4 is the representation of the sounds and murmurs heard at the time this tracing was taken. There was a systolic mitral murmur with the double heart beats, and during the long pause a long murmur occupying nearly the whole of diastole, stopping short, however, of the succeeding first sounds—that is to say, there was no presystolic murmur of the crescendo type. The diagnosis of the heart condition from the

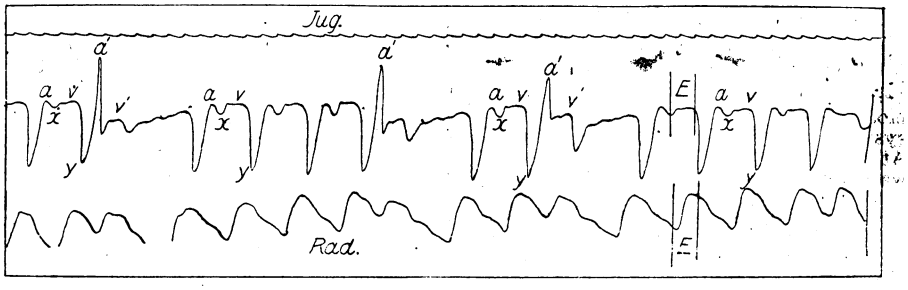

Fig. 2.

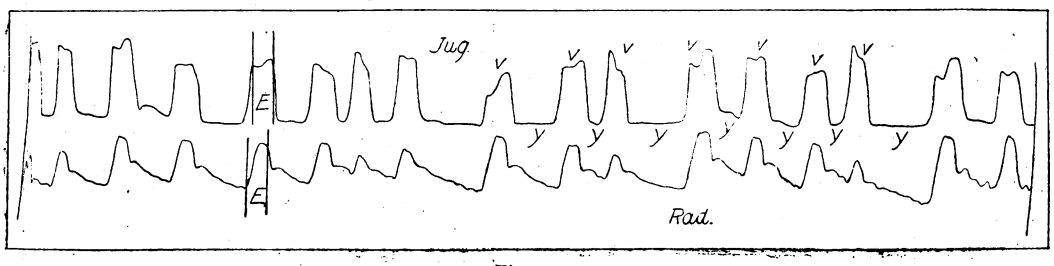

Fig. 3.

nature of the murmurs and jugular pulse was mitral incompetence and mitral stenosis, with paralysis of the auricle from over-distension. This was verified at the necropsy, where, in addition, there was found tricuspid stenosis.

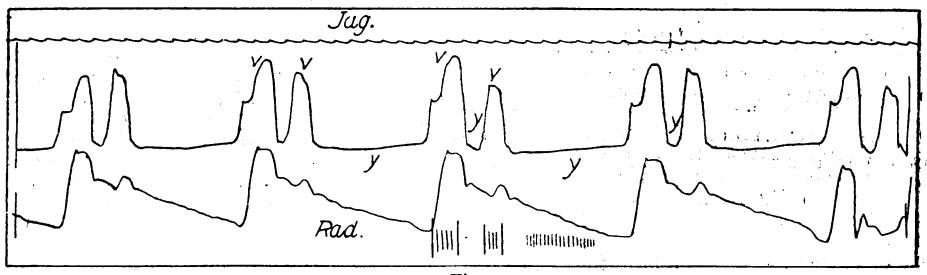

Fig. 4.

The Extra Systole.

In the pulse of many people an intermission is sometimes detected, or a small beat is felt, occurring earlier than usual, and followed by a pause longer than usual. If a tracing of the radial be taken, there will often be found a small pulse wave, even when the finger failed to recognize the beat at the irregular periods, as *B* and *C*, Fig. 5. At other times there may be no sign of a beat having taken place, as at *A* and *D*, Fig. 5. It can, however, be inferred with confidence that during *A* and *D* a systole did occur, but it was not of sufficient strength to propagate a wave into the radial artery from the fact that the period, including the long pause and the preceding pulse beat, equals two ordinary pulse periods, for a reason to be explained later. If the jugular tracing be studied, a very interesting state of affairs is disclosed. The wave *a* is due to the auricular systole, as was shown in describing Fig. 1. At the time of the irregular period, *C*, the wave *a'* occurs before the usual time occupied by the other auricular waves *a*. Following this is the carotid wave *c'*—a little later than *c* usually follows *a*, but at a time corresponding with the small beat in the radial. The wave *v'* is due to the extra systole of the right ventricle appearing late, in accordance with the interpretation given in describing Fig. 1. Here we have an instance of an extra systole set up in the auricle and followed by an extra systole of the ventricles. In passing it may be pointed out that here the carotid wave *c'* follows the wave *a'* at an interval slightly longer than occurs in the normal rhythm of the heart. The reason for this may involve the question whether the conducting power of the muscular fibres between the auricle and ventricle had completely recovered from their exhaustion after the preceding ventricular systole, but need not further concern us in discussing the subject in hand. If now we examine the irregular period *B*, we find a very different state of affairs. The auricular wave *a'* appears at the usual time after the preceding *a*, and is succeeded at the usual time by the following wave *a*. We can say, therefore, that here the auricle pursued its wonted rhythm; but it is not followed by the wave *c* due to the carotid pulse. If we look at the radial we find that the small wave there occurs slightly before its time; in fact, it occurs at the same time as the auricular wave *a'*—in other words, there is an extra systole which has only affected the ventricles, and has occurred at the same time as the auricular systole.

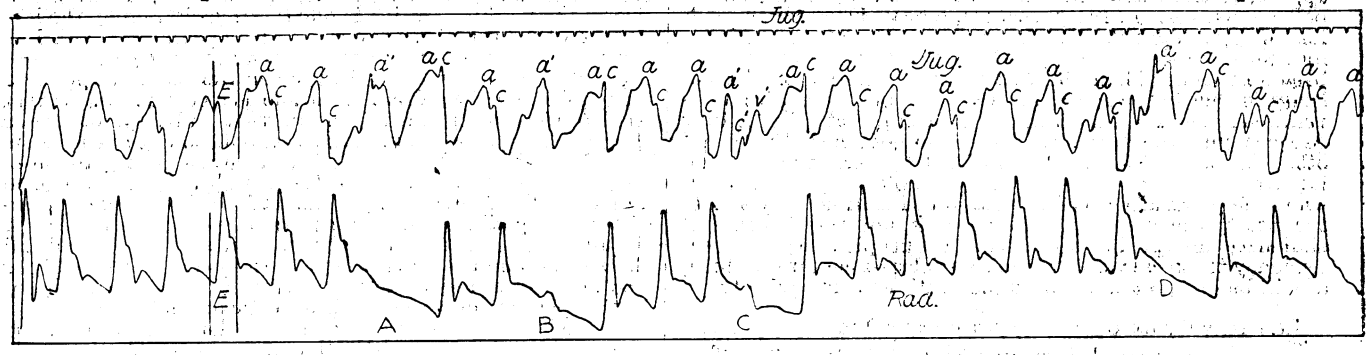

Fig. 5.

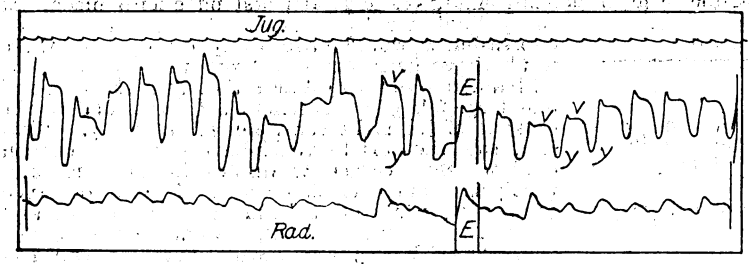

Fig. 6.

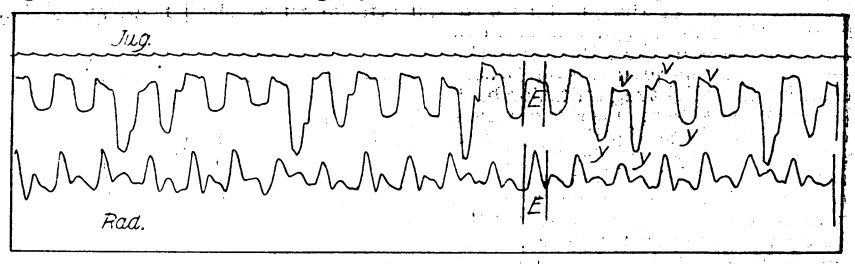

Fig. 7.

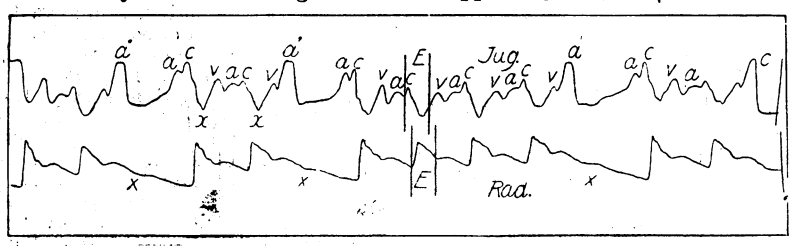

Fig. 8.

When we come to examine the character of the irregular periods *A* and *D* another change is found. Although there is no radial pulse beat, it can be inferred with certainty that an extra systole occurred from the fact that the time occupied by the long pause and preceding pulse corresponds to the time occupied by two regular pulse periods. Another evidence is found in the change in the jugular pulse. In the irregular periods *A* and *D* there is a large broad wave *a'* very different from the waves *a'* in the irregular periods *B* and *C*. The explanation that I give is, that here the ventricle has

begun to contract before the auricle and that the whole of that wave is really ventricular. In favour of this explanation is the fact of the absence of the radial pulse, which indicates that the extra systole of the ventricle has occurred earlier after the preceding beat than in the irregular periods *B* and *C*. The presence or absence of a wave in the radial pulse due to an extra systole depends among other things upon the time of the occurrence of the extra systole; if it occurs near the usual time for the appearance of the normal pulse, it will be large (see Fig. 21), if it occurs just shortly after the preceding ventricular systole it may be small or absent. The fact that it is present in the periods *B* and *C* and absent in *A* and *D* points to this being the likely explanation. I find in numerous tracings from this patient taken at different times that when there is a broad wave in the jugular pulse at the time of the extra systole, there is an absence of a corresponding pulse beat in the radial, or the pulse beat is very small; whereas, when there is a large or well-marked radial beat, it has occurred late in the diastolic period, and the auricular wave *a'* is of normal duration (Fig. 21). This fact is of importance in considering the changes that take place in the veins during a sudden change in the heart's rhythm, and will be referred to again.

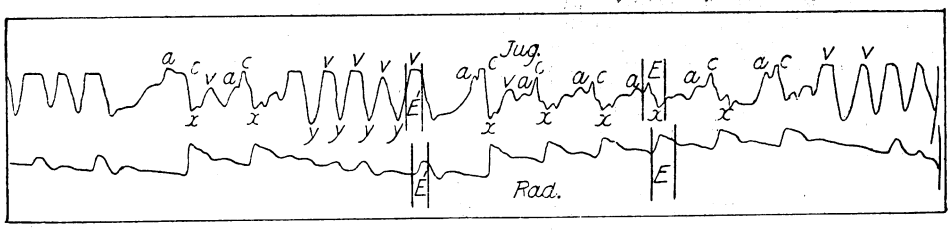

Fig. 9.

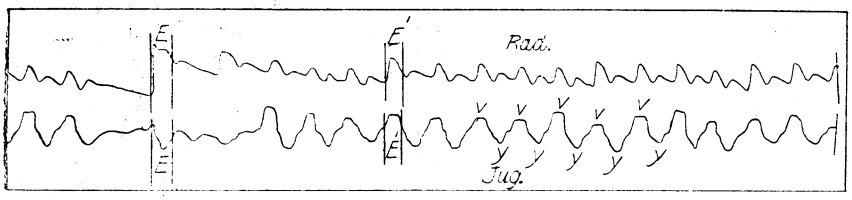

Fig 10.

THE NATURE OF THE EXTRA SYSTOLE.

There is not space here to note the investigations of many observers, both experimental and clinical, to explain what the extra systole is. The salient facts are as follows: During the contraction of the muscular fibres of the heart and for a short time after, the heart muscle cannot be stimulated to further contraction—that is, it is "refractory"; hence it is impossible to set up a tetanic contraction of the heart muscle. The stimulus that normally produces contraction originates periodically at the mouths of the great veins and passes from thence downwards to auricle and ventricle. The auricle or ventricle may be stimulated to contract by some extra stimulation before the normal periodic stimulus is due, so that there arises a premature contraction of the auricle or ventricle—an extra systole. When the periodic stimulation arrives from the great veins it finds the auricle or ventricle in a refractory state; they do not respond but remain quiescent until the next periodic physiological stimulus arrives. Hence, as Engelmann has explained, there is a long pause—the so-called compensatory pause—after the extra systole, and hence also the fact that the period occupied by the extra systole and the preceding beat equals that of two pulse periods, as shown in Figs. 2 and 5.

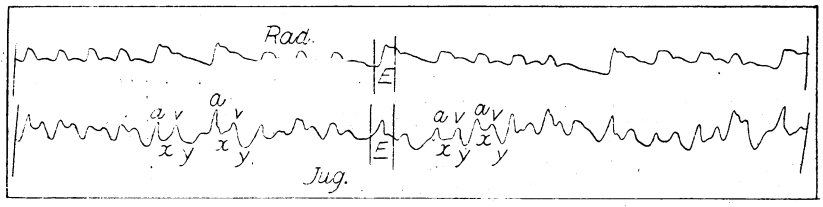

Fig. 11.

From what has been said in regard to the extra systole, it will be seen that the starting point of the heart's contraction may not necessarily be at the great veins, as they debouch into the auricles, but the stimulus may originate in either the auricle or ventricle. So far, the evidence has only shown that the ventricle can start the rhythm in isolated beats. The question arises—Can it be shown to affect the heart in a series of beats? I have proof that the ventricle can take on the inception of the rhythm of the heart, and when it does so the rhythm is almost invariably irregular—the heart staggers, so to speak. The chief exception to the heart being irregular is when the rate is very frequent—110 to the minute or over. It might seem that then the stimulus to contraction affects the heart as soon as the refractory stage has passed off.

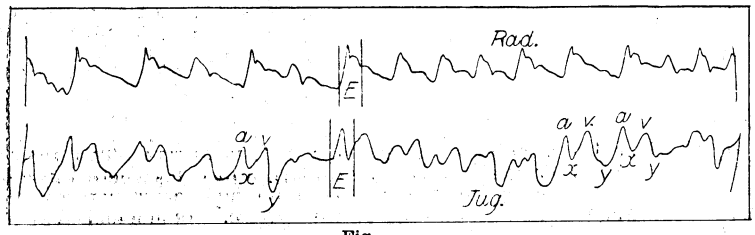

Fig. 12.

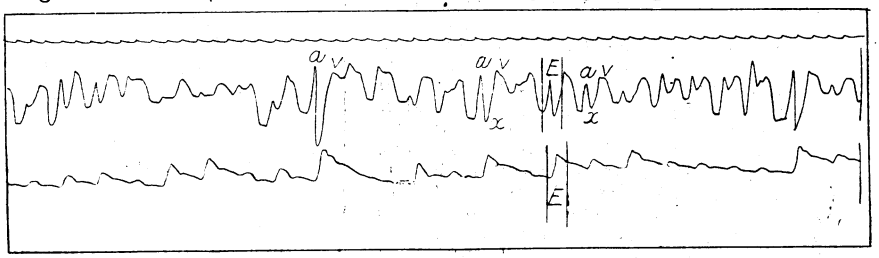

Fig. 13.

The cases I have so far studied can be grouped under three heads:

1. Those cases where there is auricular paralysis from over-distension secondary to disease of the mitral valves.
2. Those cases where there is no evidence of auricular action, but where the movements in the veins demonstrate that the rhythm of the heart is due to the ventricle.
3. Those cases where the auricle and ventricle can be demonstrated to contract simultaneously.

1. *Those Cases in which there is Auricular Paralysis from Over-distension Secondary to Disease of the Mitral Valves.*

This class has already been dealt with in

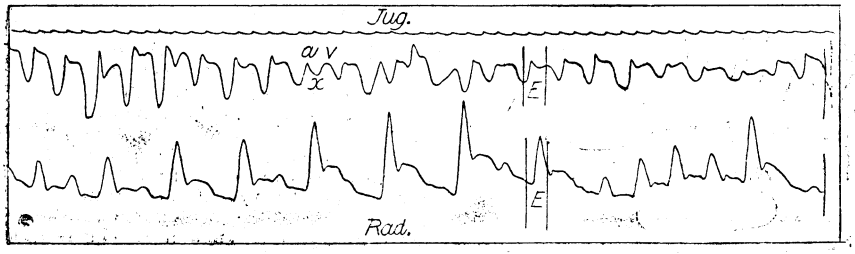

Fig. 14.

describing Figs. 3 and 4, which are good examples of the type of irregularity when the heart is acting slowly. This condition is easy of recognition if there is a jugular pulse and the movements recognized. As a rule the jugular pulse is present and is often of enormous size. In some instances I have watched the cases for six, seven, and eight years, and have taken tracings at intervals, and never found the pulse once regular after the paralysis of the auricle could be demonstrated, whilst previous to this the heart was regular in its action save for the rare occurrence of an extra systole. In Fig. 2 one can see that the heart is beginning to stagger from the presence of three premature systoles. For some months

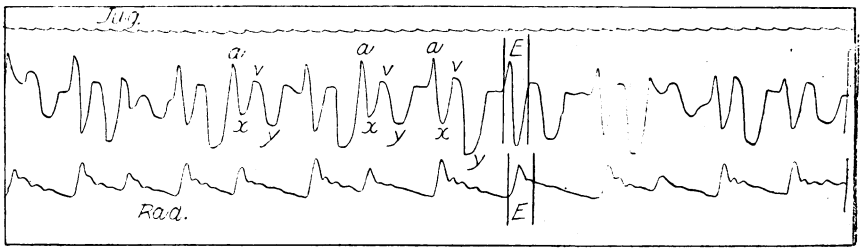

Fig. 15.

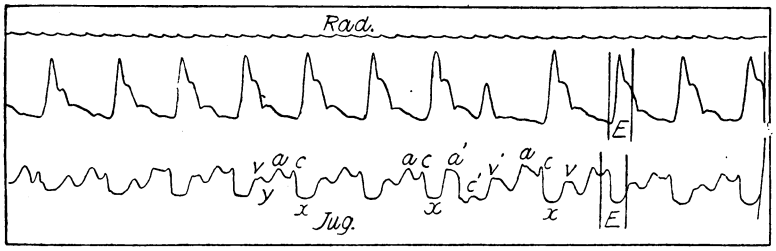

Fig. 16.

reviously I had taken numerous tracings from this patient and never once got an irregular beat. As I have already pointed out, the auricle was evidently enormously over-distended and almost paralysed. In this class of case there may be long periods of very rapid action when beats of the most varied size and duration may be present, as in Fig. 6. This is from an old-standing case of mitral disease with great enlargement of the heart, and is a good illustration of what is spoken of as the mitral pulse. It will be seen here that the wave in the jugular is synchronous with that of the radial, and the fall *y* corresponds in time with the ventricular diastole; in other words, there is a complete absence of any sign of the systole or diastole of the auricle.

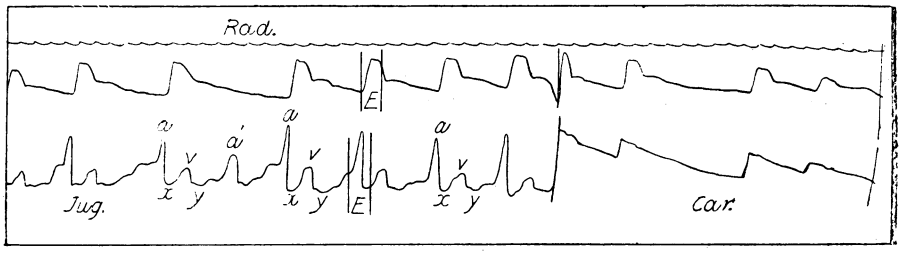

Fig. 17.

2. Those Cases in which there is no Evidence of Auricular Action during the Irregular Period, but where the Movements in the Veins demonstrate that the Rhythm of the Heart is due to the Ventricle.

This condition of heart irregularity is usually well and permanently established when the patient comes first under observation. There is then always a history of shortness of breath and attacks of weakness extending back for months or years. When once recognized, the condition persists usually to the end of life, and the fatal termination is probably due to the long-continued irregular action of the heart. Fig. 7 represents a typical tracing of this condition. There is here, as in the preceding class of cases, one large wave, *v*, synchronous with, and due to, the systole of the right ventricle, and one large depression, *y*, synchronous with, and due to, the diastole of the right ventricle. This was taken from a man aged 48 years, suffering from emphysema and bronchitis, and who had for many years suffered from attacks of weakness and breathlessness. He came under observation two months before he died. During the whole of this time the pulse was continuously irregular, though often slower than is shown in the tracing. In addition to the bronchitis and emphysema there was great enlargement of the heart (apex beat in the sixth interspace and 2 in. outside the nipple line), a systolic murmur at the apex, enlargement and pulsation of the liver, and extensive dropsy.

At the necropsy there was found emphysema of the upper part of the lungs, congestion of the bases, great dilatation of the left ventricle and auricle, the mitral valves being perfectly healthy, but the orifice permitted the passage of three fingers. The right ventricle was hypertrophied, the columnae carneae being two or three times thicker than those of the left ventricle; the tricuspid valves were healthy, but the orifice permitted the passage of four fingers. The auricles were very greatly dilated, with the walls everywhere thinned, except for a thick band that stretched across between the appendix and the cavity of the auricle. As one looked at the thin-walled auricle one could easily conceive its inability to contract, but what the meaning was of that thick hypertrophied band of muscular fibres I could not divine. If it did contract—and one cannot imagine it to hypertrophy without functionating — it must have done so at the same time as the ventricle, for there was no sign of independent contraction in the venous pulse.

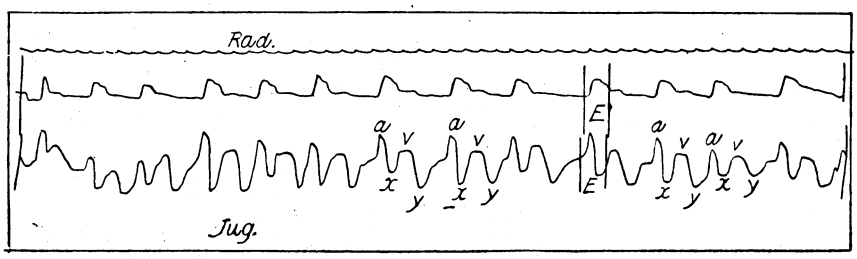

Fig. 18.

The manner in which this condition of permanent irregularity arises can be better illustrated by taking a less advanced instance, and I am fortunate in being able to give tracings from a case where the condition was only temporary, and where the gradual passage from the normal to the extremely irregular and rapid action of the heart was demonstrable. In this patient, a female aged 35, eight months pregnant with her second child, the heart was usually regular,

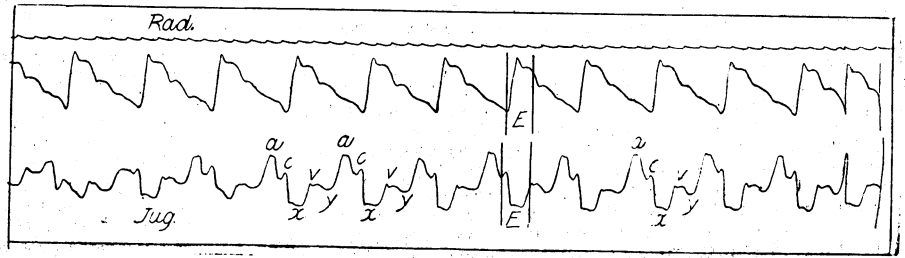

Fig. 19.

but she was subject to attacks of palpitation which distressed her greatly. I have taken numerous tracings of her pulse (radial and jugular) during these attacks. When the heart was beating regularly there was often no sign of the jugular pulse, and when present it was of very small size. When the palpitations began the heart became irregular and the veins of the neck speedily filled up with waves of blood sent back from the right heart, and good tracings were obtained of these waves. Fig. 8 shows one phase of the irregularity where the radial pulse shows intermissions at ×, after every few beats. This is due to extra systoles, as can be inferred from the fact that the long pause and the preceding pulse beat together equal the time occupied by two pulse periods. That it was a

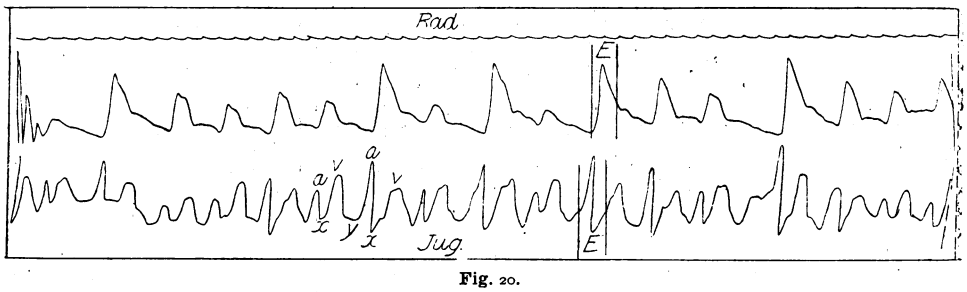

Fig. 20.

abnormal symptom was a pulse of continuous and varying irregularity. He had several attacks of extreme heart failure, with dropsy and oedema of the lungs, from which he made partial recoveries. Fig. 11 was taken during one such breakdown in November, 1895. Fig. 12 was taken in January, 1896, after he had made a partial recovery. In the latter tracing the pulse is slower, but still irregular. At no time after I first recognized the irregular pulse did I ever find it regular. In these two tracings the most striking and peculiar feature is the jugular pulse. Here there is no sign of an auricular wave at the usual period in the cardiac cycle, that is, before the period (*E*), nor is there a fall corresponding to the usual time of the auricular diastole, occupying the first portion of the period (*E*). During the ventricular systole (period *E*) the jugular pulse is divided into two by a depression of varying size. This fall in the tracing means that blood must have been absorbed from the veins during that time. It could not have been by the diastole of the ventricle, for the radial pulse shows the ventricle was in systole at that time. It must, therefore, have been due to the auricular diastole, and the preceding wave must have been due to the auricular systole, that in fact both chambers must have contracted at the same time. Such, indeed, is the only explanation I can suggest to explain the peculiar changes that have taken place in the rhythm of the heart. Although I was forced to this conclusion some years ago in the interpretation of the movements in the vein by applying rigidly the facts known to us of the possible causes, yet the interpretation seemed so incredible I dare not include these tracings and this interpretation in my book on the pulse, but waited till more convincing light could be thrown upon the subject. Quite recently I have had the opportunity of demonstrating these changes in a remarkable manner in another patient.

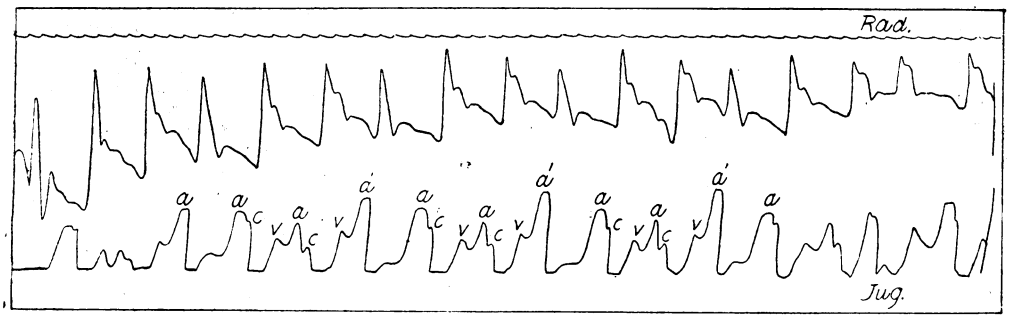

Fig. 21.

ventricular extra systole can be recognized from the jugular pulse by the fact that the auricle maintains its regular rhythm during this irregular period. The increased size of the auricular wave *a'* indicates that when it contracted the ventricle was in systole, hence a larger volume of blood was sent back by the auricle. In Fig. 9 we can observe the sudden change of the heart from the normal rhythm to one of varied frequency and strength. The normal pulse beats can be recognized always by the fact that during the period (*E*) of the ventricular systole there is always a well-marked fall *x* in the jugular tracings due to the auricular diastole. On the other hand the abnormal beats showed a rise *v*, during the ventricular systole (*E'*), indicating that the ventricle has taken on the inception of the rhythm. It will also be noted that when there is no sign of a pulse in the radial there is a well-marked beat in the jugular. In Fig. 10 we have a similar state of affairs, only that there is here but one regular pulse period (*E*), all the rest being abnormal in the sense that it is the ventricle that is giving the heart its rhythm.

The question arises what was the auricle doing during this abnormal rhythm? That it was not paralysed is evident in that it at once returns to work immediately the normal rhythm is resumed. It may be that this class of cases should not be grouped separately, some of the cases like those from whom Fig. 7 was obtained belonging to the first group, while those with irregularities shown in Figs. 9 and 11 should properly be included in the following group. Still as there is a difference in the character of the jugular pulse I provisionally include them in a separate group.

3. *Those Cases where the Auricle and Ventricle can be Demonstrated to Contract Simultaneously.*

As in the last group, the irregularity is usually permanently established when first met with, and the patient often gets gradually worse until he dies of failure of the heart. People may, indeed, go about for years with the heart acting in this disorderly manner, but they are always rather feeble, and there is a distinct limitation of the field of cardiac response. Figs 11 and 12 are from a man in comfortable circumstances who died of heart failure in July, 1897, aged 65 years. I had known him since 1879, and until the last few years of his life had looked upon him as a healthy, vigorous man. During the last few years of his life he suffered from weakness and shortness of breath on exertion. The only

The following case I give in some detail, as the information afforded by the jugular pulse is of the most interesting and instructive character.

The patient is a widow, nulliparous, aged 57. I have known her for over twenty years as a healthy, sober, and industrious person. She keeps a small grocer's shop, works hard, and is in very comfortable circumstances. I have attended her at rare intervals for slight ailments, as quinseys and mild attacks of influenza. The only abnormalities I have found present in her case are a pulsation in the veins of the neck, and a cantering rhythm of the heart sounds when the heart is regular, an occasional intermission of the pulse, and at certain times a systolic murmur heard at the apex and at midsternum. The feature that chiefly concerns us here is the occasional irregularity of the heart as observed in its effects on the radial and jugular pulses. Until three years ago this consisted in the occurrence of an extra systole, sometimes at very rare intervals at other times more frequently, as in Fig. 5, which was taken from this patient in July, 1893. As a rule, the extra systole was of ventricular origin, but in rare instances it was of auricular origin (*C*, Fig. 5 and Fig. 16). Since 1900 she has had occasional attacks of "palpitation," in one of which attacks I got tracings of the radial and jugular pulses that agree in all particulars with the tracings obtained during the attacks about to be described.

On October 2nd, 1903, she felt weak, exhausted, and a

distressing fluttering sensation within her chest, and I found her heart's action extremely irregular, and the tracings of the radial and the jugular were similar to those in Fig. 13. This attack of "palpitation," as she called it, lasted four or five hours. On October 19th she was again seized with a similar attack, which lasted the whole of the day. The tracings (Fig. 13) of the jugular and radial convey a very true picture of the degree of arrhythmia. Next day the pulse was quite regular, and the jugular pulse presented its usual normal character of an auricular wave before the ventricular systole, and an auricular depression during the ventricular systole, as in Fig. 19.

On October 22nd the heart again became irregular. Fig. 14 shows the jugular and radial pulse during this period. This attack continued without intermission until November 1st. From October 27th the heart acted much more slowly than in Fig. 14, but still there was a distinct absence of regularity in the rhythm of the heart, as shown in Fig. 15. This slow action of the heart, with the curiously doubled beat in the jugular during the period of ventricular systole, continued until November 1st, when the heart became quite regular, with the exception of an occasional extra systole, as in Fig. 16. On November 2nd the heart became irregular, as in Figs. 17 and 18. Towards the evening of the next day, however, the heart was again quite regular except for an occasional extra systole. On November 4th the heart became quite regular, as in Fig. 19, and no extra systole was detected. The patient gradually regained strength, the jugular pulse diminished in size and gradually disappeared, but could be recalled to a slight extent when the patient held her breath. The auricular systole always maintained its normal position in the cardiac cycle during this period. On November 14th she had another attack, and the same feature was again present in the jugular pulse (Fig. 20). This attack persisted until November 19th, when the pulse again became normal with the exception of the occasional occurrence of extra systole. In Fig. 21 the extra systoles occur after every second beat, giving the appearance of a regular irregularity. This did not continue long, the extra systole appearing at much rarer intervals.

The following interpretation of the jugular pulse during irregular action of the heart seems to me the only feasible one. If we take a tracing where the heart is acting slowly, as Figs. 15 and 18, we find that during the ventricular systole there is a great fall *x* in the jugular tracing. This implies that at that time some great fall in pressure had taken place in the chest, which emptied the veins of their contained blood. Only two factors could produce this, the diastole of the auricle or the diastole of the ventricle. As, however, it is shown that the ventricle is in systole, it can therefore only be due to the diastole of the auricle. In that case the wave *a*, preceding *x*, must have been caused by the systole of the auricle, because the presence of a diastolic period necessitates the previous occurrence of a systole. Therefore the wave *a*, though it occurs at the time of the ventricular systole, is in reality due to the auricle, and here the auricle and ventricle have contracted together. It can be shown by Fig. 19 that the auricular systole lasts about $\frac{1}{10}$ sec., and the ventricular systole nearly $\frac{1}{5}$ sec. When they contract together the former systole ceases much earlier than the latter, and so the contracting ventricle speedily fills up the dilating auricle and sends a wave (*v*, Figs. 15 and 18) back into the veins towards the end of its systole. After that it passes into diastole, and the vein again empties itself during the fall (*y*). The rise in the veins after *y*, when the pulse is slow, is due to stasis in the veins. After this the cycle of events is again resumed. When the heart beats rapidly the period of stasis is absent, and even the fall due to auricular diastole may not be evident, as in many of the beats in Fig. 14, so that the racing of the jugular pulse assumes the same character as in Figs. 6 and 7.

In analysing these tracings there are a great many novel and instructive points well worthy of consideration. It will be possible to deal here only with those that have an immediate connexion with the irregular rhythm. One of the most striking features is the position in the cardiac cycle of the auricular systole during regular and irregular action of the heart. When the heart beats regularly, as in Fig. 19, the auricular systole is always found to occupy the normal position in the cardiac cycle. Even with the somewhat frequent occurrence of extra systole, as in Fig. 21, the auricle maintains its rhythm. When, however, the heart acts irregularly the auricular wave in the venous pulse does not precede the ventricular systole. Not only is this the case in the rapid heart action, as shown in Figs. 13 and 14, but also when the heart is beating slowly, as in Figs. 15, 18, and 20. In other words, when the rhythm of the heart originates at the auricles, or at the auricular mouths of the veins, it is always regular. When, however, the ventricle takes on the inception of the rhythm the heart is always irregular.

The explanation for this is to be sought for in the nature of the functions of the muscular fibres of the heart. Independent of any nervous connexion, as Engelmann has shown, the muscular fibres possess, amongst other attributes, the power of creating a stimulus and the power of conducting a stimulus from fibre to fibre. During a contraction of the muscular fibres these powers are for a time abolished, and their return takes place gradually during diastole. Normally the power of creating the stimulus arises with greater rapidity in the region where the great veins open into the auricle. From hence the muscular fibres convey the stimulus from auricle to ventricle. One great characteristic of the stimulus-production of the veins is its periodicity—that is to say, the production of the stimulus is carried on with great regularity, and hence the continuous and equal rhythm in the regular pulse and the regular succession of events in Fig. 19. In such tracings as Fig. 21 the regular appearance of the wave *a* and *a'* shows that the stimulus has maintained its periodic character and the irregularity is limited to the ventricles. But as all muscular fibres possess this power of originating the stimulus, certain abnormal conditions may affect the muscle fibres of the ventricle, so that the stimulus-production arises prematurely, not only at rare intervals producing the occasional extra systole, but continuously so that the origin of the stimulus-production is in the ventricle, and not in the veins. Such, indeed, is the conclusion drawn from the study of these tracings, and from their study it can be asserted that when the ventricle gives the rhythm to the heart movement it is an irregular one—the only exception being, so far as my observations have gone, when the heart beats with great rapidity as in paroxysmal tachycardia, and for periods of rapid regularity like those represented in Fig. 10. Not only can one demonstrate in these tracings the power of creating the stimulus by the ventricle, but one can also show that the conduction of the stimulus takes place in a manner the reverse of what happens in the normal rhythm.

If one measures the time in Fig. 16, from the beginning of the auricular wave *a* to the appearance of the carotid wave *c*, it is found to be approximately $\frac{2}{10}$ sec. (The movements of the time marker represent $\frac{1}{5}$ sec.) This period we know is taken up by two events, first, the contraction of the auricle, and secondly, the presphygmic interval of the ventricular systole. The wave of contraction normally passes on without stop from the auricle to the ventricle, so that we know that the top of the wave (*a*) indicates the time when the wave of contraction passes from the auricle to the ventricle. The time therefore from the top of the wave (*a*) to the appearance of the carotid pulse (*c*) represents the time during which the ventricle is contracting before it opens the aortic valves (what has been called the presphygmic interval). It will be found that the auricular systole lasts $\frac{1}{10}$ sec. and the presphygmic interval also $\frac{1}{10}$ sec. In Fig. 17 the radial and jugular pulses were taken simultaneously in the first part of the tracing and the radial and carotid in the latter part of the tracing. It will be found on careful measurement that the wave (*a*) appears in the jugular vein exactly at the same time as the carotid pulse. We know from what has been said above that the presphygmic interval occupies $\frac{1}{10}$ sec., so therefore we can infer that $\frac{1}{10}$ sec. before the wave (*a*) due to the auricular systole appears the ventricle had been contracting, and that it takes $\frac{1}{10}$ sec. from the beginning of the ventricular systole for the conduction of the stimulus back to the auricle. It may here be stated that there is no presphygmic interval in the auricular contraction, as the auricle has to overcome no resistance in the pressure of the veins compared with what the ventricle has to overcome in the aorta and pulmonary artery.

EXPERIMENTAL OBSERVATIONS.

There is no lack of observations in experiments on animals confirming the above interpretation. Gaskell says, "The ease with which a reversal of the beats of the heart can be observed is well exemplified in the hearts of the skate and tortoise, and in both cases it is clearly seen that the only factor requisite is to start a rhythm with a rate quicker than that of the natural sinus rhythm; that as might be expected the heart beat starts from the place where the rate of the spontaneous contractions is quickest."....."Further Mac

William has shown that in the mammalian, just as in the cold-blooded heart a reversal of the sequence takes place when the ventricle is made to beat at a quicker rate than the great veins and auricles." More recently H. E. Hering has been able to induce ventricular extra systoles by raising the pressure within the ventricle. In these experiments he registered at the same time the movements of the four chambers of the heart and the variation of the carotid pressure. In one experiment on a rabbit he shows a continued series of contractions where the starting point of the contraction was in the left ventricle. In another experiment the heart spontaneously took on a different rhythm where the starting point was also from the left ventricle. But not only did the ventricle start contracting independently, but it also caused the auricle to contract and the time that the auricular contraction followed the ventricular practically corresponded to the time that the ventricular contraction followed the auricular when the rhythm was normal.

CONCLUSIONS.

From the foregoing considerations I think it may be considered demonstrated that the ventricle can take on the inception of the rhythm, and that when it does so the heart beats irregularly. It may further be presumed that whenever there is a continuous irregularity as distinct from occasional or rhythmic irregularity, the ventricle is giving the rhythm. Exceptions to this are rare, as in such peculiar rhythmical irregularity as is shown in Fig. 4 (due in all probability to digitalis), and in the extremely rapid action of the heart as is shown in Fig. 10. I am inclined to believe from the evidence I have at hand that in paroxysmal tachycardia the ventricle also takes on the inception of the rhythm.

The cause of the continuous irregularity is to be sought for in the exalted irritability of the heart muscle and not in the stimulation of the heart through the nervous system. So far as I know no experiment has demonstrated that stimulation of a cardiac nerve can produce a continuous cardiac rhythm where the contraction begins with the ventricle. Hering's experiments were carried out in hearts isolated from their nervous connexions. Further, Hering's experiments demonstrate that raising of the ventricular pressure can produce directly the premature contraction of the ventricle. I have had on several occasions the opportunity of observing in predisposed hearts—that is, hearts which give rise occasionally to extra systole—a continuous irregularity persists for several hours when the heart had been subjected to violent and unwonted exertion. As most cases that come under observation have this condition well and permanently established, and accompanied with considerable enlargement of the heart and symptoms of inefficient cardiac action, one has been inclined to attribute the continuous irregularity to the dilatation of the heart.

As I have had several opportunities of noting the beginning of the symptoms associated with attacks of continuous irregularity, I am convinced that the dilatation and ineffective action of the heart is secondary to the irregularity. When the irregularity begins there is uneasiness in the chest, a shortness of breath on exertion, a sense of weakness, which increases as the attack continues. Coincident with these symptoms are certain very definite changes in the circulation. The radial pulse becomes larger and very compressible, the veins in the neck become greatly engorged, the face becomes dusky, and the lips blue. The area of the heart's dullness extends beyond the right of the sternum, and the superficial structures of the chest wall become tender to pressure in the peculiar manner I have found so frequently associated with dilatation of the heart. These symptoms, which follow definitely the beginning of an attack of continuous irregularity, are those we meet with in advanced cases, and it is for that reason I suggest that it is the irregularity that is the cause of the inefficient heart action, and not the dilatation that causes the irregularity.

Treatment should therefore be directed to lessen the irritability of the heart muscle and to protect the heart from being exposed to causes that excite it to increased activity. While drugs such as opium, and the bromides do help in reducing the irritability of the heart muscle, in my experience no remedy equals prolonged rest in bed.

EXPLANATION OF TRACINGS.

In all these tracings the jugular and radial pulses are taken simultaneously. The space *E* represents the time of ventricular systole when the semilunar valves are open. In the radial tracings it is always calculated from the beginning of the upstroke of the radial pulse, and is always a little later than the same period in the jugular tracing, which is calculated from the time of the appearance of the carotid pulse.

The wave *a* in the tracings from the jugular pulse is due to the systole of the right auricle sending back into the jugular a wave of blood; the fall *x* is due to the emptying of the jugular by the diastole of the right auricle, and the fall *y* is due to the emptying of the jugular by the diastole of the right ventricle.

The wave *v* is due to the systole of the right ventricle. When it appears late in the period of ventricular systole, it is because it has to fill the dilating auricle before it can appear in the veins. When it occupies the whole time of the ventricular systole, the auricle then fails to contract and to dilate, or does so at an unusual period in the cardiac cycle.

The wave *c* is due to the carotid impact. It is not really from the jugular but is due to the fact that the carotid is so close to the jugular that in most cases a tracing of the jugular cannot be taken without the effect of the carotid pulse being perceptible in the tracing. It is, however, of considerable value in determining the time of the several events in the jugular pulse.

The time marked on each figure represents one-fifth of a second.

Fig. 1.—The various events in the jugular pulse show the normal time relationship. The jugular pulse is of the auricular type. Mrs. S., October 17th, 1903.

Fig. 2.—From the same patient as gave Fig. 1, taken a week later. The fall *x*, which was the greatest depression in Fig. 1 is here very small, and the fall *y*, which was small in Fig. 1, is the greater. The wave *v* fills up nearly the whole space occupied by the fall *x* in Fig. 1, and indicates greatly-increased engorgement of the right heart. The radial pulse is irregular, due to the frequent occurrence of extra-systoles. The large wave *a'* in the jugular is due to the regular occurrence of the auricular systole, and its increased size is due to the fact that when it contracts the ventricle is already in systole, and hence a large wave is sent back into the veins. From these evidences one recognizes that the extra systoles are of ventricular origin. This jugular pulse is intermediate between the auricular type as in Fig. 1, and the ventricular type as in Fig. 3. Mrs. S., October 24th, 1903.

Fig. 3.—The jugular pulse is of the ventricular type—that is, there is no sign of auricular contraction, and the wave *v*, occupies the whole period of ventricular systole and the depression *y* the whole time of ventricular diastole. Mrs. A., March 25th, 1902.

Fig. 4.—From the same patient as Fig. 3, after taking digitalis for three weeks. The pulse counted at the wrist is 30 per minute, the small wave in the radial being imperceptible. During the long pauses there is no sign of auricular action. The shading underneath represents the time of the murmurs, that is, a systolic murmur during the large and small beats, and a long murmur during the diastolic period. Mrs. A., April 12th, 1902.

Fig. 5.—The jugular pulse is of the auricular type. The tracing shows four extra systoles, three of them due to the ventricle (A, B, and D), and one due to the auricle (C). Mrs. T., July 25th, 1893.

Fig. 6.—The jugular pulse is of the "ventricular" type. From a case of advanced mitral disease. Mrs. F., October 18th, 1903.

Fig. 7.—The jugular pulse is of the "ventricular" type. From an old-standing case of bronchitis and emphysema, with irregular action of the heart. T. W., November 30th, 1903.

Fig. 8.—During irregular action of the heart. The auricle preserves its rhythm, there being a large wave (*a'*) during the premature contraction of the ventricles (compare Fig. 2).

Fig. 9.—Showing the rapid change of the jugular pulse from the auricular to the ventricular type. From the same patient as gave Figs. 8 and 10.

Fig. 10.—Showing only one beat of the auricular type (*E*), all the other beats being of the ventricular type (*E'*). From the same patient as gave Figs. 8 and 9.

Fig. 11.—The irregular rhythm is of ventricular origin, and the auricular wave (*a*) occurs during the ventricular systole (E). J. S., November 3rd, 1895.

Fig. 12.—Shows the same as Fig. 11, with a slower action of the heart. J. S., January 1st, 1896.

Fig. 13.—Rapid irregularity, with a well-marked fall *x* due to the auricular diastole during ventricular systole (*E*). Mrs. T., October 19th. 1903.

Fig. 14.—Rapid irregularity with the fall *x* only occasionally evident during ventricular systole (*E*). Mrs. T., October 22nd, 1903.

Fig. 15.—The heart is slower, but still irregular and the fall *x* is extremely well marked during the ventricular systole. Mrs. T., October 30th, 1903.

Fig. 16.—The normal rhythm of the heart has returned, and the normal sequence of events is shown by the jugular pulse. The occasional extra systole is of auricular origin. The wave *a'* is compounded of the ventricular wave *v* of the preceding systole and the premature auricular systole. This is inferred from the larger size of the wave and from the fall that succeeds it; *c'* is due to the carotid, and will be found to occur at the same time as the small pulse beat in the radial. This extra systole is of the same nature as *C* in Fig. 5. Mrs. T., October 10th, 1903.

Fig. 17.—In the latter part of this tracing the carotid is taken at the same time as the radial. The rhythm of the heart is irregular, and the wave *a* and the fall *x* show that the auricular systole and diastole occur during the ventricular systole. There is an occasional missed beat, and the wave *a'* shows at this time that the auricle contracted, but was neither succeeded nor accompanied by a ventricular contraction, as shown by the absence of waves in both radial and jugular tracings Mrs. T., October 21st, 1903.

Fig. 18 shows the same incidents as Fig. 15. Mrs. T., November 3rd, 1903.

Fig. 19 shows a perfectly normal pulse, with all the events in the cardiac cycle following one another in a normal manner. Mrs. T., November 4th, 1903.

Fig. 20.—Inferences drawn from the radial pulse would assuredly mislead in recognizing the cause of the irregularity, as here there seem normal pulse beats followed by extra systoles. The jugular tracing demonstrates the real nature of the irregular rhythm, which shows the occurrence of auricular systole (*a*) and diastole (*x*) during the ventricular systole (*E*). Mrs. T., November 14th, 1903.

Fig. 21 shows the frequent occurrence of extra systoles of ventricular origin, and gives the clue to the explanation of the whole series of irregularities from which the patient suffered. The explanation of the extra systoles and regular appearance of the auricular waves is the same as that given from Fig. 2. Mrs. T., November 19th, 1903.

ON SOME HAEMOGLOBIN INVESTIGATIONS IN CASES OF CHRONIC HEART DISEASE.

BY PROFESSOR THEODOR SCHOTT, M.D.,
Bad-Nauheim.

THE physical and dietetic treatment of chronic heart complaints in general, as well as the special treatment by baths and gymnastics, are so widespread nowadays that in the most recent literature the question is much less frequently asked whether these methods have a good effect on heart diseases, but rather in what way they act on the heart and body generally. It would lead us too far to repeat here the various opinions of the subject. The readers of the BRITISH MEDICAL JOURNAL would gain more information from the works of Bezly Thorne, Grainger Stewart, Lauder Brunton, Bowles, William Broadbent, Douglas Powell, and others.

In this short essay I only desire to set forth the results of a series of investigations which I undertook last summer with my assistant, Dr. Wolfson, for the special purpose of giving some support to my brother's opinion and my own that the combined treatment by baths and gymnastics has a tonic action not only on the heart but on the whole system. If also the patients should show when the cure is finished a more robust appearance, increased strength of muscle, especially displayed in greater ease in walking and climbing, better sleeping and improved appetite, etc., in short, greater bodily strength, yet it is not superfluous, but rather desirable to become acquainted with those clinical methods of investigation which make it possible for medical men living at a distance to get a clear idea whether such treatment is really a tonic. Besides the weighing of the patient during and after the period of treatment, my former researches, which were principally directed to the state of the heart and vessels, had included heart diagrams, sphygmograms, and blood-pressure tracings, while some other investigations, especially those relating to the composition of the blood, had not been carried out. The reason for not having made the last named was that for general purposes former methods of investigation left much to be desired; either too much time was needed for each separate investigation, or the mass of blood to be removed was so large that it must be taken direct from a vessel; in other words, such fuller investigations as would have extended over several weeks were scarcely applicable. Besides this, with most apparatus the blood had to be diluted or modified.

In the autumn of 1902 I obtained Dare's[1] haemoglobinometer. I must not allow myself to go more fully into a description of this instrument here, I only desire to describe its most essential merits; the first is that one drop of undiluted blood is sufficient for the investigation. For this purpose Dare has constructed a pipette so evenly that in an area of $\frac{1}{2}$ square centimetre one drop of blood is sufficient to fill the capillary space completely. In the apparatus there is a standard solution, the red colour of which corresponds to that of a solution of blood containing 100 per cent. haemoglobin. The drop of blood in the apparatus is compared with this standard solution. Candle light is used, so that the investigator is independent of daylight. The scale is on a revolving graduated wheel, and in that way, as the observer looks through a camera on to a white surface, the proportion of leucocytes in the blood need not be considered.

By following these directions every practitioner can in a short time learn how to make similar haemoglobin investigations within two minutes, and further so to diminish the sources of error that the differences between two observations and the readings of two observers amount eventually to only 1 to 2 per cent.

After sufficient practice on healthy people we began our observations on various forms of heart disease. Naturally care was taken that—besides the cautious manner of living which those suffering from heart disease must observe, with respect to the kind of nourishment, especially as to the nourishing power of the food taken—on the whole no essential change was made. In order to obtain correct comparisons the same hour was always chosen for the same patient for observation, but above all things care was taken that an equal time always elapsed after the last meal. Also it was made the rule that no examination of blood should take place during or after menstruation, or if it did take place it was not to be looked on as of any value as a comparison. The usual investigations of the heart with regard to heart diagrams and heart sounds, the sphygmogram, and above all successive observations with Gaertner's tonometer[2] always went hand in hand. In this way last summer 120 patients were examined under successive observations, and I permit myself to report here some of them very shortly and cursorily, reserving a more detailed description for a later time. In all cases except where otherwise stated the patients were subjected to combined treatment by baths and gymnastics.

CASE I. *Weak Heart, Dilatation of the Left Ventricle; Anaemia.*—Miss M., aged 18.

May 26th. Before the first bath, haemoglobin 69 per cent. (blood pressure 85 mm. Hg.).

June 3rd. After the sixth bath, haemoglobin 78 per cent.

June 23rd. After the eighteenth bath, haemoglobin 79 per cent.

July 9th. After the twenty-seventh bath, haemoglobin 81 per cent. (blood pressure 110 mm. Hg.).

The patient appeared much more robust after the cure, could walk for hours on level ground, and could climb hills without much difficulty. The treatment consisted of baths only.

CASE II. *Weakness of the Cardiac Muscle; Dilatation of both Ventricles; Anaemia.*—Countess St., aged 52 years.

May 25th. After the fourth bath, haemoglobin 60 per cent. (blood pressure 85 mm. Hg.).

July 13th. After the thirty-third bath, haemoglobin 75 per cent. (blood pressure 100 mm. Hg.).

This very feeble patient had taken iron at home for several weeks without result; during the first three weeks of her stay in Nauheim three bottles of haematicum (Glausch) were taken. The face was still pale at the end of the cure, though rather less so than at first, but the patient felt much stronger.

CASE III.—*Mitral Insufficiency after Acute Rheumatic Polyarthritis; Anaemia.*—Miss M. D., aged 15.

June 5th. Before the first bath, haemoglobin 53 per cent. (blood pressure 80 mm. Hg.).

June 14th. After the sixth bath, CO_2 (Spring 7.), haemoglobin 65 per cent.

June 23rd. After the twelfth bath, CO_2 (Spring 7.), haemoglobin 74 per cent.

July 3rd After the nineteenth bath, CO_2 (Spring 7.), haemoglobin 79 per cent.

July 15th. After the twenty-second bath (third effervescent bath), haemoglobin 81 per cent.

July 30th. After the twenty-seventh bath (eighth effervescent bath), haemoglobin 79 per cent. (blood pressure 105 mm. Hg.).

The patient took no kind of medicine, either for the heart or for anaemia. On leaving she felt completely recovered, and only complained of slight palpitation after climbing stairs and hills. Within the first fortnight she gained in weight 3 lb., and at the end of her stay 9 lb.

CASE IV. *Aortic Insufficiency after Acute Rheumatic Polyarthritis.*—Mr. R. F., aged 14.

June 2nd. Before the first bath, haemoglobin 55 per cent. (blood pressure 105 mm. Hg.).

July 7th. After the twenty-fourth bath (of which nine were sprudel baths), haemoglobin 78 per cent. (blood pressure 118 mm. Hg.).

The young man. who looked anaemic before, looked blooming after the treatment.

CASE V. *Mitral Stenosis.*—Miss H., aged 18.

June 9th. Before the first bath, haemoglobin 68 per cent. (blood pressure 100 mm. Hg.).

June 15th. After the sixth bath, haemoglobin 80 per cent.

June 28th. After the fourteenth bath, haemoglobin 80 per cent.

July 4th. After the sixteenth bath (one sprudel bath, Spring 7), haemoglobin 83 to 84 per cent. (blood pressure 120 mm. Hg.).

The remaining observations of haemoglobin and blood pressure all gave the last-mentioned result. Altogether twenty-four baths were taken.

CASE VI. *Mitral Stenosis and Insufficiency.*—Miss A., aged 23. Whether the condition was congenital or acquired in the first year of life could not be determined.

July 2nd. Before the first bath, haemoglobin 64 per cent. (blood pressure 85 mm. Hg.).

August 3rd. Before the first sprudel bath (Spring 7), haemoglobin 76 per cent.

September 15th. After the thirtieth bath (of which sixteen were sprudel baths), haemoglobin 81 per cent. (blood pressure 120 mm. Hg.).

The patient who had suffered much in youth from heart disease, and