A CASE IN WHICH THE VAGUS INFLUENCED THE FORM OF THE VENTRICULAR COMPLEX OF THE ELECTROCARDIOGRAM *

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BUNDLE-BRANCH BLOCK

In 1910, Eppinger and Rothberger[1] published electrocardiographic tracings illustrating the effects of dividing the right and left branches of the His bundle on the ventricular complex. Eppinger and Stoerk,[2] later in the same year, were able to report two clinical cases of bundle-branch block in which the diagnoses, made with the aid of electrocardiograms, were confirmed at section. Since that time similar cases have been reported by a large number of authors. Carter,[3] in his report of twenty-two cases, concludes that the ventricular complexes seen in cases of bundle-branch block exhibit, in contrast to normal ventricular complexes, the following characteristics. They are diphasic rather than polyphasic and are composed of (1) a primary deflection or QRS group, whose amplitude is greater than that of the normal ventricular complex and whose duration is longer, being one-tenth second or more; and (2) of a secondary deflection in the opposite direction from the first, an exaggerated T. The primary deflection usually comprises at least one-third of the entire ventricular complex and is often notched. These abnormal complexes, although produced by supraventricular stimuli, resemble very closely the electrical complexes produced by ventricular extrasystoles. When the block is in the right branch of the His bundle, as happens most often (twenty-one of Carter's twenty-two cases were of this type), the initial deflection is upward in Lead 1 and downward in Lead 3; and when the block is in the left branch the direction of the initial deflection in these leads is reversed. The fact that the right branch is so much more frequently affected than the left is probably due to its greater length

* Submitted for publication Aug. 2, 1915.

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1. Eppinger, H., and Rothberger, J.: Ueber die Folgen der Durchschneidung der Tawaraschen Schenkel der Reizleitungssystem, Ztsch. f. klin. Med., 1910, lxx, 1.

2. Eppinger, H., and Stoerk, O.: Zur Klinik des Elektrokardiogramms, Ztschr. f. klin. Med., 1910, lxxi, 157.

3. Carter, E. D.: Clinical Observations on Defective Conduction in the Branches of the Auriculo-ventricular Bundle, THE ARCHIVES INT. MED., 1914 xiii, 803.

before division. In most of the cases reported the bundle-branch block has been permanent, so that it has been impossible to compare the abnormal complexes with the normal complexes of the same case. In one of Carter's patients, however, the block was transient and such a comparison could be made, and in two of Mathewson's[4] patients transitions from the normal to the abnormal complexes were recorded electrocardiographically. In one of the patients reported by the latter, right and left bundle-branch block alternated irregularly with the normal mechanism.

EFFECT OF VAGI ON THE VENTRICULAR ELECTROCARDIOGRAM IN ANIMALS

Since the original experiments of Eppinger and Rothberger, abnormal ventricular complexes similar in form to those obtained by cutting one branch of the A-V bundle have been observed under a variety of experimental conditions. They may arise in at least three ways. First, they occur when there is a block in one of the branches of the His bundle even when the ventricles are responding to supraventricular stimuli. Second, they are found when heterogenetic beats or extrasystoles arise within the ventricles below the bifurcation of the A-V bundle. Third, they may occur when homogenetic beats arise in the branches of the His bundle. There is a possibility, of course, that the latter may originate within the walls of the ventricles outside the specialized tissues, but since it seems likely that homogenetic beats rarely if ever arise outside of the specialized tissues in the auricles, it is reasonable to suppose that the same is true of the ventricles.

Abnormal ventricular complexes of this type have occasionally been associated with stimulation of the vagi in animals. If heart block be produced experimentally by mechanical destruction of the A-V bundle, the ventricular complexes of the resulting idioventricular rhythm are always of the normal form. When, however, A-V dissociation is produced in animals by strong stimulation of the vagi, the ventricular complexes are often abnormal and typically diphasic. This was pointed out by Einthoven[5] as early as 1906 and similar observations have been made by Kahn[6] and by Kraus and Nicolai.[7] In such cases the ventricular contractions are of the homogenetic type since they always occur rhythmically and at a slow rate. We must assume,

4. Mathewson, G. D.: Lesions of the Branches of the A-V Bundle, Heart, 1912-1913, iv, 385.

5. Einthoven, W.: Le Telecardiogramme, Arch. internat. de physiol., 1906, iv, 132.

6. Kahn, R. H.: Beiträge zur Kenntnis des Elektrokardiogramms, Arch. f. d. ges. Physiol., 1909, cxxvi, 197.

7. Kraus, F., and Nicolai, G. F.: Das Elektrokardiogramm des gesunden und kranken Menschen, Leipzig, 1910.

therefore, that the vagus stimulation influenced the idioventricular electrocardiogram in these instances either because it altered the location of the idioventricular pace-maker or produced bundle-branch block.

Whether or not vagus stimulation may alter the form of the ventricular complexes when the ventricles are responding to supraventricular stimuli has been much discussed. In 1909, Hering[8] recorded abnormal ventricular complexes during vagus stimulation in a dog; and each ventricular beat was preceded by an auricular beat, as shown by the electrocardiogram and the suspension curve. Hering was of the opinion that these ventricular beats were supraventricular in origin, an interpretation which has been questioned by others.[9] If Hering's view is the correct one, we must assume a bundle-branch block to account for the form of the ventricular complexes. A similar assumption would be necessary to explain the abnormal ventricular complexes obtained by Kraus and Nicolai[7] by stimulating the auricles during complete vagus inhibition.

Rothberger and Winterberg[10] found that stimulation of either accelerator nerve during vagus inhibition almost always gave rise to rhythmic and typically diphasic ventricular complexes. In some of their figures[10] (Fig. 1c p. 466) each abnormal ventricular complex is preceded by an auricular contraction. It seems to us possible that in these instances the abnormal ventricular beats were responses to supraventricular stimuli. If this be true we would again be dealing with an abnormal spread of a supraventricular stimulus over the ventricles as a result of nervous influences. The effect of vagus stimulation was complicated in these experiments, however, by the effect of accelerator stimulation, and, moreover, the type of ventricular complex depended on which accelerator was stimulated, so that conclusions as to the relationship of the vagi to the abnormal complexes are difficult to draw. A summary of the experimental evidence shows that although it is possible that vagus stimulation produced bundle-branch block in animals in certain instances, unquestionable examples of this phenomenon are lacking.

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8. Hering, H. E.: Experimentelle Studien am Saügetieren über das Elektrokardiogramm, Arch. f. d. ges. Physiol., 1909, cxxvii, 155.

9. Kahn, R. H.: Das Elektrokardiogramm, Ergebn. d. Physiol., 1914, xiv, 1. Samojloff, A.: Weitere Beiträge zur Elektrophysiologie des Herzens, Arch. f. d. ges. Physiol., 1910, cxxxv, 417. Rothberger, J., and Winterberg, H.: Ueber die Beziehung der Herznerven zur Form des Elektrokardiogramms, Arch. f. d. ges. Physiol., 1910, cxxxv, 506.

10. Rothberger, J., and Winterberg, H.: Ueber die experimentelle Erzeugung extrasystolischer ventrikulärer Tachycardie durch Acceleransreizung, Arch. f. d. ges. Physiol., 1911, cxlii, 461.

So far as we know, no clinical case has as yet been reported in which stimulation of the vagus produced diphasic ventricular complexes. In the following case vagus stimulation produced such complexes although the ventricles were responding to supraventricular stimuli. The case also presents other unusual features. The history and examination of the patient have been recorded in another place[11] (Case 2).

BUNDLE-BRANCH BLOCK PRODUCED BY VAGUS STIMULATION

This patient showed at times no less than four separate and distinct rhythms. Rhythm 1, the normal rhythm, is illustrated in Figures 1, 2 and 3. The electrocardiograms have a normal outline in Leads 1 and 2, while in Lead 3 the QRS group of the ventricular complex is one of those bizarre forms occasionally recorded in this lead even in people with normal hearts.[12] T is partially inverted in Lead 3. The P-R interval of the normal rhythm is about 0.17 second and the a-c interval about 0.20 second.

The second rhythm observed, which may be called Rhythm 2, is illustrated in Figures 4, 5 and 6. Both the auricular and the ventricular complexes are abnormal. P is upright in Lead 1 and inverted in Leads 2 and 3. The P-R interval is reduced to about 0.10 second and the a-c interval to about 0.15 second. The reduction of the As-Vs interval with the inversion of P in Leads 2 and 3 indicates that the site of origin of this rhythm was in the neighborhood of the A-V node. The fact that the P-R interval is only slightly reduced means that the pace-maker was in the upper part of this region.

The ventricular complexes of Rhythm 2 are abnormal in all leads. In contrast to those of the normal rhythm they exhibit the following characteristics: they are typically diphasic rather than polyphasic. The initial deflection is of greater amplitude and of longer duration, its duration being 0.10 second or more. The final deflection extends in the opposite direction from the initial deflection in all leads and is exaggerated especially in Leads 2 and 3. These characteristics indicate that the abnormality of the ventricular complexes of Rhythm 2 is due to bundle-branch block. The fact that the initial deflection is upright in Lead 1 and inverted in Lead 3 indicates that the right branch of the His bundle is the one affected. Rhythm 2 may therefore be described as an atrioventricular rhythm with right bundle-branch block.

The relationship between the normal rhythm and Rhythm 2 was as follows. During the first few days that the patient was under

11. Wilson, Frank N.: Three Cases Showing Changes in the Location of the Cardiac Pace-Maker, Associated with Respiration, THE ARCHIVES INT. MED., 1915, xvi, 86.

12. Lewis, T.: Clinical Electrocardiography, London, 1912.

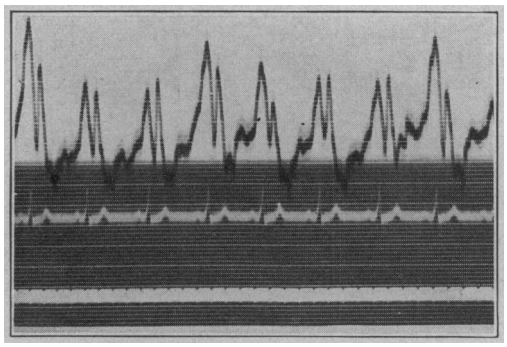

Fig. 1.—In this, as in the figures which follow, an ordinate of 1 cm. is equal to 1 millivolt. Rhythm 1, Lead 1. P-R interval, 0.17 second; a-c interval, 0.20 second. Duration of QRS group, 0.08 second. Heart rate 75.

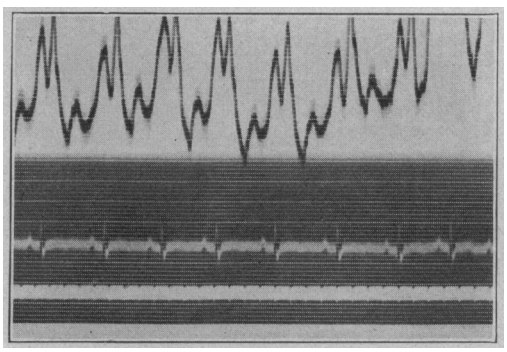

Fig. 2.—Rhythm 1, Lead 2. The P-R and a-c intervals are the same as in the previous figure. Duration of QRS group, 0.08 second. Heart rate 75.

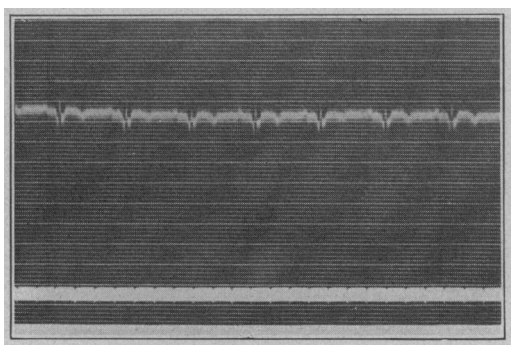

Fig. 3.—Rhythm 1, Lead 3. The P-R interval is the same as in Figure 1. Duration of QRS group, 0.10 second. The QRS group is bizarre and T is partially inverted.

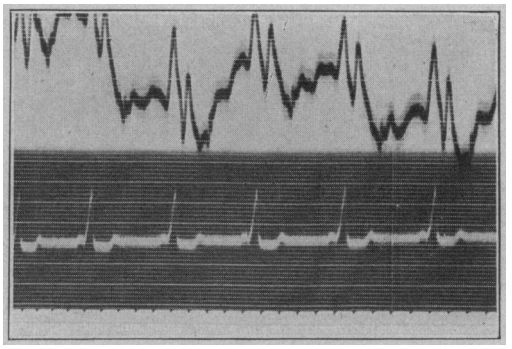

Fig. 4.—Rhythm 2, Lead 1. P-R interval, 0.10 second, a-c interval 0.15 second. Duration of QRS group, 0.10 second. The amplitude of R′ is greater than that of R in Figure 1. T is inverted. P is upright. Heart rate 66.

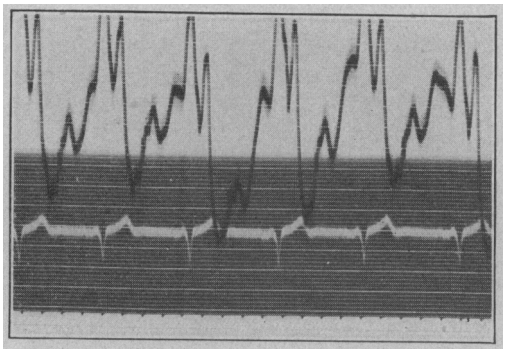

Fig. 5.—Rhythm 2, Lead 2. The P-R and a-c intervals are the same as in Figure 4. Duration of QRS group, 0.10 second. The ventricular complex is diphasic and T is exaggerated. P is inverted. Heart rate 62.

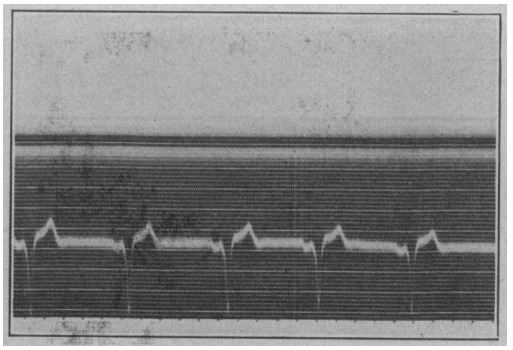

Fig. 6.—Rhythm 2, Lead 3. The P-R interval is the same as in Figure 4. Duration of QRS group, 0.13 second. The amplitude of the initial deflection is greater than that of the initial deflection of the ventricular complexes in Figure 3. T is exaggerated. P is inverted. Heart rate 62.

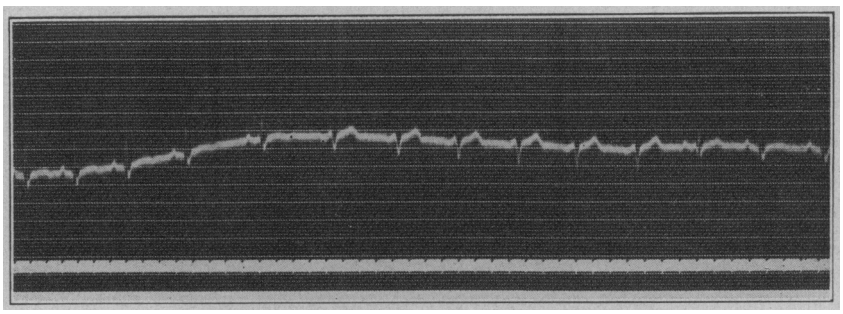

Fig. 7.—Lead 2. Rhythm 1 converted into Rhythm 2 by moderately deep respiration. Note the transitional ventricular complex at the end of the abnormal rhythm.

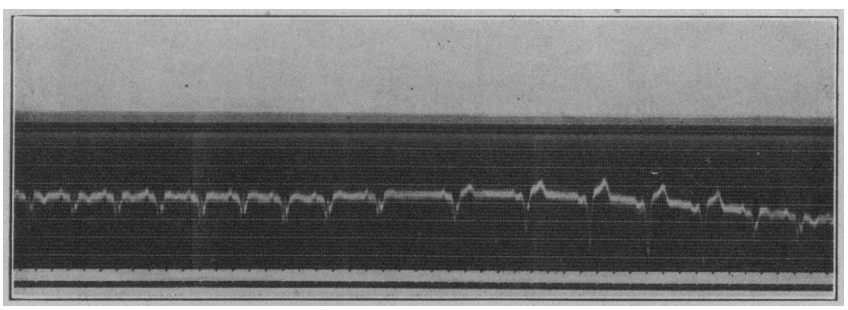

Fig. 8.—Lead 3. Rhythm 1 converted into Rhythm 2 by deep respiration. At the beginning of the abnormal rhythm the change in the form of P is sudden while the change in the form of the ventricular complex is gradual.

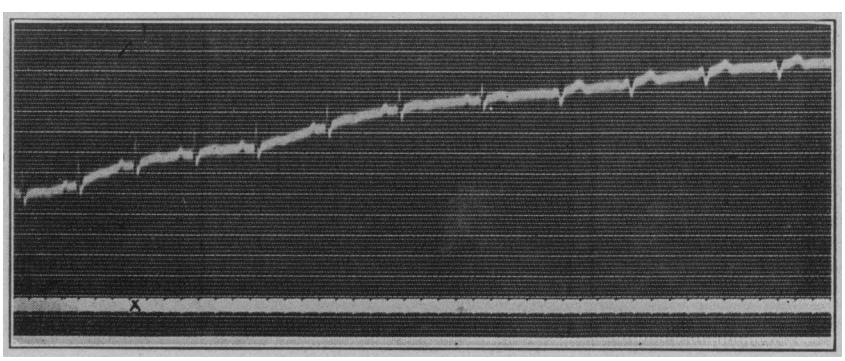

Fig. 9.—Lead 2, Rhythm 1 converted into Rhythm 2 by pressure on the right vagus. Pressure was begun at x and continued until the abnormal rhythm appeared.

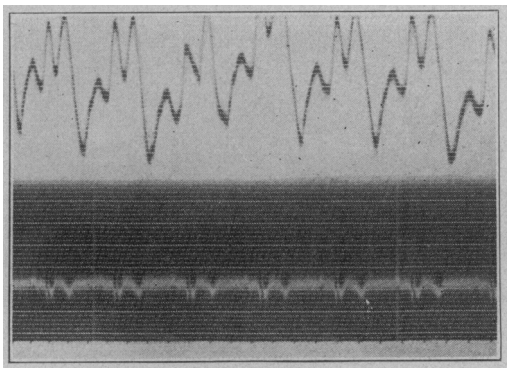

Fig. 10.—Rhythm 1, Lead 3. Taken at the end of an atropin experiment. Rhythm 2 was constantly present before the atropin was given.

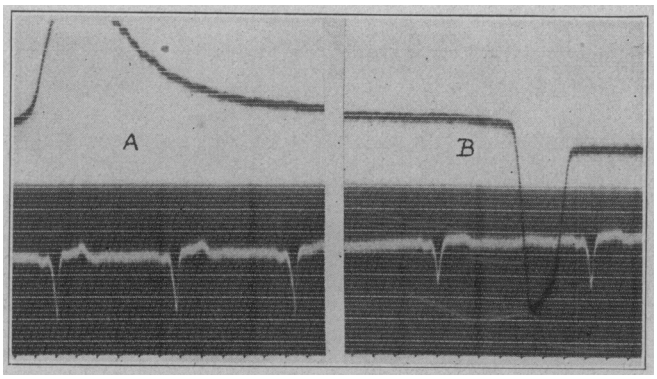

Fig. 11.—Rhythm 2, Lead 3. Rhythm 2 slowed by right vagus pressure; A, beginning, and B end of a period of right vagus pressure lasting eleven seconds.

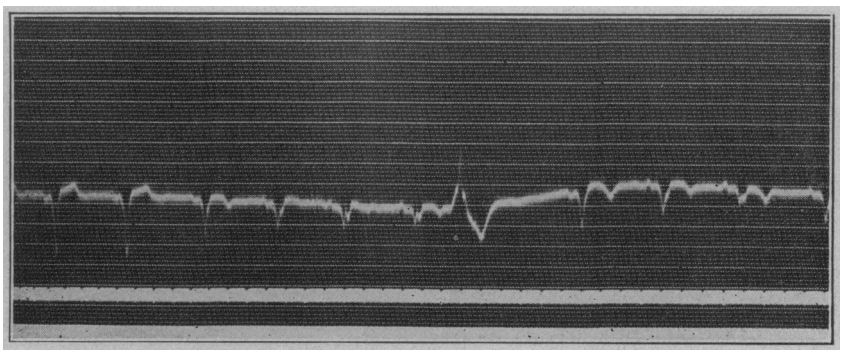

Fig. 12.—Lead 3. The first part of the figure shows a gradual transition from Rhythm 2 to Rhythm 1. This transition took place spontaneously. The P complex of the third cycle is transitional between those that precede and those that follow it. A ventricular extrasystole occurs.

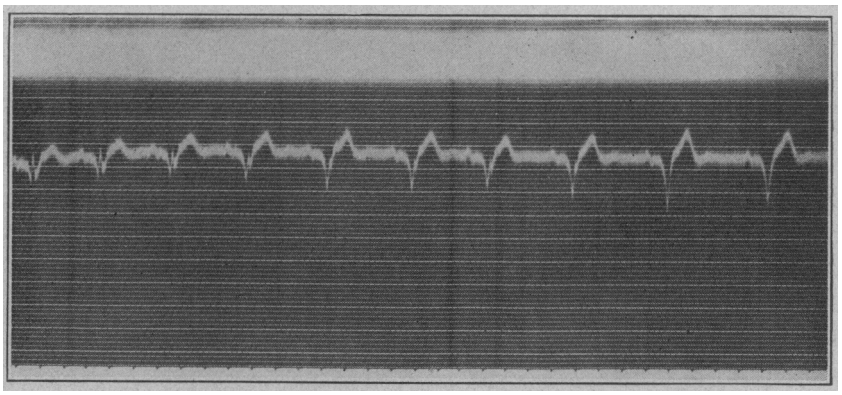

Fig. 13.—Lead 3. P is upright throughout the figure and the P-R interval is that of Rhythm 1. There is a gradual transition from complexes of the normal type to those characteristic of right branch block. Note the disappearance of the upright spike of the normal ventricular complex during the transition.

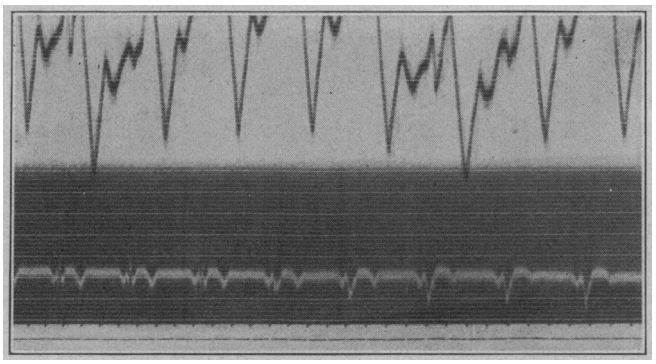

Fig. 14.—Lead 3. P is inverted and the P-R interval is reduced. The first four ventricular complexes are of the normal type. The last four ventricular complexes are diphasic and are of the type associated with Rhythm 2 except that their amplitude is much less. This curve was taken during an atropin experiment. The last four ventricular complexes probably represent incomplete branch block.

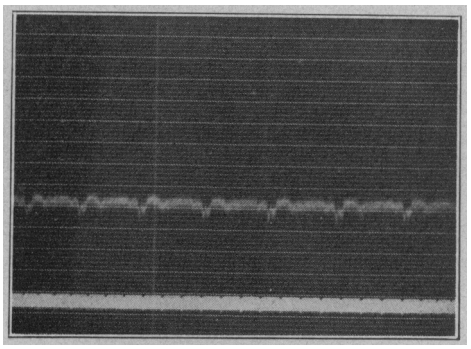

Fig. 15.—Lead 3. The inverted auricular complexes of Rhythm 2 are associated with the ventricular complexes of Rhythm 1.

observation, the normal rhythm was the one usually present. At this time Rhythm 2 could be produced almost at will by having the patient take a deep breath (Figs. 7 and 8). The abnormal rhythm appeared shortly after the beginning of expiratory slowing and after persisting for a number of beats disappeared as the heart rate quickened. The normal rhythm could also be converted into the abnormal rhythm by pressure upon the vagus nerves (Fig. 9). No difference between the two vagi was noted. Although, when the first electrocardiograms were taken, Rhythm 2 disappeared shortly after it was produced by deep breathing or vagal pressure, as time went on it showed a greater and greater tendency to persist, and at the end of the first week of the examination it was the rhythm usually present. At this time the normal rhythm could be produced by having the patient take a rapid succession of deep breaths. It was also possible to convert Rhythm 2 into the normal rhythm by the subcutaneous administration of 1/50 grain of atropin (Fig. 10) and this was done on five different occasions. After the normal rhythm had been produced by atropin it was impossible to cause Rhythm 2 to return by forced respiration or by any other means used to produce it before it appeared spontaneously.

Rhythm 2 could be slowed considerably by vagus stimulation (Fig. 11) the right vagus being somewhat more effective than the left. This shows that in this patient the upper portion of the junctional tissues was under vagus control.

To sum up; the normal rhythm when spontaneously present could be converted into an atrioventricular rhythm with right branch-bundle block by indirect or direct stimulation of the vagus nerves, and the abnormal rhythm when spontaneously present could be converted into the normal rhythm by the administration of atropin in doses sufficient to paralyze the vagi. This group of facts can lead to but one conclusion and that is that the vagi were partially responsible both for the change in the location of the pace-maker and for the abnormality of the ventricular complexes. The relationship of these nerves to the former has been discussed elsewhere[11] (Case 2).

Before discussing the effect of the vagus on the form of the ventricular complexes, it is necessary to examine the relationship between the A-V rhythm and the bundle-branch block. The fact that both are so constantly associated in the present case might lead one to suspect that the abnormal location of the pace-maker was entirely responsible for the abnormality of the ventricular complexes. It is difficult to see how this could be possible, for as has been pointed out above, the pace-maker must have been situated high up in the junctional tissues above the division of the His bundle. Even in complete heart block, when the pace-maker is much lower and nearer to the bifurcation of the A-V

bundle, the ventricular complexes are normal. Furthermore, in the present case, although the change in the location of the pace-maker was an abrupt one the change in the form of the ventricular electrocardiogram was often very gradual (Fig. 8). Transitional P waves were sometimes seen at the onset or offset of the A-V rhythm (Fig. 12) but these were due not to a gradual change in the location of the pace-maker but to interference between two contraction waves, one beginning at the sinus and the other at the A-V node.[11] That the abnormality of the ventricular complexes did not depend on the presence of A-V rhythm is also demonstrated by the fact that transitions between the normal and the abnormal complexes sometimes occurred without the pace-maker leaving the sinus node (Fig. 13). At other times, on the other hand, an A-V rhythm of the same type as that of Rhythm 2 was associated with ventricular complexes of the

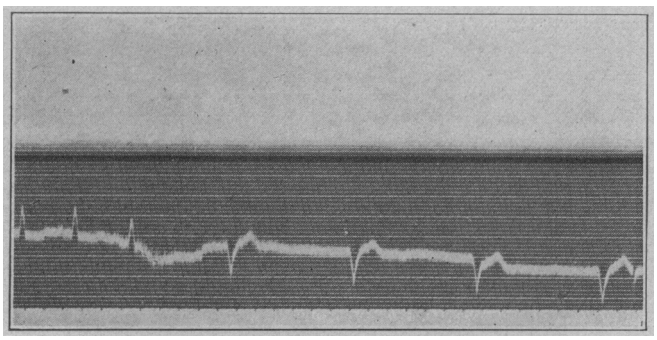

Fig. 16.—Lead 1. This curve illustrates the production of diphasic ventricular complexes in a second patient (see text). P cannot be seen during the abnormal rhythm.

normal type (Figs. 14 and 15). It would seem therefore that the bundle-branch block of Rhythm 2 did not depend on the abnormal site of impulse formation but that both were coordinated effects of vagus influence.

It is of course improbable that the bundle-branch block was due to vagus influence alone for it is rarely possible to produce diphasic ventricular complexes by vagus stimulation. We have carried out this procedure in a considerable number of patients and we have been able to produce such complexes in but one other instance (Fig. 16). The unsatisfactory character of the records obtained in that instance made it impossible to tell whether or not the ventricles were responding to supraventricular stimuli. It is probable therefore that in the previous patient, conduction through the right branch of the A-V bundle was already impaired and that this rendered it especially susceptible to vagus influence.

INCOMPLETE BUNDLE-BRANCH BLOCK

The characteristics of the abnormal ventricular complexes produced by the complete interruption of conduction through one branch of the A-V bundle are fairly well known both from experimental and clinical observations. Very little is known, however, about the ventricular complexes of incomplete block in one of the branches of the A-V bundle. For this reason, gradual transitions between normal ventricular complexes and those of bundle-branch block are of par-

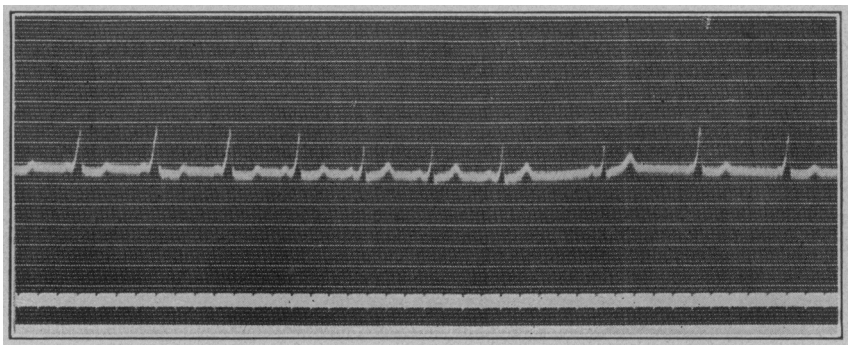

Fig. 17.—Lead 1. A spontaneous transition from Rhythm 2 to Rhythm 1 and the return to Rhythm 2. The transitions are sudden.

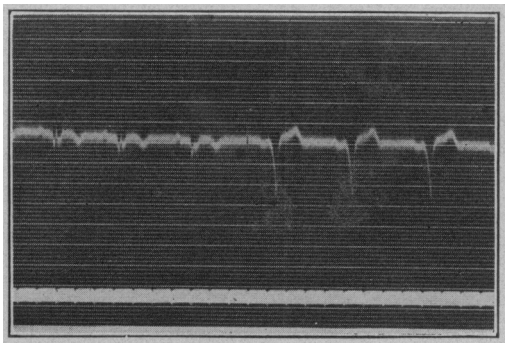

Fig. 18.—Lead 3. A sudden transition from Rhythm 1 to Rhythm 2.

ticular interest for presumably they represent varying degrees of partial bundle-branch block and their analysis may aid in the recognition of this condition when transitions are absent. In the present case, some of the transitions from the normal to the abnormal complexes were sudden (Figs. 17 and 18); but as a rule they were gradual. For the purpose of determining the characteristics of the ventricular complexes of partial bundle-branch block spontaneous transitions should be chosen

in order to eliminate the effects of the respiratory changes in the position of the heart on the ventricular complex. Such spontaneous transitions from normal to abnormal and from abnormal to normal complexes are shown in Figures 12 and 13. From the analysis of a large number of such transitions it was found that changes in the QRS group and in T usually appeared simultaneously. The most constant feature of the transitional complexes was their diphasic character, which was usually evident even in those nearest the normal complexes, and this change is probably present in the lightest grade of block. On the other side, the transitional complexes differed from the fully developed abnormal complexes principally in their lesser amplitude. As one proceeds from the abnormal complexes to those typical of complete branch-bundle block the amplitudes of the QRS group and of T gradually increase, and as these two deflections are in opposite directions the diphasic character of the complexes is gradually accentuated (Figs. 8 and 13). Although an increase in the duration of the initial deflection usually appeared with the first of the abnormal complexes it was usually much less striking than their diphasic character. The disappearance of the upright spike of the QRS group of the normal ventricular complex in Lead 3 during the transitions is interesting (Figs. 8 and 13).

VENTRICULAR COMPLEXES SUGGESTING LEFT BUNDLE-BRANCH BLOCK AT THE BEGINNING OF ATROPIN EXPERIMENTS AND DURING PAROXYSMS OF TACHYCARDIA

The effect of atropin on Rhythm 2 was complicated by a new rhythm which usually appeared about eight minutes after the injection of 1/50 grain of atropin sulphate and recurred with frequent transitions to the normal rhythm for from three to five minutes. This third rhythm is illustrated in Figures 19 (Lead 1) and in 20 (Lead 3). Although no P waves are seen in the electrocardiogram, it is evident from the tall venous waves which occurred during this rhythm (Fig. 21) that auricles and ventricles contracted simultaneously. This indicates that the site of origin of Rhythm 3 was in the lower junctional tissues. The change in the location of the pace-maker from the upper (Rhythm 2) to the lower (Rhythm 3) junctional tissues after atropin is probably analogous to the appearance of A-V rhythm in normal individuals under similar conditions.[13] The transitions between Rhythm 1 and Rhythm 3 differed from those between Rhythm 1 and Rhythm 2 in that they were usually sudden (Figs. 19, 20 and 22). Ventricular complexes which may be regarded as transitional in form

13. Wilson, Frank N.: THE ARCHIVES INT. MED., page 989, this issue.

between the ventricular complexes of the normal rhythm and those of Rhythm 3 were frequently recorded, however, and these usually occurred during a period just preceding the appearance of Rhythm 3. These transitional complexes (Fig. 23) appeared only after atropin and were not associated with any change in the location of the pacemaker as is shown by the length of the P-R interval and the form of the P waves which precede them (Fig. 23). It seems likely, therefore, that in Rhythm 3 as in Rhythm 2 the abnormality of the ventricular complexes did not depend on the abnormal site of impulse formation.

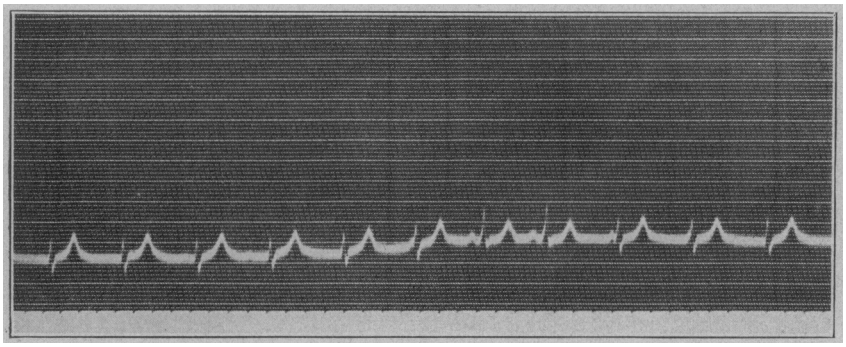

Fig. 19.—Rhythm 3, Lead 1. The ventricular complexes are not diphasic but T is exaggerated. A transition from Rhythm 3 to Rhythm 1 occurs. P cannot be identified during Rhythm 3.

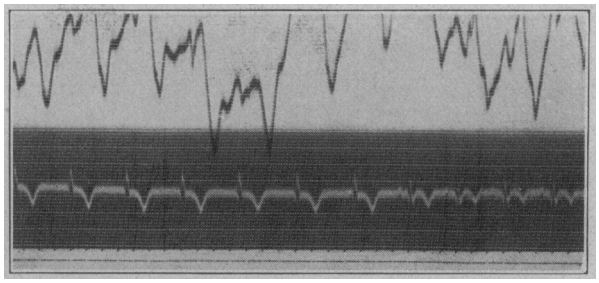

Fig. 20.—Lead 3. A transition from Rhythm 3 to Rhythm 1 is shown. P is not visible during Rhythm 3.

Transitions between a sinus rhythm and Rhythm 3 are illustrated in Figures 19 and 22. The P wave gradually approaches the ventricular complex and finally disappears within it. The reverse process occurs when the abnormal gives place to the normal rhythm (Figs. 20 and 22). The fact that the P waves remain normal during the transitions indicates that the auricular contractions which they represent were responses to the sinus node. The shortening of the P-R interval indicates, therefore, that a lower center has escaped and that

the ventricles are responding to this center. After several heart cycles the higher rate of this lower center enabled it to replace the sinus node as pace-maker for the auricles also. The reverse process took place when the inherent rate of the sinus became more rapid than that of the ectopic center. When transitions occurred between Rhythm 2 and Rhythm 3 a similar phenomenon took place. The inverted P of Rhythm 2 gradually approached and disappeared into the ventricular

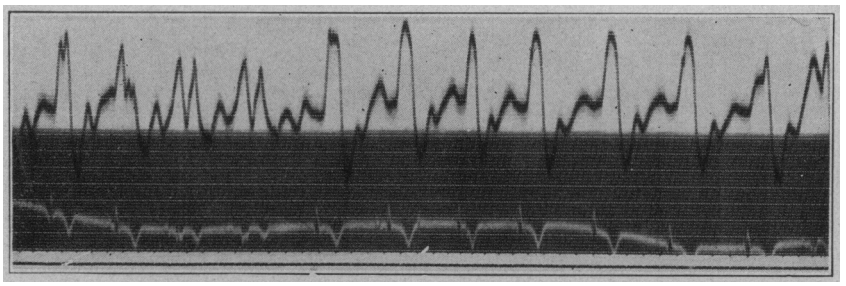

Fig. 21.—Lead 3. A transition from Rhythm 1 to Rhythm 3 occurs in the last part of the figure. The venous pulse during Rhythm 3 is dominated by a tall wave such as is usually seen when auricles and ventricles contract simultaneously. This wave is no broader that the *a* wave which occurs during the normal rhythm (Cycles 3 and 4). Note that even when P only just emerges from the ventricular complex in the second cycle from the right hand side of the figure *a* and *c* are separately indicated in the venous pulse.

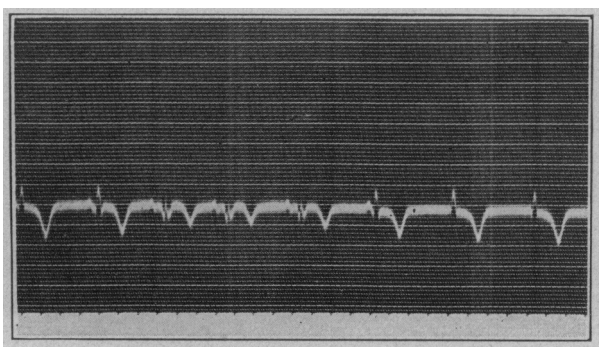

Fig. 22.—Lead 3. A transition from Rhythm 3 to sinus rhythm occurs and also a transition in the reverse direction. Notice that P moves out of the ventricular complex at the end of Rhythm 3 and reenters the ventricular complex when Rhythm 3 reappears.

complex. The period of transition lasted for only one or two complexes, however, because of the close proximity of the two opposing centers (Fig. 24). The fact that P never emerged between R and T indicates that the impulses from the center which was responsible for Rhythm 3 must have reached the auricles at some time during the R wave, or in other words that these impulses arose from the region of

the A-V node itself. The tall venous waves of Figure 21 also indicate that during Rhythm 3 auricular systole fell during the first portion of ventricular contraction.

The ventricular complexes of Rhythm 3 are distinctly abnormal. In Lead 3 (Fig. 24) they are typically diphasic. The duration of the initial deflection is not distinctly lengthened but its amplitude is increased and T is exaggerated. In Lead 1 (Fig. 19) the ventricular

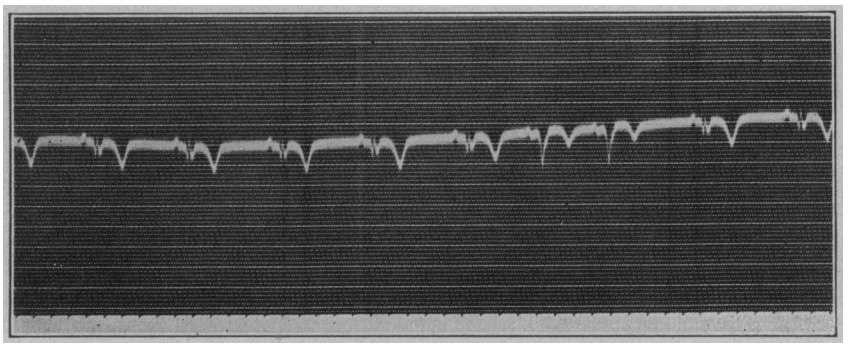

Fig. 23. Lead 3. Taken during an atropin experiment. The ventricular complexes of the first part of the figure are transitional between those of the normal type and those seen in Rhythm 3. Two ventricular complexes transitional in form between the ventricular complexes in the first part of this figure and the ventricular complexes of Rhythm 2 occur in the latter part of the curve.

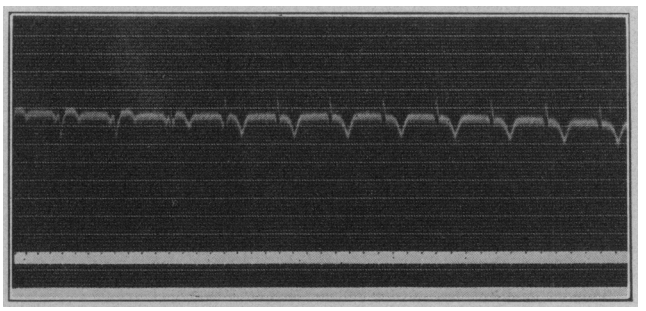

Fig. 24.—Lead 3. A transition from Rhythm 2 to Rhythm 3 occurs. The inverted P wave enters the ventricular complex at the beginning of Rhythm 3. The period of transition is short.

complexes are not diphasic. When compared with the normal, however, R is shorter while S and T are exaggerated. Before discussing the question of the significance of these abnormal complexes let us examine a fourth rhythm observed in this patient.

This patient had at times paroxysms of tachycardia which always began and ended suddenly. During the six weeks that he was under observation two such attacks were recorded. The rhythm present at

these times is illustrated in Figures 25, 26 and 27. Both attacks were stopped by Valsalva experiments and the end of one of them is shown in Figure 28. In this figure it will be seen that the abnormal rhythm ceased abruptly and after a long postparoxysmal pause the sinus rhythm returned. The rate of the heart during the paroxysm was about 170. In contrast to most patients who are subject to attacks of paroxysmal tachycardia our patient exhibited very few single extrasystoles; only two (Figs. 12 and 28) were recorded in a large number of tracings and these being of the ventricular type bore no relationship to the attacks of tachycardia.

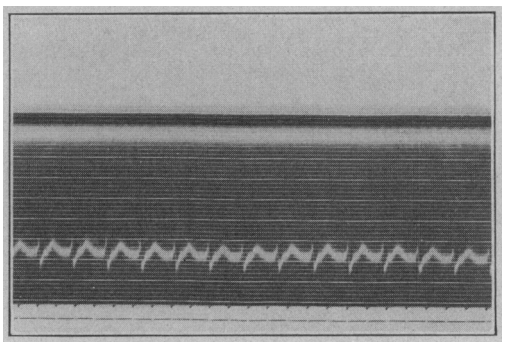

Fig. 25.—Rhythm 4, Lead 1. The ventricular complexes are like those in Figure 20. P is not seen.

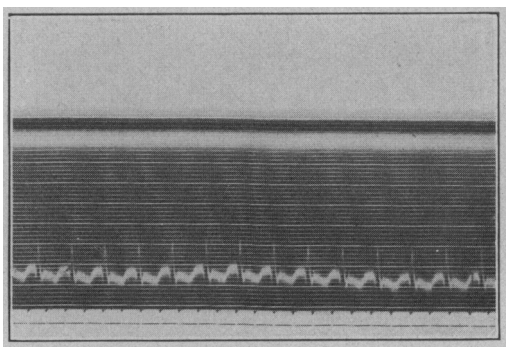

Fig. 26.—Rhythm 4, Lead 2. The ventricular complexes are diphasic. P is not seen. The heart rate is 170.

The electrocardiograms taken during the attacks show no definite P waves and except for the difference in heart rate resemble very closely those obtained during Rhythm 3. The venous pulse is of the positive type usually seen in paroxysmal tachycardia (Fig. 27). Such a phenomenon may be due either to the fact that the auricular contractions fall on the ventricular contractions of the prevoius heart cycle

or it may be due to the fact that both chambers are stimulated simultaneously from a pace-maker in the junctional tissues. There are two reasons for thinking that the latter explanation is the correct one in the present case. The first is the marked similarity between the electrocardiograms of Rhythms 3 and 4. This close resemblance between the electrocardiograms of the two rhythms makes it seem likely that they originated at the same point. The second reason for thinking

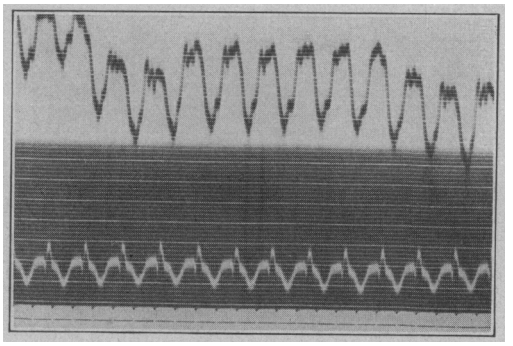

Fig. 27.—Rhythm 4, Lead 3. The ventricular complexes are typically diphasic and like those of Rhythm 3 in this lead. P is not seen.

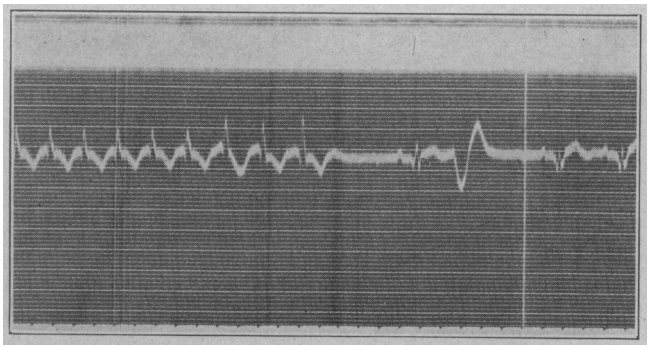

Fig. 28.—The end of an attack of paroxysmal tachycardia is shown. There is a long postparoxysmal pause followed by a normal contraction and then an extrasystole. The last ventricular complex of the paroxysm does not differ from the preceding ones.

that Rhythm 4 arose in the junctional tissues is that the last ventricular complex of the paroxysm does not differ from the rest (Fig. 28). This is not the case when auricular contraction falls on the ventricular contraction of the previous heart cycle; for when this occurs the last ventricular complex of the paroxysm, unlike the others is not modified by a simultaneous P. Paroxysmal tachycardia originating within the

junctional tissues is comparatively rare. Cases have been studied electrocardiographically by Lewis[14] and by Cohn.[15]

The abnormal ventricular complexes of Rhythms 3 and 4 may have been due to an abnormal spread of the contraction wave over the ventricles because of a block in the conduction system. These complexes strongly suggest a block in the left branch of the His bundle but they are not perfectly typical. This discrepancy may be explained by assuming that the block was a partial one affecting the left branch itself or a complete one affecting one of the divisions of this structure.

Rothberger and Winterberg[9] have shown that the accelerator nerves when stimulated separately may profoundly change the contour of the ventricular complexes even when these are responses to supraventricular stimuli. These investigators have obtained complexes of somewhat the same outline as those of Rhythms 3 and 4 by stimulation of the accelerator but we regard it as improbable that the abnormal complexes of these rhythms were due to abnormal activity of this nerve.

In the present case therefore the ventricular complexes were at times normal, at times typical of right bundle-branch block, and at still other times suggestive of left bundle-branch block. It does not seem to us that a complete explanation can be offered for all the changes observed but it should be pointed out that a similar condition of affairs has been observed both in man and in experimental animals. We have already referred to the clinical case described by Mathewson[4] in which an irregular alternation of ventricular complexes of right and left branch block with complexes of the normal type occurred. A similar heart mechanism can be produced in animals by ligation of the septal artery (Kahn[9]). The variations in the ventricular complexes in the present case differ from similar variations previously observed, however, in that they were, in a great measure at least, produced by changes in vagus influence.

SUMMARY

The case here reported showed at different times four distinct rhythms and at least three types of ventricular complexes.

In the first rhythm the ventricular complexes were normal and the pace-maker was in the normal location.

In the second rhythm the ventricular complexes were characteristic of a block in the right branch of the His bundle. These abnormal

14. Lewis, T.: Auricular Fibrillation and its Relationship to Clinical Irregularity of the Heart, Heart, 1909-1910, i, 306; Paroxysmal Tachycardia, Accompanied by the Ventricular Form of Venous Pulse, Heart, 1910-1911, ii, 127.

15. Cohn, A. E.: A Case of Paroxysmal Tachycardia, Heart, 1910-1911, ii, 170.

ventricular complexes were usually but not always associated with an atrioventricular rhythm originating in the upper levels of the junctional tissues. Both the A-V rhythm and the abnormal ventricular complexes could be produced by vagus stimulation and when spontaneously present could be abolished in favor of the normal rhythm by the administration of atropin.

Ventricular complexes, transitional in form between the normal complexes and the abnormal complexes mentioned above are discussed.

Rhythm 3 appeared during the early stages of atropin action and at no other time. The ventricular complexes of this rhythm were abnormal and suggested a block in the left branch of the His bundle. The pace-maker was situated in the region of the A-V node. Ventricular complexes transitional in form between those of the normal rhythm and those of Rhythm 3, occurred, however, while the pace-maker was situated at the sinus node. It is probable therefore that the abnormality of the ventricular complexes of Rhythm 3 did not depend on the abnormal site of origin of this rhythm.

Rhythm 4 was a paroxysmal tachycardia which originated at the same point as Rhythm 3 and which was associated with the same type of ventricular complex.

The various changes in the mechanisms of the heart beat observed in this case occurred, in a great measure at least, in response to changes in vagus control.

Although the changes in the form of the ventricular complex were usually associated with a change in the location of the site of impulse formation, this was not invariably the case, so that it is likely that both these changes were coordinated effects of variations in vagus influence.