

THE RELATION OF CHANGES IN THE FORM OF THE VENTRICULAR COMPLEX OF THE ELEC- TROCARDIOGRAM TO FUNCTIONAL CHANGES IN THE HEART *

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Constancy of form is one of the most striking characteristics of electrocardiograms obtained at various times from the same individual. Any change in form is therefore of interest, as it indicates some alteration in the passage of the impulse of contraction through the heart or some change in the manner of the muscular contraction. Changes in heart rate and in the force of contraction are not, as a rule, accompanied by definite alterations in the form of the electrocardiogram.

The ventricular portion of the electrocardiogram is composed of a series of waves and is initiated by a group of three waves, the so-called Q, R, S group. Of these, only the R wave is constant in normal individuals in records obtained by the three leads of Einthoven. There are at present differences of opinion as to the functional activity of the ventricles responsible for this group of waves. Without taking up the various points in this controversy, it may be said that the evidence is in favor of the hypothesis that it is the passage of the impulse through the ventricles rather than the actual muscular contraction which gives rise to this part of the electrocardiogram and initiates the ventricular complex. As Einthoven¹ has suggested this Q, R, S group deserves a closer examination than has been hitherto given to it in clinical electrocardiography, as it shows the path by which the excitation wave is conducted from the auricles to all parts of the ventricular walls. It is to changes in this initial part of the ventricular electrocardiogram that attention is directed in this paper.

Before discussing the type of change to be described, it is necessary to consider several well-defined abnormalities of the ventricular complex which are frequently encountered in clinical electrocardiograms, and which are well understood, because they have been repeatedly reproduced under experimental conditions. The commonest of these is that caused by the passage of impulses arising in some abnormal point in the ventricles, the so-called ectopic stimuli, which give rise

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1. Einthoven: Different Forms of the Human Electrocardiogram and Their Significance, *Lancet*, London, 1912, i, 853.

to ventricular premature contractions or extrasystoles. When this occurs the impulse passes through the ventricles in abnormal directions, yielding a ventricular complex which is, as a rule, much deformed.

Another well-defined abnormality of the ventricular complex is caused when an impulse which reaches the ventricles by the usual path is hindered in its passage throughout the ventricles by a derangement of one of the branches of the conducting system which should convey the impulse either to the right or to the left ventricle. Such a condition of bundle-branch block has been produced in animals by cutting one or the other of the main branches of the conducting system. This procedure is followed by a great change in the form of the electrocardiogram, as first described by Eppinger and Rothberger.² Electrocardiograms similar to those obtained after this experimental procedure have been obtained from patients whose hearts have later shown, on histologic examination, a lesion of one or the other of the main branches of the conducting system. A sufficient number of cases have been studied to justify the diagnosis of the lesion from the type of the electrocardiogram. Carter,³ who reported twenty-two cases of bundle-branch block, has pointed out the requirements of the form necessary to establish the diagnosis. The initial Q, R, S group of the ventricular complex exceeds the normal time 0.1 second; the waves are large, of bizarre forms, and the final deflection T is directed oppositely to the initial ventricular wave.

Changes in the distribution of muscle mass between the two ventricles, as occurs in cardiac hypertrophy, also leads to alterations in the form of the electrocardiograms, which is especially noticeable when the records obtained by the three usual leads are compared with one another. These changes have been recently demonstrated experimentally by Fraser.⁴ They are especially noteworthy in cases of congenital malformation of the heart. That the marked changes in the form of the electrocardiogram which occur in this condition are dependent on the abnormal distribution of the muscle mass between the two ventricles has been clearly demonstrated recently in our laboratory by McCulloch.⁵ One other change in the form of the electrocardiogram remains to be mentioned, which is frequently encountered clinically, namely, alterations in the size or direction of the final wave of the complex, the so-called T wave. This wave, which is normally a long, well-defined upwardly directed wave, may become diminished

2. Eppinger and Rothberger: Ueber die Folgen der Durchschneidung der Tawaraschen Schenkel des Reizleitungssystems, *Ztschr. f. klin. Med.*, 1910, lxx, 1.

3. Carter: Clinical Observations on Defective Conduction in the Branches of the Auriculoventricular Bundle, *THE ARCHIVES INT. MED.*, 1914, xiii, 803.

4. Fraser: Changes in the Electrocardiograms Accompanying Experimental Changes in the Rabbit's Heart, *Jour. Exper. Med.*, 1915, xxii, 292.

5. McCulloch: *Am. Jour. Dis. Child.*, 1916, xii, 30.

in size, disappear or become inverted. Such a change is frequently observed in heart disease, especially when the myocardium is damaged. Kraus and Nicolai⁶ have laid stress on the inverted or negative T wave as an indication of myocardial weakness, and probably all who have made use of the electrocardiogram in the study of clinical material will agree that this change frequently accompanies other signs of myocardial disease. The fact, however, that such a change takes place in the T wave when digitalis is administered, as pointed out by Cohn, Fraser and Jamieson,⁷ renders its significance uncertain as an indication of myocardial weakness. It is mentioned here as being the one change in form of the ventricular complex which has been hitherto recognized as giving evidence as to the functional efficiency of the heart muscle.

The object of the present paper is to discuss a series of records which show changes in the form of the ventricular electrocardiogram differing from those that have been mentioned. These changes consist in alterations in the form of the Q, R, S group which are apparently dependent on a functional derangement of the ventricles, hindering the normal spread of the impulse of contraction. The spread of the impulse is hindered in some cases because the conducting system of the ventricles has not had a sufficient time to recover from the effects of an impulse that has just caused a ventricular contraction, while in other cases the recovery of the intraventricular conduction is abnormally delayed, so that the impulse spreads through the ventricles properly only after a prolonged period of ventricular rest. In some instances in which a prolonged rest does not occur the electrocardiogram is of a form which indicates that the proper spread of the impulse through the ventricles is constantly lacking. Changes in form of the complexes have been observed to occur from time to time parallel to the clinical course of cardiac cases. A consideration of these changes indicates that the electrocardiographic method may demonstrate functional deficiency of the conducting system in the ventricles and in this way be a means of revealing diminished functional capacity of the ventricles.

The records have been obtained by means of the Edelmann type of string galvanometer. In all instances the tension of the string has been so adjusted that the passage of two millivolts through it, with the patient in the circuit, results in a deflection on the record of two centimeters. Only those records have been used in which this adjustment did not render the string sufficiently slack to cause any marked fling,

6. Kraus and Nicolai: *Das Elektrokardiogram*, Leipzig, 1910, p. 278.

7. Cohn, Fraser and Jamieson: *The Influence of Digitalis on the T Wave of the Human Electrocardiogram*, *Jour. Exper. Med.*, 1915, xxi, 593.

which results in a definite deformity of the curves. Omitting this precaution leads to striking errors. In lettering the curves the single letter R is used to mark the Q, R, S group.

CASE 1.—J. B., a man, 42 years of age, had been aware of an irregularity of the pulse since boyhood. Except for a heightened blood pressure (systolic 148 mm. of mercury, diastolic 95), moderate cardiac enlargement and a trace of albumin in the urine, he gave no signs of disease. He had never had a serious acute illness, and his only excess had been very constant hard work as a lawyer. His life had always been one of unrestricted activity. An examination was undertaken only as a means of determining the type of cardiac arrhythmia. Electrocardiograms (Fig. 1) revealed a sino-auricular block, a complete cardiac cycle being omitted every few beats. The arrhythmia was complicated, however, by the occurrence of an idioventricular beat, always occurring near the end of the long pause caused by the missed beat and immediately before the auricular contraction which initiated the restoration of the regular rhythm. This idioventricular beat yielded a complex practically identical with that of the normal beats, indicating that it arose in the junctional tissues before the division of the conducting system, and that the impulse spread through the ventricles in a normal manner.

The special interest in this record centers on the complexes yielded by the ventricular responses to the auricular contractions which occur immediately after the onset of the idioventricular contractions. The records furnish twenty-five examples of this combination. The electrocardiograms show that the ventricular responses to these auricular contractions vary with the time that elapses between the onset of the idioventricular beats and the auricular contractions. When this time is less than 0.15 of a second, usually no ventricular response follows the auricular contraction. As this time lengthens the ventricles respond, but when it is only a little longer than 0.15 of a second, the response is abnormal, as indicated by a prolonged auriculoventricular conduction time and by a markedly deformed ventricular complex.

The longer the time between the onset of the idioventricular contraction and the following auricular contraction, the shorter becomes the auriculoventricular conduction time and the nearer to the normal becomes the form of the ventricular complex. The more abnormal ventricular complexes show a prolongation of the time occupied by the Q, R, S group, which distinctly exceeds the normal time of 0.1 of a second. Both the ascent and descent of the R wave is more gradual than normally and there is a blunt, notched, or broken apex to the R wave, which is often distinctly diminished in height. The complexes are those to which Lewis⁸ has applied the term "aberrant."

These changes in the ventricular complexes of the electrocardiogram are interpreted as follows: At times the impulse from the auricular contraction reaches the ventricles when they are still in

8. Lewis: *Observations on Disorders of the Heart's Action*, Heart, 1912, iii, 279.

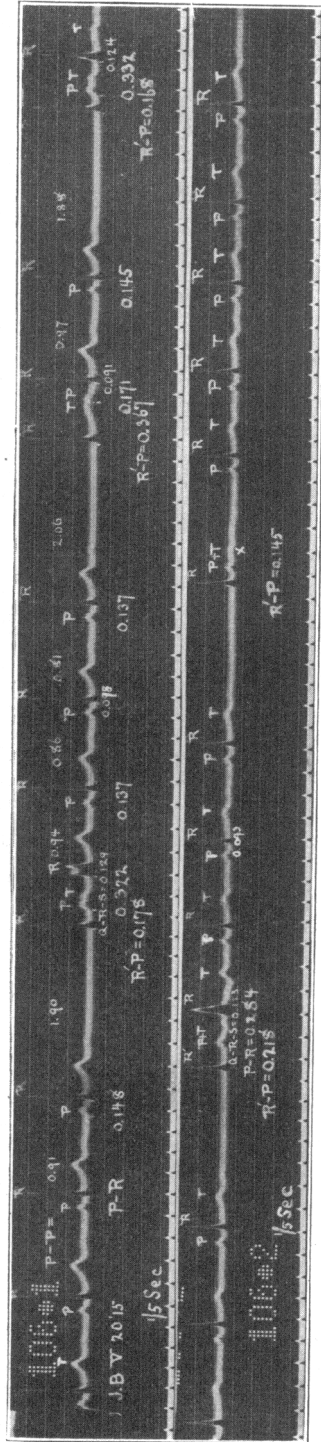


Fig. 1 (Case 1). Sino-auricular block, with idioventricular contractions. The abnormal ventricular complexes are yielded by contractions which occur before the ventricles have recovered from a previous contraction.

the refractory phase, and so no ventricular response results. Usually the impulse reaches the ventricles when they have only partly recovered, and the conduction through the ventricles is depressed under these conditions, the impulse is delayed in part of the usual course, or follows an abnormal course, being blocked along some of its usual paths. Thus the intraventricular conduction apparently shows the same depression as is present in the auriculoventricular conducting system. The prolonged auriculoventricular conduction time is in part responsible for the degree of recovery which takes place in the ventricles, as this adds to the time between the idioventricular contraction and the ventricular contraction which follows.

The electrocardiograms of this case illustrate the changes in the form of the ventricular complex which are caused by functional derangement of the ventricles, when a contraction occurs before the ventricles have fully recovered from a previous contraction. They indicate also that the degree of abnormality of the complex parallels the degree of depression of the intraventricular conduction.

CASE 2.—S. B., a woman of 63 years, suffered from atrophic cirrhosis of the liver, chronic myocarditis and chronic valvular cardiac disease (mitral insufficiency). On May 2, 1915, when the first record was made, she was cyanotic, markedly dyspneic, and there was general anasarca. The heart was much enlarged, cardiac dullness extending 18.5 cm. to the left of the midsternal line, and besides the systolic murmur in the apex region there was a pericardial friction rub. The heart was beating at a rate of 87 per minute, and the systolic blood pressure was 110 mm. of mercury, the diastolic 65 mm.

The electrocardiograms (Fig. 2) are very abnormal in form. The Q, R, S group is composed of a series of waves, none of which resemble those of the normal electrocardiogram. The period of time occupied by the occurrence of this group of waves is distinctly longer than the normal 0.1 of a second.

On May 18, when electrocardiograms were again obtained (Fig. 3), the patient was much improved. The dyspnea, cyanosis and edema were distinctly less. The systolic blood pressure had risen from 110 to 120 mm. of mercury without change in the diastolic pressure. The heart rate was 82 per minute. The electrocardiograms show an arrhythmia caused by premature contractions of auricular, nodal and ventricular origin. There is a distinct change in the form of all ventricular complexes as compared with those seen in Figure 2. The most striking feature of the record is the change in the form of the ventricular complexes which occur after a period of increased ventricular rest. This change is seen after the compensatory pause following premature ventricular beats and when premature auricular beats are blocked. Under these conditions the ventricular complexes are much more nearly normal in form.

This case illustrates that changes in the form of the ventricular complexes may occur with improvement of the cardiac efficiency, as evidenced by the clinical improvement, and with increased periods of ventricular rest. The usual diastolic pause between the regular beats was not long enough to allow the intraventricular conduction to recover sufficiently to allow the impulse to pass normally. This case is, therefore, an example of those in which the recovery of the intra-

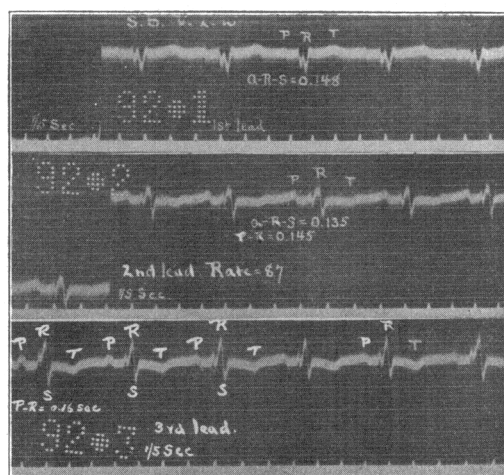


Fig. 2 (Case 2).—First record, May 2, 1915. Form of ventricular complexes abnormal in all leads. The Q, R, S time is prolonged; P-R time normal. The patient showed signs of marked cardiac decompensation.

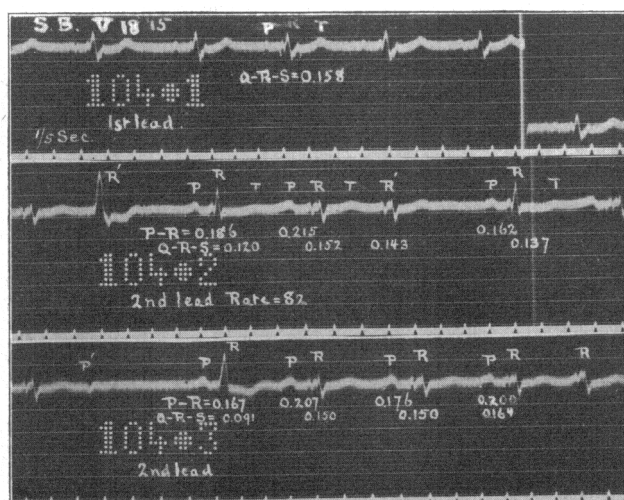


Fig. 3 (Case 2).—Second record, May 18, 1915. The form of ventricular complexes differs from that seen in Figure 2. The Q, R, S time and the P-R time are distinctly prolonged; the form of the ventricular complexes more nearly normal after a prolonged ventricular rest. The patient showed distinct clinical improvement.

ventricular conduction is abnormally delayed, so that the impulse spreads through the ventricles properly only after a prolonged period of ventricular rest.

CASE 3.—A man, 33 years of age, came into the outpatient department complaining of pain in the chest and shortness of breath. The heart dullness extended 5 cm. to the right and 15 cm. to the left of the midsternal line. The heart sounds were distant and blurred, and the heart was beating very rapidly and irregularly. The electrocardiograms (Fig. 4) obtained on this day show a heart rate of 174 per minute. The ventricular complexes occur irregularly and are of unusual forms, while no auricular complexes are visible. The main ascents and descents of the Q, R, S group are gradual, and the time occupied by this group is distinctly prolonged, but difficult to determine accurately. The slight variation of form from beat to beat may be caused by combinations of ventricular complexes with the waves of auricular fibrillation. The patient refused to remain in the hospital. He was put on digitalis and returned in forty-eight hours, after two drams of the tincture had been taken. On this admission there was distinct improvement in symptoms and the heart rate was greatly reduced, varying from 77 to 100 beats per minute. Electrocardiograms (Fig. 5) obtained this day are typical of auricular fibrillation. The arrhythmia is much more marked, and after the longer diastolic pauses, the ventricular complexes have a much more normal form than after the shorter pauses. This is especially noticeable in the first lead, where the abnormal, blunt, low complexes, composed of an R wave with a slow ascent and descent become, after prolonged diastoles, sharp pointed, higher waves resembling closely those usually seen in auricular fibrillation. A study of the various records convinces that this change in form is not caused by combinations of the ventricular complexes with the waves of auricular activity. The fact that this patient died suddenly two days after his second visit, when under a severe emotional strain, suggests that the heart muscle was badly damaged.

The case is one in which the ventricles responded to impulses from the fibrillating auricles at a very rapid rate, and the periods between contractions were not of sufficient duration to allow complete recovery of the intraventricular conduction. The cardiac slowing which resulted from the administration of digitalis caused some improvement in this respect, and resulted in longer ventricular rests, which were followed by ventricular contractions yielding normal or nearly normal complexes.

CASE 4.—A man, aged 59, suffering from syphilis and complete heart block, was under almost continuous observation for a period of nearly two years, during which time numerous electrocardiograms were obtained. During the first six months he had many attacks of unconsciousness and convulsions, with prolonged periods of ventricular inactivity, giving the typical picture of Stokes-Adams syndrome. After March, 1914, these did not occur, and electrocardiograms similar to those shown in Figure 6 were constantly obtained until June 26, 1915. On this day a marked change in the form of the ventricular complexes was noted (Fig. 7). It is seen that a marked change occurs in the form of the Q, R, S group in all leads and the time occupied by the group greatly exceeds the normal. The broad form with the notched top seen in the first lead is especially noteworthy, as it resembles closely the abnormal form seen in the first lead of the first and third cases.

On the day before the record was made the patient had three short "sinking spells," in which he became semiconscious. These spells lasted two

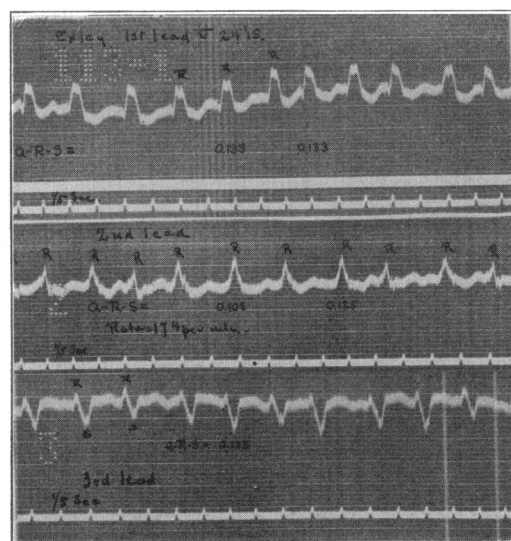


Fig. 4 (Case 3).—May 24, 1915. Auricular fibrillation; very rapid ventricular rate, with markedly deformed ventricular complexes.

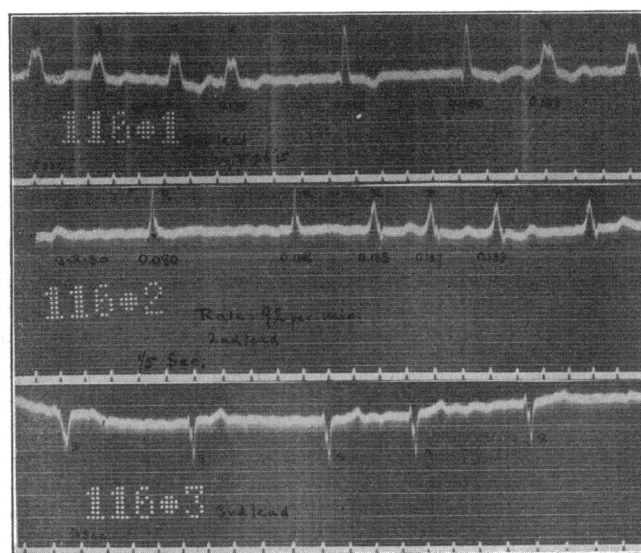


Fig. 5 (Case 3).—May 26, 1915. Auricular fibrillation; after taking tincture of digitalis for forty-eight hours. Striking change in the form of the ventricular complex after ventricular pauses, especially in the first lead.

or three minutes only and came on just as the patient was about to "drop off" to sleep. These spells continued for several days and gradually disappeared. There was no change in the pulse rate at these times of syncope, it remaining between 35 and 43 per minute. During the time when these attacks of syncope occurred the patient was in a critical condition. There was marked cyanosis, dyspnea, Cheyne-Stokes respiration and collections of fluid in the chest, peritoneum and lower extremities. His death was expected hourly, and there seemed to be extreme cardiac insufficiency. But he gradually improved, however, and July 5 he was much better, the cyanosis, dyspnea and edema being less marked. On this date electrocardiograms (Fig. 8) showed another change in form. The following note was made just after the records were taken: "Electrocardiograms taken this afternoon show a very interesting alternation in the form of the Q, R, S group of the ventricular complexes. They are first of the type formerly seen and then of the type which appeared constantly a week or so ago. There is apparently no difference in the effectiveness of the ventricular contractions yielding the varying complexes, as no alternation of the pulse is noted in an examination made directly after the records were obtained, either by palpation of the radial pulse or by auscultation over the brachial artery with the sphygmomanometer applied. The systolic blood pressure is 105 mm. of mercury, the diastolic 45. All beats come through with equal intensity."

At first sight one would be inclined to interpret these changes in form as being caused by contractions set up by impulses arising from two distinct foci, resulting in the passage of the impulses along two different sets of paths. A record obtained with the first lead, however, is evidence for the belief that the impulse of contraction was spreading properly through the ventricles only in alternate beats (Fig. 9). It is seen in this record that a transition form occurs (the third and fifth complexes), suggesting that the spread of the impulse was either not always complete or was not at the same rate through each ventricle. This conception of the variations in the spread of the impulse seems to explain these transition forms of ventricular complexes more satisfactorily than assuming the presence of a shifting focus of stimulus formation.

By July 27, 1915, the patient's condition was greatly improved. He no longer had dizzy spells and was able to be up and about. The electrocardiograms on this date were of the same form as seen previous to the period of marked cardiac insufficiency (Fig. 10).

This case shows a transient but extremely marked change in the form of the ventricular complexes during complete heart block, occurring during a period of great cardiac insufficiency. The abnormally slow rate of the ventricles allows the element of functional fatigue alone to be ruled out. Here the intraventricular conduction was so deranged that the long ventricular pauses at first did not lead to its recovery, while later the intraventricular conduction was apparently normal or partially recovered in alternating beats. Finally the form of the complexes returned to what must be considered the normal for this case, showing that the functional derangement of the ventricles responsible for the change in form was transient.

CASE 5.—A colored woman, aged 30, suffered from a generalized infection with *Streptococcus viridans*, acute endocarditis, embolism, cardiac dilatation and hypertrophy and syphilis. On Oct. 17, 1915, she had moderate dyspnea. The cardiac dullness extended 3.5 cm. to the right and 12 cm. to the left of the midsternal line. The heart rate was 91 per minute, while the systolic blood pressure was 120 mm. of mercury, the diastolic 72. There were no marked

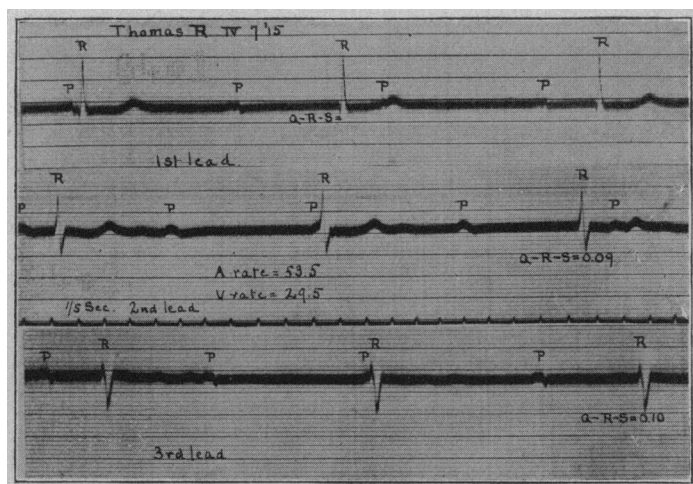


Fig. 6 (Case 4).—April 7, 1915. Complete heart block; the usual type of ventricular complex; normal Q, R, S time.

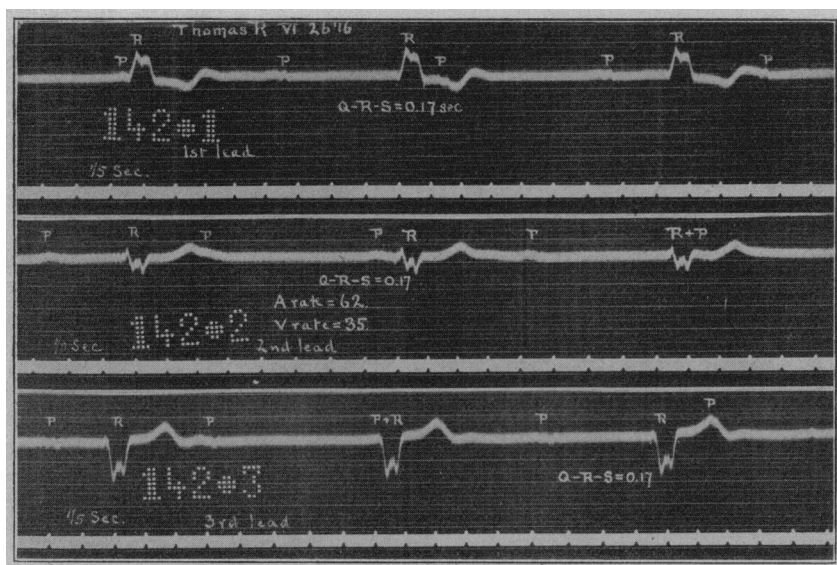


Fig. 7 (Case 4).—June 26, 1915. Striking change in form of the ventricular complex accompanying severe symptoms. Compare with Figure 6.

signs of cardiac insufficiency. The electrocardiograms obtained on this day showed high R waves, with sharp ascents and descents in all leads (Fig. 11).

On Dec. 11, 1915, the patient was very ill and showed definite signs of decompensation. The systolic blood pressure had fallen 27 mm. of mercury, while the diastolic pressure was unchanged. The heart rate was 110. The outline of cardiac dulness was unchanged. Electrocardiograms (Fig. 12) show a distinct change in form of the ventricular complexes, the R waves being much less high, and the ascent and descent, especially in the first lead, being slow. The Q, R, S time is not, however, definitely changed.

In this case well-defined changes in the ventricular complexes occurred synchronously with distinct decrease in cardiac efficiency. The form of the complexes resembles sufficiently those accompanying definite functional depression in the previous cases to suggest that the change which occurred during failure of the cardiac efficiency is the result of the cardiac failure and indicates presumably derangement of the intraventricular conduction.

CASE 6 is an example of change in form of the ventricular complex which occurred synchronously with a marked improvement in symptoms and apparently because of functional improvement of the ventricles. The patient was a woman of 24 who suffered from aortic and mitral insufficiency and who showed at times alternation of the heart beat. On June 17, 1915, when the record shown in Figure 13 was obtained, there was dyspnea while at rest in bed and other signs of cardiac decompensation. The electrocardiogram shows a slightly slowed ascent and descent in the R waves, and in Lead I the blunt, split-topped form is seen, which suggests that seen in some of the previous cases.

These abnormalities are less marked, and almost absent in records obtained on May 24, 1916 (Fig. 14), when the patient was no longer in the hospital, but returned merely for observation. On this day there was slight dyspnea after walking to the heart station, but no other symptoms suggesting cardiac insufficiency.

The change in form in the ventricular complexes which accompanied marked improvement of the cardiac efficiency seems to indicate that the abnormalities of the complexes observed during definite decompensation were dependent on the cardiac insufficiency. The fact that in this case alternation of the heart beat was not accompanied by alternation in the form of the ventricular complexes is evidence in favor of the belief that the abnormalities seen in Figure 13 were not caused by changes in muscular contractions as such, and makes it appear more likely that they resulted from defective conduction of the impulses through the ventricular musculature.

CASE 7 is included as one of marked cardiac decompensation, which showed abnormal ventricular complexes similar to those seen in the previous case. The electrocardiogram (Fig. 15) was obtained from a man with aortic insufficiency of syphilitic origin, whose heart was evidently much dilated. Here again the gradual ascent and descent of the limbs of the R waves are seen, especially in the second lead. The same blunt, notched wave is seen in the first lead, while the third lead complex is distinctly peculiar. The patient died suddenly a few days after the record was obtained, following the intravenous administration of strophanthin. It seemed not unlikely that ventricular fibrillation resulted from the administration of the drug.

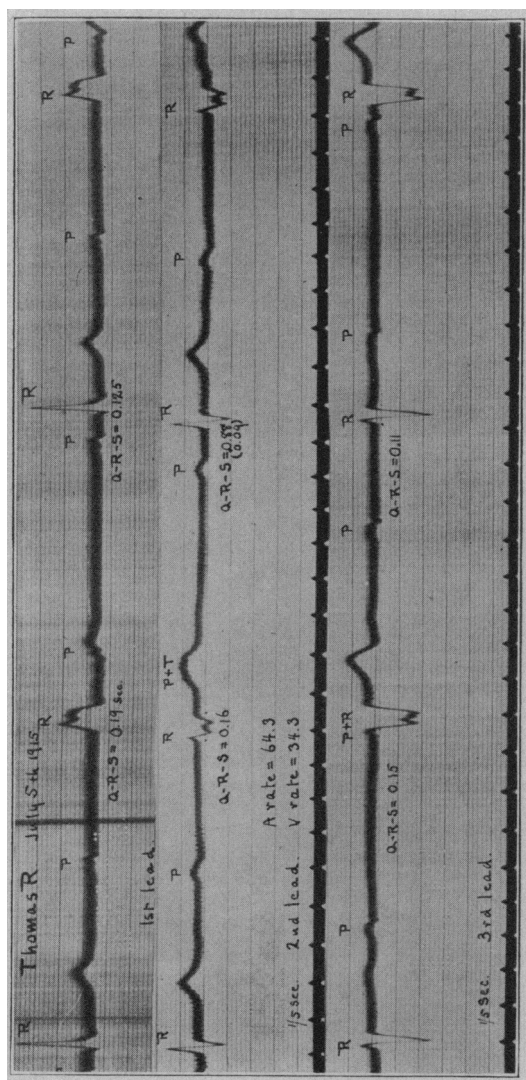


Fig. 8 (Case 4).—July 5, 1915. The form of the complexes alternate; first the usual form and then the form that appeared nine days previously.

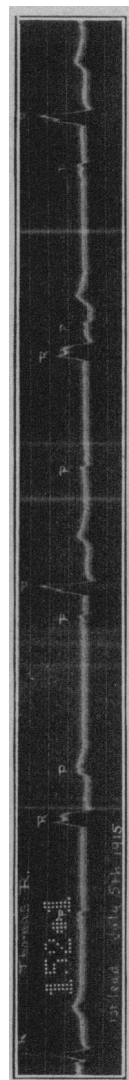


Fig. 9 (Case 4).—July 5, 1915. Transition between the old and the new forms of the first lead complexes.

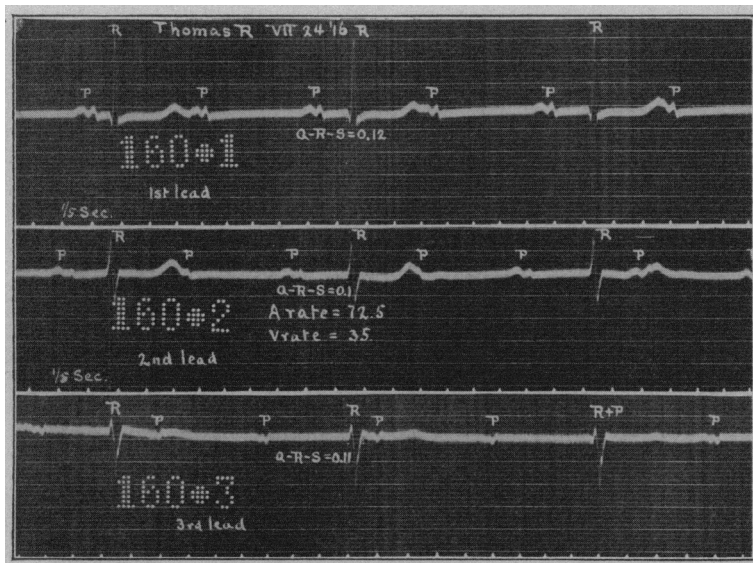


Fig. 10 (Case 4).—July 24, 1915. The form of the ventricular complexes is constantly that seen before the marked change occurred. No further change occurred.

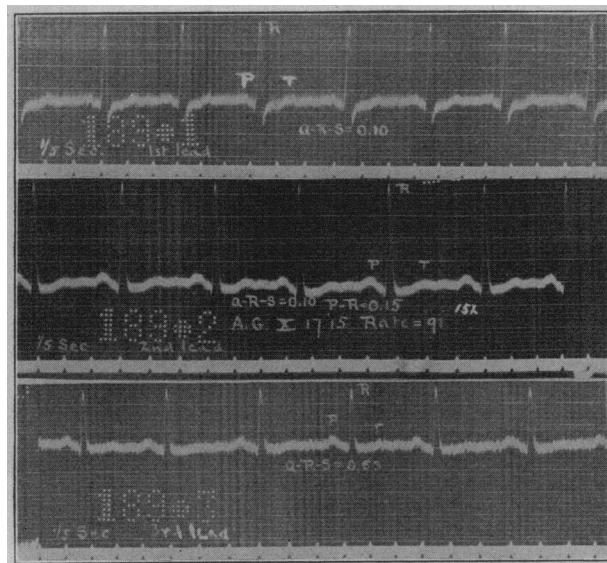


Fig. 11 (Case 5).—Oct. 17, 1915. Cardiac efficiency but slightly impaired.

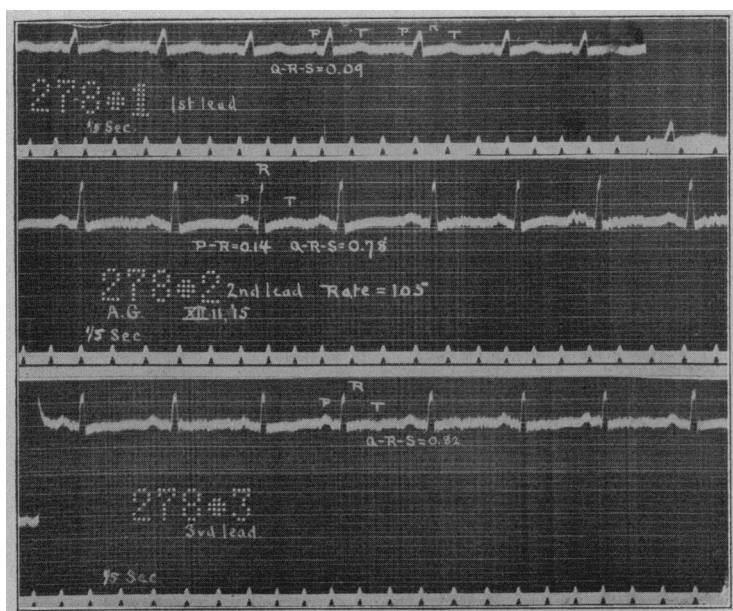


Fig. 12 (Case 5).—Dec. 11, 1915. Cardiac efficiency much impaired.

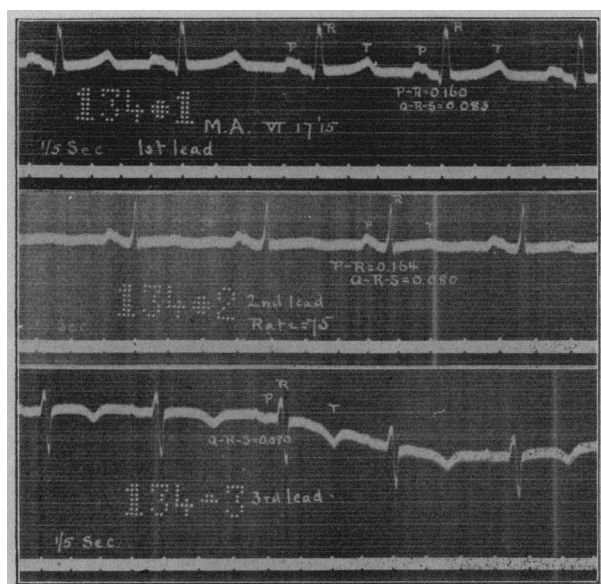


Fig. 13 (Case 6).—June 17, 1915. Marked signs of cardiac decompensation.

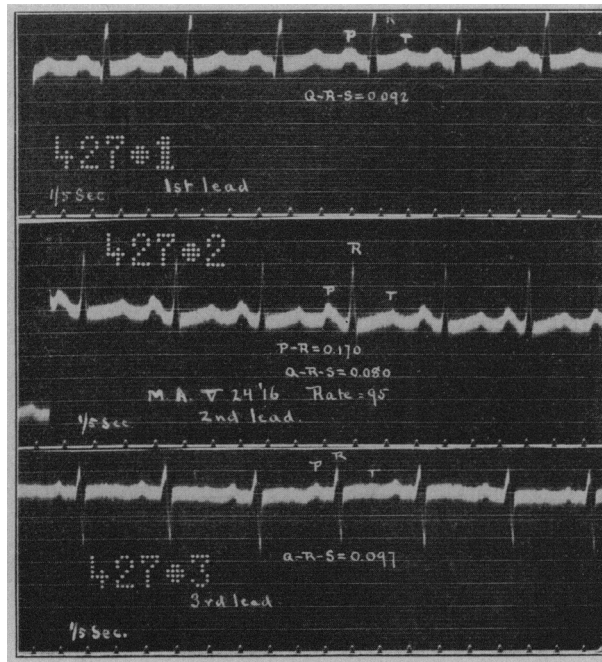


Fig. 14 (Case 6).—May 24, 1916. Patient much improved; cardiac decompensation slight.

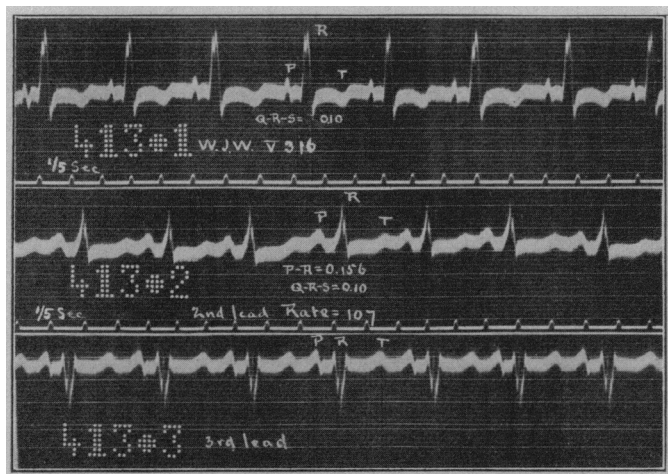


Fig. 15 (Case 7).—May 3, 1916. Patient showed signs of extreme cardiac decompensation.

COMMENT

The idea that changes in form of the ventricular complexes may depend on faulty conduction through the ventricles of impulses descending from the auricles was first suggested by Lewis.⁹ He based his hypothesis on records obtained under experimental conditions, as well as on electrocardiograms from clinical material, and developed his idea further in a second paper.⁸ He points out the association of prolonged auriculoventricular conduction with the so-called aberrant contractions, and suggests that abnormal complexes may depend on damage affecting special branches of the intraventricular conducting system. Under such conditions the impulse is at one time transmitted through the whole arborization, while at other times it passes into the arborization, but fails to course along certain given channels.

Hart¹⁰ has also recognized derangement of intraventricular conduction as a cause of contractions yielding abnormal ventricular complexes. He shows records in which the Q, R, S group of the complex is deformed in a manner somewhat similar to deformities described in this paper and he believes that the abnormalities are caused by damage to the musculature, so that the passage of impulses is interfered with in all parts of the conducting system.

The occurrence of varying ventricular complexes in cases of complete heart block has been reported by Cohn¹¹ and by Oppenheimer and Williams.¹² These cases have been interpreted as examples of either a shifting focus of stimulus formation within the ventricles or as interference with intraventricular conduction. The changes in form of the ventricular complexes of the case reported here are considered as dependent on interference of ventricular conduction.

The purpose of this paper is not primarily to record a series of cases showing derangement of intraventricular conduction. This change in function is discussed only as affording an explanation of the changes in form of the ventricular complexes which have been observed. The object of the paper is to point out certain abnormalities occurring in electrocardiographic curves which apparently accompany functional deficiency of the ventricles. This is done with the hope that the electrocardiographic method may sometimes prove of value in determining

9. Lewis: *Galvanometric Curves Yielded by Cardiac Beats Generated in Various Areas of the Auricular Musculature*, *Heart*, 1910, ii, 23.

10. Hart: *Paroxysmal Tachycardia*, *Heart*, 1912, iv, 128.

11. Cohn: *A Case of Transient Complete Auriculoventricular Dissociation, Showing Constantly Varying Ventricular Complexes*, *Heart*, 1913, v, 5.

12. Oppenheimer and Williams: *Prolonged Complete Heart Block, Without Lesion of the Bundle of His and with Frequent Changes in the Idioventricular Electrical Complexes*, *Proc. Soc. Exper. Biol. and Med.*, 1913, x, 86.

changes in the functional capacity of the ventricular musculature, perhaps the most important and one of the most obscure problems involved in the study of the heart.

Only a much wider experience and a collection of many such cases as those exemplified in this paper can determine the value of these observations from the purely clinical point of view.

SUMMARY

A series of cases is reported which yielded electrocardiograms showing abnormal ventricular complexes. These abnormalities consist in changes in the initial portion of the complexes, the Q, R, S group, and differ from those yielded by contractions caused by ectopic stimuli and from those changes which occur with bundle-branch block.

These abnormalities are apparently dependent on derangement of the intraventricular conduction, which prevents the passage of the excitation wave either along the usual paths or at the usual rate throughout the ventricles.

The normal spread of the impulse is hindered because the impulse reaches the ventricles before the conducting system has recovered from the preceding contraction, and the records indicate in some cases that this derangement disappears with prolonged ventricular rest.

These observations are taken as evidence for the belief that in cases in which the ventricular complexes constantly show certain abnormal forms, there are functional changes in the heart which prevent the normal recovery of intraventricular conduction during diastole. It is shown that changes in form of the ventricular portions of the electrocardiogram may occur synchronously with functional changes in the heart, and evidence is offered for the belief that certain abnormalities in the form of the electrocardiogram indicate functional derangement of the ventricles.