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THE POTENTIAL VARIATIONS OF THE THORAX AND THE ESOPHAGUS IN ANOMALOUS ATRIOVENTRICULAR EXCITATION (WOLFF-PARKINSON-WHITE SYNDROME)

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INTRODUCTION

IN 1930, Wolff, Parkinson, and White[1] reported a group of cases characterized by the following features: (1) the occurrence of paroxysms of tachycardia, heterotopic in origin; (2) complete absence of physical signs of heart disease when the heart rate was normal; (3) electrocardiographic peculiarities, of which the most striking were abnormal shortening of the P-R interval and a pronounced increase in the duration of the QRS complex; (4) reversion of the anomalous electrocardiogram to the normal form either spontaneously, after exertion, or after the administration of atropine. Isolated cases which seem to have been of a similar kind had previously been reported by Wilson,[2] Wedd,[3] and Hamburger.[4] In 1940, Hunter, Papp, and Parkinson[5] were able to find ninety cases of this type in the literature and to add to these nineteen cases which they had collected. This condition has been called the Wolff-Parkinson-White syndrome, but in order to avoid awkward forms of expression we shall more often refer to it as anomalous atrioventricular excitation.

A number of different hypotheses have been advanced to account for the peculiarities of cardiac mechanism which make this disorder unique. These hypotheses have recently been reviewed and classified by Hunter and his associates, and it is fair to say that none of them satisfactorily

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explains the syndrome in its entirety. The most promising and most widely accepted view was put forward independently by Holzmann and Scherf[6] and by Wolferth and Wood.[7] They suggested that the short P-R interval and the broad QRS complex are due to the transmission of impulses from auricles to ventricles by way of an accessory atrioventricular bundle, a strand of muscle of the sort described originally by Kent.[8] Quite recently, this conception has been supported by two important studies. In experiments on animals, Butterworth and Poindexter[9] passed action currents picked up from the auricular surface through a vacuum-tube amplifier and utilized the output to excite the ventricles. In this way they were able to obtain electrocardiograms strikingly similar to those seen in human cases of anomalous atrioventricular excitation. By reversing the connections and applying amplified ventricular action currents to the auricles they were also able to induce paroxysms of tachycardia simulating those often observed in this syndrome. Wood, Wolferth, and Geckeler[10] have reported a careful histologic search for muscular bridges between the auricular and the ventricular myocardium in a case of anomalous atrioventricular excitation in which death occurred during an attack of paroxysmal tachycardia. Three connections of this kind were found on the right side of the heart.

These studies are of very great importance, but they must be regarded as suggestive rather than decisive. The experiments of Butterworth and Poindexter[9] demonstrated that excitation of the epicardial surface of the ventricles by the action currents of adjacent auricular muscle, or inferentially by the transmission of auricular impulses across an accessory atrioventricular bundle, could account for the brevity of the P-R and the abnormal length of the QRS interval, and also suggested a way in which a physiologic or anatomic anomaly of this sort might lead to paroxysms of tachycardia. Nevertheless, they left many questions relating to these phenomena unanswered. Muscular bridges of the kind found by Wood and his co-workers[10] were originally described by Kent,[8] and have recently been observed by Glomset and Glomset[11] in hearts that were presumably normal. It would appear, therefore, that human hearts in which they can be found are much more numerous than those that exhibit anomalous atrioventricular excitation. This consideration raises doubt as to their significance.

In view of this situation, it seemed desirable to ascertain whether unipolar precordial and esophageal leads, which have proved of great value in the study of other abnormalities of the ventricular complex, would yield data consistent with the hypothesis in question.

CLINICAL OBSERVATIONS

We have had the opportunity of studying ten cases of anomalous atrioventricular excitation which were discovered in the course of routine electrocardiographic examination or referred to us for investigation.

Brief abstracts of the case histories are presented below. The electrocardiographic data will be considered separately.

CASE 1.—A schoolboy, aged 13 years and of somewhat deficient intelligence, entered the hospital Oct. 7, 1942, for the correction of convergent strabismus which had been present since infancy. Along the left margin of the sternum there was a moderately loud, rough, systolic murmur, but the heart was not enlarged; the blood pressure was normal, and there were no cardiac symptoms. A corrective operation on the eyes was performed October 29, and the patient's convalescence was uneventful.

CASE 2.—A male professor, aged 36 years, came in for a checkup examination on Feb. 14, 1942. He had no complaints referable to the heart and appeared to be in good health. Soft systolic murmurs were heard at the cardiac base and apex, but there was no enlargement of the heart either on physical or roentgenologic examination. Apart from the anomalous electrocardiogram, no abnormalities of any sort were discovered. Late in July, 1942, this man was found dead in his automobile, which was standing at the side of the road. He was known to have been normally active a few days prior to his death. The results of an autopsy carried out by the coroner could not be ascertained.

CASE 3.—A male storekeeper, 34 years of age, entered the hospital March 20, 1942, complaining of occipital headaches for the preceding four years, and of mild dyspnea on exertion and slight edema of the ankles for several months. During the preceding three years he had been subject to paroxysms of tachycardia, forty-five minutes to four hours in duration. The blood pressure was 186/116; there was slight edema of the ankles, and the heart was slightly enlarged to the left. The urine contained albumin and granular casts, and renal function was depressed (urea clearance, 29 and 22 per cent of normal). There was no improvement on a conservative regime, and a bilateral splanchnicectomy was performed Aug. 31, 1942. Twelve days later the patient was discharged; at this time the blood pressure was 105/55. He returned for a checkup examination on Aug. 16, 1943, and reported that he had had a few attacks of tachycardia, but was working regularly. The blood pressure was then 162/120; apart from the absence of edema, the physical signs were not notably different from those found on previous occasions. There was, however, a change in the electrocardiogram; the T deflections, previously inverted in precordial leads V_4, V_5, and V_6, had become upright. It has been observed that the inverted T waves often seen in hypertensive heart disease frequently return to normal after operations of the kind performed on this patient.

CASE 4.—A foundry worker, 30 years old, entered the hospital Nov. 11, 1942, complaining of attacks of nocturnal dyspnea and palpitation. Three attacks of this sort had occurred during the preceding three months. The duration of the paroxysms varied from ten to fourteen hours, and attempts to prevent them by the administration of digitalis and quinidine had not been successful. There was a moderately loud, apical, systolic murmur, but the heart was not enlarged either on physical or roentgenographic examination, and the blood pressure was normal. The rest of the physical examination and the routine laboratory tests were negative. The administration of quinidine, 0.2 Gm. three times daily, was advised, but this treatment failed to prevent occasional paroxysms of tachycardia.

CASE 5.—A young man, aged 34 years, was referred to the William J. Seymour Hospital (Eloise, Mich.) on July 30, 1941, for examination in connection with the Selective Service program. He presented no cardiac symptoms. The blood pressure was 190/120, and there were mild changes in the retinal arteries of the kind often associated with arterial hypertension. The heart was not definitely enlarged either on physical or roentgenographic examination. The remainder of the physical examination was negative.

CASE 6.—A male physician, aged 28 years, who was attached to the Heart Station, was found to have an anomalous tracing when he was used as a subject in the course of a test of some electrocardiographic equipment. He was subject to renal glycosuria, but was otherwise well, and physical and roentgenographic examination of the heart was negative.

CASE 7.—A male laborer, 37 years old, entered the hospital June 9, 1934, complaining primarily of joint pains associated with swelling and limitation of motion. For about three years he had also been subject to paroxysms of tachycardia lasting from twelve to twenty-four hours. These were accompanied by mild dyspnea, slight precordial distress, and occasional choking sensations. The heart was not enlarged, but roentgenographic examination disclosed slight widening and tortuosity of the thoracic aorta and minor prominence of the pulmonary artery. Frequent extrasystoles were noted, but no murmurs were heard, and the blood pressure was normal. The knees, elbows, and wrists showed changes characteristic of chronic atrophic arthritis, and there was some hypertrophic arthritis of the spine. The patient was under treatment for a considerable period, during which a number of paroxysms of tachycardia, supraventricular in origin, were observed. These were successfully treated with acetyl-β-methylcholine chloride and with quinidine. The regular administration of the latter reduced the frequency of the attacks.

CASE 8.—A female clerk, aged 24 years, requested an examination on July 29, 1943. She had been rejected for service with the Armed Forces on account of a cardiac murmur and arrhythmia. Examination disclosed a fairly loud, late systolic, apical murmur and an inconstant systolic click. Sinus arrhythmia and occasional extrasystoles were noted. The blood pressure was normal, and the remainder of the physical examination was negative.

CASE 9.—A male office worker, 25 years old, was examined Oct. 6, 1941, with reference to frequent attacks of rapid heart action during the preceding ten years. One of the most recent of these had persisted for thirty hours. Physical examination of the heart was negative, and the blood pressure was normal. Roentgenographic examination of the chest and the routine laboratory tests gave no further information.

CASE 10.—A housewife, aged 48 years, was admitted to the William J. Seymour Hospital (Eloise, Mich.) because of an involutionary psychosis early in February, 1941. She gave a history of paroxysms of tachycardia, but had no other symptoms referable to the heart. No cardiac abnormalities were discovered on examination, and the blood pressure was normal. This patient is still under observation; her mental condition has gradually deteriorated.

In summarizing the clinical aspects of these ten cases of anomalous atrioventricular excitation we may mention that all of the patients were under 50 years of age, and that all except two were males. Three ex-

hibited anomalies other than that involving the heart; we refer to the presence of renal glycosuria in Case 6, of mental deficiency and strabismus in Case 1, and of an involutional psychosis in Case 10. Half of the patients were subject to paroxysms of rapid heart action. Clinical evidence of structural heart disease was found in only one instance, in which it was associated with arterial hypertension. One other patient had an abnormally high blood pressure (Case 5), one had chronic atrophic arthritis (Case 7), and a third died suddenly and unexpectedly from an unknown cause (Case 2).

ELECTROCARDIOGRAPHIC OBSERVATIONS

Material.—The standard limb leads and unipolar* precordial leads from the six standard precordial points (Leads V_1 to V_6, inclusive) were taken in all ten of the cases upon which this report is based. Unipolar leads from the tip of the ensiform process (V_E) were taken in eight cases, multiple unipolar leads from the back and right side of the chest in five, and multiple unipolar leads from the esophagus in four. The esophageal leads were taken in the manner described by Nyboer,[12] and the unipolar limb leads according to Goldberger's technique.[13]

The analysis of our records would have been easier if we had taken all of the leads mentioned in every instance. Sometimes we did not do this because the time and length of the patient's visit did not offer the opportunity. More often, however, a number of these leads were not taken because the problems which prompted us to employ them at a later stage of our work had not yet presented themselves.

The Working Hypothesis and Its Implications.—We accepted, as a working hypothesis, the view that in cases of the kind under consideration auricular impulses reach the ventricles by way of an accessory atrioventricular bundle. If this is the case, it is clear that the order of ventricular activation during the first part of the QRS interval must depend to a considerable extent upon whether the ventricular muscle in which this bundle terminates lies on the inner or on the outer aspect of the ventricular wall. It must also be acknowledged that, if the existence of one anomalous tract of this kind is admitted, we cannot dismiss the possibility that two or more may exist. There are, moreover, reasons for supposing that, even if other atrioventricular bridges are present, the His bundle continues to function.

These considerations make the interpretation of the ventricular deflections which depict anomalous atrioventricular excitation particularly difficult. The situation is much more complicated than those encountered in the analysis of the curves that represent bundle branch block, ventricular hypertrophy, and myocardial infarction. In these conditions the cardiac impulse reaches the ventricles by way of the His

*The term *unipolar* is used to indicate that the exploring electrode was paired with a *central terminal* connected through resistors of 5,000 ohms to each of the extremity electrodes employed in taking standard limb leads. For practical purposes it may be assumed that the potential of a central terminal of this sort is not affected by the heart beat; or what amounts to the same thing, that it is zero throughout the cardiac cycle.

bundle and spreads through the ventricular walls from within outwards. The various types of QRS complexes inscribed in unipolar precordial leads under these circumstances may be interpreted with confidence because the principles involved have been established by recording and comparing the potential variations of the epicardial surface, the ventricular cavities, and the precordium in experiments on animals. We cannot, however, assume that a QRS pattern which has a known significance when ventricular excitation takes place in the normal fashion must have the same significance when auricular impulses are transmitted to the ventricles along anomalous paths.

Previous observers have found that when anomalous and normal beats are recorded in the same tracing, the sum of the P-R and the QRS interval is the same, or very nearly the same, for both. This suggests that the broad QRS complexes represent premature anomalous activation of the ventricular muscle, combined with normal activation by way of the His bundle. Assuming that this is true, we must conclude that, in relation to auricular events, some fraction of this muscle is activated earlier, but none can be activated later, than would be the case if the cardiac impulse reached the ventricles by way of the His bundle only. We may, then, refer to that part of the anomalous QRS complex which encroaches upon the normal P-R interval as the premature component. The point at which this component ends cannot be ascertained with certainty unless normal ventricular complexes have been recorded on the same tracing. In other cases we may consider that this point falls about 0.08 to 0.10 second ahead of the RS-T junction. We may also speak of the anomalous QRS complex as consisting of an anomalous component and a normal component. The former, which represents action currents produced by muscle activated by way of an aberrant pathway, is in part premature and in part superimposed upon the latter, which represents the action currents of muscle activated by the normal route. It has been observed that the premature component, that is to say, the premature part of the anomalous component, is almost invariably of relatively low voltage and displays no steep slopes. In the majority of cases it is fused with the first part of the succeeding fraction of the QRS complex, and gives rise to basal slurring or notching of the earliest prominent QRS deflection. The size and character of this premature component have been interpreted as evidence that when the aberrant impulse first reaches the ventricular muscle it spreads slowly and does not immediately gain access to the Purkinje plexus. One of the principal objects of our investigation was to ascertain, if possible, the location of the muscle activated prematurely by an anomalous route.

The Type Case of Group A.—Depending on the form of the ventricular complex in precordial leads, we have divided our cases into two groups, A and B. Case 1 is the type case of the first group. In this instance, reversion of the anomalous to the normal type of electrocardiogram sometimes occurred spontaneously and could be induced by the ad-

ministration of amyl nitrite. Transitions from excitation of the one sort to excitation of the other were recorded in a variety of leads. In the standard limb leads the normal beats (labeled *n*) are represented by deflections of the kind often seen in the electrocardiograms of healthy young subjects (Fig. 1). In Lead I the QRS complex displays relatively small R and S components, and in Leads II and III it consists of a small Q wave followed by a tall R wave. The mean electrical axis of this complex has a nearly vertical direction, suggesting that the angle made by the long axis of the heart with the long axis of the trunk was a small one. The T waves are inverted in Lead III, as is often the case in electrocardiograms of this type. The anomalous beats (labeled *a*) are represented by patterns of very different form. Apart from the brevity of the P-R and the broadening of the QRS interval, there is conspicuous basal slurring of the first QRS component, and the mean electrical axis of QRS is nearly horizontal. The T deflections are inverted in Lead I. Normal complexes were recorded in only one of the unipolar limb leads, Lead V_F; in this lead they have the same outline as in Leads II and III. The anomalous complexes of Lead V_R show pronounced slurring of the first part of the large initial Q wave, and in Lead V_L the initial R deflection is deformed in a similar way.

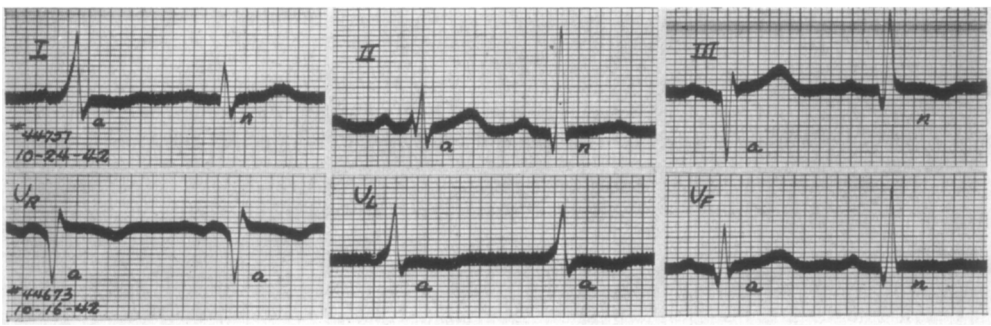

Fig. 1.—Case 1. Standard and unipolar limb leads. Complexes which represent anomalous atrioventricular conduction are labeled *a*, and complexes of the normal type are labeled *n*.

In Fig. 2, five unipolar precordial leads, a unipolar lead from a point overlying the spinal process of the eighth dorsal vertebra (D_{VIII}), and unipolar leads from four levels of the esophagus are reproduced. In the precordial leads the normal beats (labeled *n*) are represented by deflections of the usual type. In Leads V_1 and V_2 the R deflection is small, the R peak falls early in the QRS interval, and S is large; in Lead V_5 the R deflection is large, the summit of this deflection comes later, and small Q and S waves are present. In Leads V_3 and V_E the R and S deflections are approximately equal in size; we may speak of these leads as from the transitional zone. In all of these precordial leads the QRS complex of the anomalous beats (labeled *a*) is dominated by an R deflection which is considerably taller than the R wave of the normal beats and displays pronounced slurring of the basal part of its ascending

limb. The S component of the anomalous and that of the normal QRS complex are largest in the same lead (V_2). The former is small in the leads from the left side of the precordium and the lead from the ensiform process. In Lead V_1 a broad, bifid R wave is the only QRS component. The Q deflection of the normal complex of Lead V_5 does not occur in its anomalous companion. The differences between the T waves of the two kinds of ventricular complexes are as great as the differences between the QRS deflections. The long Q-T interval in Lead V_1 seems to be due to the fusion of a large U wave with the terminal part of T.

In the lead from the auricular level of the esophagus (Lead E_{29}), the difference in length between the P-R interval of the anomalous and that of the normal beats is especially conspicuous. The auricular and ventricular complexes of the latter are of the kind usually seen in unipolar leads from this region. The anomalous complexes are similar in general outline, but the QRS interval is much longer, the descending limb of QS has a much more gradual slope, and the T wave is upright. In the esophageal lead from a point 6 cm. farther from the nares (Lead E_{35}), the initial, slurred part of the QRS complex is still below the isoelectric line, whereas, in the leads from still lower levels (Leads E_{41} and E_{51}), this premature component is positive. The normal ventricular complexes of these last leads are very similar to, and the anomalous complexes very different from, those of the same species in Lead V_5. The deflections of Lead E_{51} are like those of Lead E_{41}, except that the anomalous beat displays a conspicuous R′ which follows the onset of P by approximately the same interval as the R summit of the normal beat. When the long axis of the heart occupies a relatively vertical position, leads from these levels of the esophagus (12 cm. or more below the level

TABLE 1

CASE 1. INTERVALS IN FIG. 2. MEASUREMENTS IN SECONDS

LEAD	1		2		3		4	
	a	n	a	n	a	n	a	n
V_1	.093	.124	*{.138 .171	.139	–	.161	.191	.199
V_2	.101	.142	.166	.166	.188	.186	.220	.225
V_3	.096	.145	.172	.171	.202	.196	.220	.196
V_5	.085	.110	.153	.149	.175	.170	.195	.196
V_E	.097	.147	.181	.182	.203	.198	.225	.220
D_{VIII}	.068	.132	–	.173	.152	.153	.209	.199
E_{29}	.059	.128	–	.191	.154	.159	.190	.191
E_{35}	.071	.121	–	.166	.142	.142	.201	.183
E_{41}	.105	.135	.131	.174	.154	†{.147 .198	.207	.198
E_{51}	.117	.135	*{.139 .180	.175	‡{.162 .202	†{.147 .202	.220	.214

Key:

a—anomalous; n—normal.
Column 1—interval from beginning of P to beginning of QRS.
Column 2—interval from beginning of P to peak of R.
Column 3—interval from beginning of P to peak of Q, QS, or S.
Column 4—interval from beginning of P to end of QRS.

*Two R peaks present.

†First measurement to peak of Q, second to peak of S.

‡Two S peaks present.

where large diphasic or multiphasic P complexes are obtained) ordinarily yield ventricular deflections like those of leads from the left side of the precordium, and may, therefore, be considered semidirect leads from the surface of the left ventricle. It will be noted that the complexes of the unipolar dorsal lead (Lead D_{viii}) closely resemble those of Lead E_{35}.

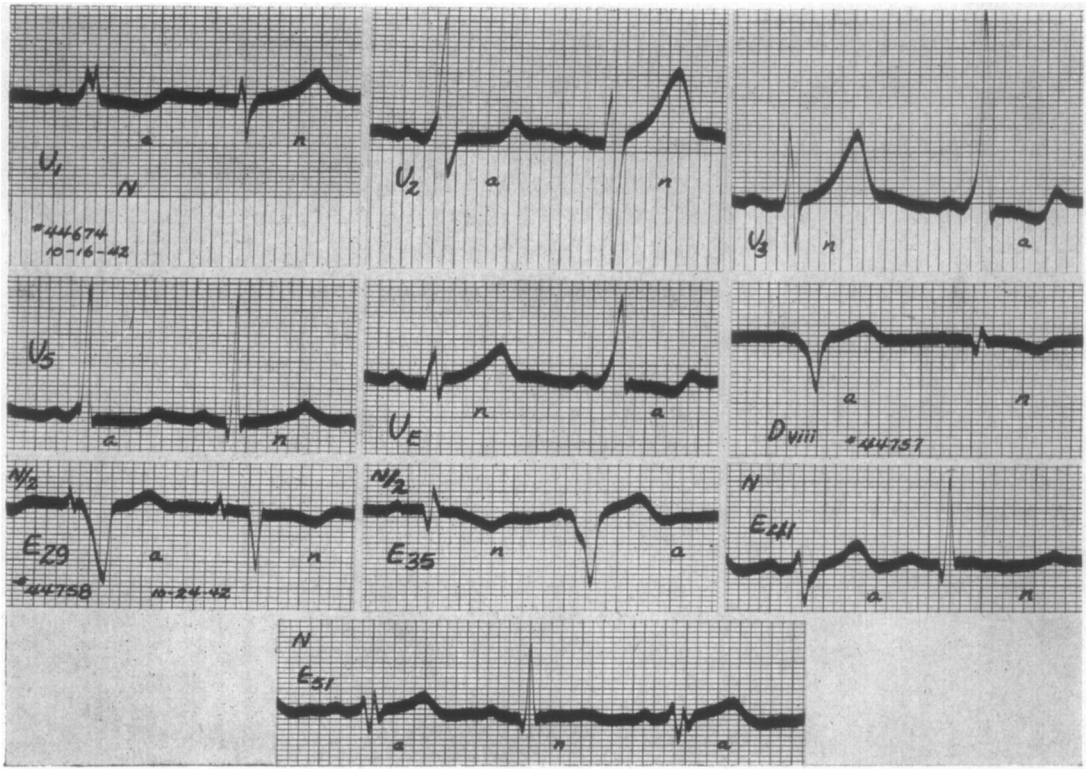

Fig. 2.—Case 1. Precordial Leads V_1, V_2, V_3, V_5, and V_E. A unipolar lead from the region of the eighth spinal process (D_{viii}). Four unipolar esophageal leads; these leads are labeled E, followed by a number which gives the distance (in centimeters) of the exploring electrode from the nares. Complexes labeled *a* are anomalous, those labeled *n*, normal. In this and in subsequent figures the symbol *N* indicates that the lead was taken with the electrocardiograph at the normal sensitivity (1 cm. equals 1 mv.) the symbol *N/2* indicates that the sensitivity was reduced to one-half the normal (1 cm. equals 2 mv.).

Despite these great differences in form between the normal and the anomalous ventricular complexes, measurements show that in many leads the two types of QRS groups are structurally related. We have already mentioned that the interval from the beginning of the P wave to the end of the QRS complex (the RS-T junction) is of the same length when atrioventricular excitation is anomalous as when it is normal. Measurements of the curves of Fig. 2 are in accord with this statement, and also show that the corresponding peaks of the chief QRS components of the two kinds of curves usually occur at approximately the same time in relation to the P wave. Table I gives, for each of the leads shown in Fig. 2, the intervals from the onset of P to (1) the onset of the initial

QRS component, (2) the peak of R, (3) the peak of the chief downward deflection (Q, QS, or S), and (4) the RS-T junction.

This table indicates that the anomalous P-R interval is certainly more than 0.02, and probably more than 0.05, second shorter than the normal P-R interval in this case. The largest difference was found in the lead from the auricular level of the esophagus, where it amounted to nearly 0.07 second. In Leads V_2, V_3, V_5, and V_E the R peaks of the paired complexes occur at the same time, within a few thousandths of a second, in relation to auricular events. In Lead V_1 the first peak (0.138) of the bifid R of the anomalous complex corresponds to the R summit (0.139) of the normal complex. In Lead E_{41} the R peaks of the two kinds of complexes do not correspond (0.131 and 0.174), and evidently differ in origin. In Lead E_{51} the normal R deflection corresponds in time not to the initial R, but to R′ of the anomalous complex. The paired intervals of the third column of Table I, which give the times of the apices of the largest negative QRS deflections, are in good agreement. In the case of Lead E_{41}, the apex of the anomalous S (0.154) corresponds more nearly to the apex of the normal Q (0.147) than to that of the normal S (0.198). The paired intervals of the last column, which give the time of the RS-T junction in relation to the beginning of P, are also alike except in two or three instances (Leads D_{VIII}, E_{35}, and E_{41}), in which the end of one or both of the QRS complexes is poorly defined.

These measurements clearly support the view that the excitatory process reached the epicardial surface of the anterior wall of the left ventricle at the normal time (in relation to auricular events) and by the normal route, even when some parts of the ventricular myocardium were activated prematurely by an anomalous mechanism. We cannot regard it as fortuitous that the R peak of the anomalous and that of the normal QRS complex of the leads from the left side of the precordium are separated from the beginning of the P wave by the same interval. It is clear, then, that the premature component of the anomalous QRS complex of these leads cannot be ascribed to forces produced by premature excitation of the anterior wall of the left ventricle. As regards the significance of this component in the leads from the right side of the precordium, the situation is similar. There is no evidence that the anterior wall of the right ventricle was activated prematurely. In Leads V_2 and V_E the single R peak of the anomalous, and that of the normal, beat bear the same relation to the P wave. This is likewise true of the first R summit of the anomalous, and the R peak of the normal, QRS complex in Lead V_1. The second R summit of this lead, which is somewhat like that seen in right bundle branch block, cannot be attributed to activation of the anterior wall of the right ventricle unless we suppose that the cardiac impulse reached the epicardial surface in this region abnormally late. This supposition would imply that the

right branch of the His bundle was not functioning, and is not supported by the character of the ventricular complexes of the other leads.

A complete set of the esophageal leads, taken when the cardiac mechanism was continuously anomalous, is reproduced in Fig. 3. The premature component of QRS is inconspicuous in Lead E_{15}; in Leads E_{18}, E_{21}, E_{24}, E_{27}, E_{29}, and E_{33} it is negative, and in the last four of these leads it is conspicuously large. The brevity of the P-R interval in the leads in which this component is largest is apparently due chiefly to the comparatively large magnitude of its earlier fractions; in some of the other esophageal leads these are inconspicuous or isoelectric. In Leads E_{36}, E_{39}, E_{42}, E_{45}, and E_{51} the premature component is positive and relatively small. It will be observed that the QRS complexes of the leads from the highest levels of the esophagus are the inverse of QRS complexes of the leads from the lowest levels. We have already noted that the latter display both an R and an R′ deflection, and are very different in form from the normal complexes of the same leads and from the anomalous complexes of the leads from the left side of the precordium.

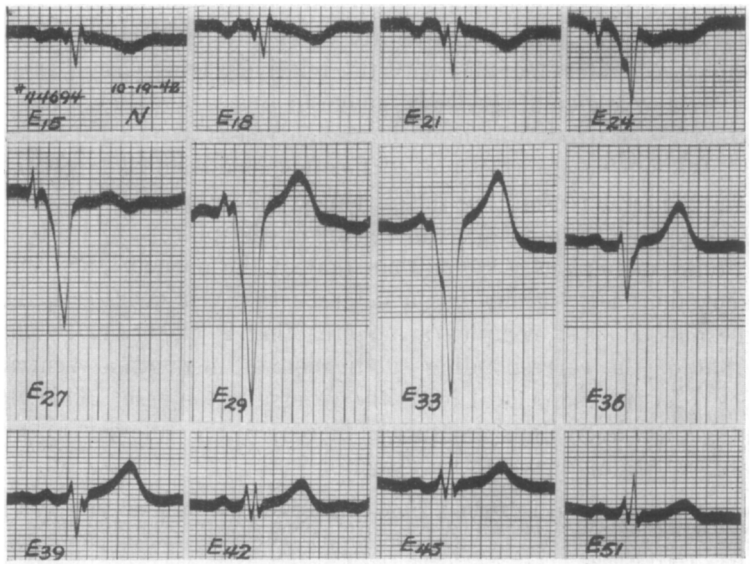

Fig. 3.—Case 1. Esophageal leads. The number which follows *E* gives the distance (in centimeters) of the exploring electrode from the nares.

Unipolar leads from the back and the anterolateral aspect of the right side of the thorax are shown in Fig. 4. The premature component of QRS is clearly negative in the leads from the eighth dorsal spine (D_{viii}), the right posterior axillary line (RPAL), the right midaxillary line (RMAL), and the right anterior axillary line (RAAL). It is clearly positive in the leads from the left midaxillary line (V_6), the left posterior axillary line (LPAL), the left scapular line (LScL), and a line midway between the right sternal margin and the right midclavicular

line (V_{3R}).* In the lead from the right midclavicular line (RMCL) the premature component is isoelectric. All of these leads were taken from points at the level of the cardiac apex.

In this instance the muscle activated prematurely must have been in the dorsal wall of the heart near the ventricular base, or in the neighboring part of the ventricular septum. This conclusion is supported by the following considerations:

a. The orientation of the electrical forces generated by the heart during the premature fraction of the QRS interval indicates that throughout that period the excitatory process was spreading from the dorsal toward the ventral, and from the basal toward the apical, parts of the myocardium. In this part of the cardiac cycle the potential of the auricular and subauricular† levels of the esophagus and that of a zone extending from the eighth dorsal spine around the right side of the chest to the right anterior axillary line were negative, whereas the potential of the ventricular levels of the esophagus and that of a zone extending from the right parasternal line across the precordium and around the left side of the chest to the left scapular line were positive.

b. The earliest fractions of the premature component of QRS are most conspicuous in the leads from the auricular and subauricular levels of the esophagus. This, together with the relatively large size of this component as a whole in these leads, suggests that when they were taken the exploring electrode was near the region where premature activation began. We believe, in other words, that this component is large in these leads for the same reason that the auricular deflections are large in them.

c. It has been pointed out that in all of the precordial leads the R wave of the anomalous is taller than that of the normal QRS complex. The anomalous QRS group has a net area that is algebraically larger, and the anomalous T complex a net area that is algebraically smaller, than that of the corresponding subdivision of the normal ventricular complex. In the esophageal leads the reverse is the case. This clearly indicates that anomalous excitation increased the number of muscle units activated in a dorsoventral direction.

*Leads from points on the right side of the chest similar in location to the points from which the standard precordial leads are taken are conveniently differentiated from these by adding R to the subscripts of the standard symbols of the leads to which they correspond.

In normal subjects and in cases of right ventricular hypertrophy, left ventricular hypertrophy, right bundle branch block, and left bundle branch block, the ventricular complexes of unipolar leads from the left posterior axillary line and the left scapular line (at the level of the cardiac apex) are usually similar to those of the leads from the left side of the precordium. Exceptions to this general rule occur in those cases in which the transitional zone is displaced to the left. In these the leads from the left side of the precordium display complexes intermediate in form between those of the leads from the right side of the precordium and those of the leads from the left back. The ventricular complexes of the latter are then like those usually seen in Leads V_5 and V_6 in the type of heart disease present. As a general rule the ventricular complexes of the leads from more lateral parts of the right anterior chest wall are similar to those of Lead V_1; exceptionally, they are like those usually present in this lead in cases of the kind being studied. The deflections of the leads from the right back are variable in form, but often resemble those of Lead V_R. The QRS complex is ordinarily minus-plus diphasic. In right ventricular hypertrophy the second phase usually has the greater voltage, and in left ventricular hypertrophy the first phase usually has the greater voltage. In right bundle block the second component is often very broad. In left bundle branch block the first or negative phase is usually the larger, and the second or positive phase may be absent.

†Less than 10 cm. below the point at which the largest auricular deflections were recorded.

d. The form of the QRS deflections in the leads from the lower subauricular and the higher ventricular levels of the esophagus suggests that the parts of the dorsal ventricular wall nearest the exploring electrode were activated earlier when excitation was anomalous than when it was normal (Fig. 2). In Lead E_{35} the normal QRS group displays a final R deflection; in the anomalous beat, R is wholly lacking. In Lead E_{41} the normal beat exhibits a prominent Q and a late R peak, whereas in the anomalous complex R is small and its peak falls in the premature part of the QRS interval (Table I). In Lead E_{51} the situation is similar except that here the anomalous QRS complex has an R′ in addition to the initial R wave. This R′ summit comes at the same time as the normal R peak of the same lead. Fig. 3 shows that it is embryonic in Lead E_{39} and progressively larger in the leads from lower levels, which suggests that it represents the response of some of the lowest portions of the dorsal ventricular wall to the normal excitation wave. The leads from the left back (Fig. 4, LPAL and LScL) exhibit QRS deflections of similar form, whereas the QRS complex of these leads is normally dominated by a late R deflection.

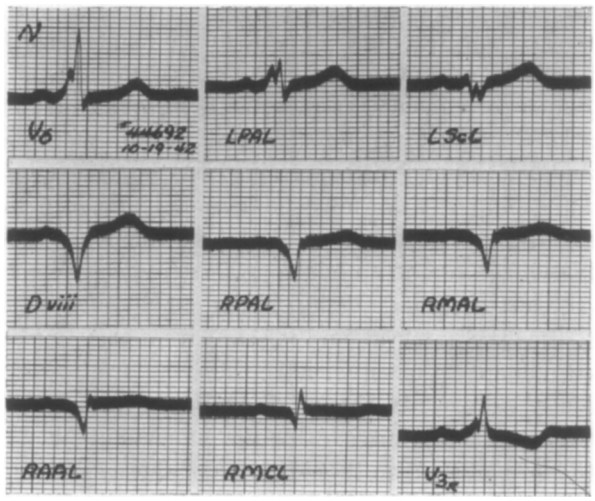

Fig. 4.—Case 1. Unipolar leads from the back and from the anterior right hemithorax at the level (approximately) of the cardiac apex. *LPAL*, left posterior axillary line; *LScL*, left scapular line; D_{VIII}, eighth dorsal spine; *RPAL*, right posterior axillary line; *RMAL*, right midaxillary line; *RAAL*, right anterior axillary line; *RMCL*, right midclavicular line; V_{3R}, halfway between the right midclavicular line and the right sternal margin.

It seems probable that the anomalous excitatory process invaded the subepicardial muscle first, and spread toward the endocardium, but lack of information as to what effects might be produced by dorsoventral activation of septal muscle makes it impossible to be sure that such was the case. The esophageal leads throw no light on the question as to whether the excitation wave spread through the dorsal ventricular wall from without inwards or vice versa. When the premature component of the anomalous QRS complex is negative in one of these leads,

the initial component of the normal complex is likewise negative. Normally, the ventricular cavities are negative throughout the QRS interval, and the initial negative component of QRS in leads from the auricular and subauricular levels of the esophagus is presumably due to the transmission of the potential of the ventricular cavities to these regions. That anomalous atrioventricular excitation gives rise to initial negativity of the left ventricular cavity as a whole seems unlikely, for, in the leads from the left side of the precordium, the premature component of the anomalous QRS complex is positive even when the normal QRS complex displays a conspicuous Q deflection. The evidence bearing upon its effect upon the initial potential of the cavity of the right ventricle is less conclusive. In the leads from the right side of the precordium the premature component of the anomalous QRS complex is positive in Case 1, but not in all the other cases of our series. It would seem that positivity of this component in all of the standard precordial leads must be due to activation of the dorsal ventricular wall from without inwards or to dorsoventral activation of septal muscle, if we are warranted in excluding premature activation of the anterior ventricular wall on the grounds previously mentioned.

The occurrence of a second R summit in the anomalous QRS complex of Leads V_1 and V_{3R} is not easy to explain satisfactorily. If the first R summit, which corresponds, as regards its relation to P, to the normal R peak, marks the completion of the excitation of the anterior wall of the right ventricle by impulses arriving via the His bundle, the later fractions of the bifid R wave must be of septal origin in the sense that they represent the overbalancing of opposing forces by those generated by the activation of septal muscle in a left to right direction. In the leads from the left back and the left axilla, an S deflection occupies this same part of the QRS interval, and it is apparent that this deflection and the second R summit in question have the same origin. It seems likely that abnormally early activation of parts of the posterior and posterolateral wall of the left ventricle by the anomalous excitation process prevented the development in these regions of those electric forces which, late in the QRS interval, normally opposed the septal forces referred to.

It may be pointed out here that our observations are not in accord with any of those hypotheses which attribute the electrocardiographic features of the syndrome under consideration to an anomaly of conduction or of impulse formation affecting the right or left branch of the His bundle. An anomaly of this kind should give rise to a QRS pattern characteristic either of complete or of incomplete bundle branch block. Left bundle branch block decreases the size of the R deflection and enormously increases the area of the S wave in the leads from the right side of the precordium. Right bundle branch block does not greatly change the height of the R wave, but substantially decreases the net area of QRS in the leads from the left side of the precordium. It does not abolish Q waves in these leads in cases in which they are present when

the ventricles are activated in the normal way. We feel sure, therefore, that an anomaly of the kind specified could not give rise to electrocardiograms of the kind reproduced in Fig. 2.

Classification of Cases; Groups A and B; the Electrical Axis.—As regards the form of the anomalous ventricular complex in certain leads, all the cases of our series are very much alike. With respect to the form of this complex in other leads, there are great differences between them. The leads from the left side of the precordium, particularly Leads V_4 and V_5, belong to the first class (Figs. 2, 6, 9, and 11). The anomalous QRS complex of these leads is always dominated by a large R wave, and the basal part of the ascending limb of this deflection is invariably slurred or notched by a positive premature component. In most instances there is also a small S wave in one or both of the leads mentioned, but Q is never present in either. In Lead V_6 the ventricular deflections have essentially the same form as in Leads V_4 and V_5, except that the voltage of R is almost always smaller, on occasion much smaller, as in Cases 2 and 4, and S is sometimes considerably deeper (Cases 1, 2, 3, and 4). Depending on the form of QRS in the leads from the right side of the precordium, particularly Leads V_1, V_2, and V_E, our cases have been divided into two groups: Group A, in which R is the sole, or by far the largest, deflection in all of these leads, and Group B, in which S or QS is the chief QRS deflection in at least one of them. Cases 1, 2, 3, 4, and possibly 7 fall in Group A (Figs. 2, 6, and 11), and Cases 5, 6, 8, and 9 fall in Group B (Figs. 9 and 11); Case 10, in which the form of QRS in these leads varied greatly, will be discussed separately. In the four cases in which esophageal leads were taken, the QRS complexes of the leads from the auricular and subauricular levels have essentially the same outline (Figs. 2, 3, 8, and 16). With one exception (Case 1), the ventricular deflections of the leads from the lowest levels of the esophagus are like those of the leads from the left side of the precordium. Leads from the back and the anterolateral aspect of the right side of the chest were taken in Cases 1, 3, 4, 8, and 10. In all of them the lead from the eighth dorsal spine exhibits a broad QS deflection similar to that seen in the leads from the auricular levels of the esophagus (Figs. 4, 7, and 8). The QRS complexes of the lead from the left scapular line resemble those of the leads from the left side of the precordium in only one instance. In most normal subjects, in bundle branch block, and in ventricular hypertrophy the complexes of the leads from the left back and those of the leads from the left side of the precordium are usually strikingly similar in form.

As to the limb leads, there are pronounced variations in the form of the ventricular complexes of Leads V_L and V_F, and therefore in the position of the electrical axis from case to case, but in Lead V_R the QRS deflections have approximately the same general outline in all instances (Figs. 1, 5, and 10). Left axis deviation is very common in anomalous

atrioventricular excitation, and the electrocardiograms in half of our series of ten cases exhibit it. In the three cases of these five in which reversion of the anomalous ventricular complexes to the normal form was recorded in the limb leads, the mean electrical axis of the normal

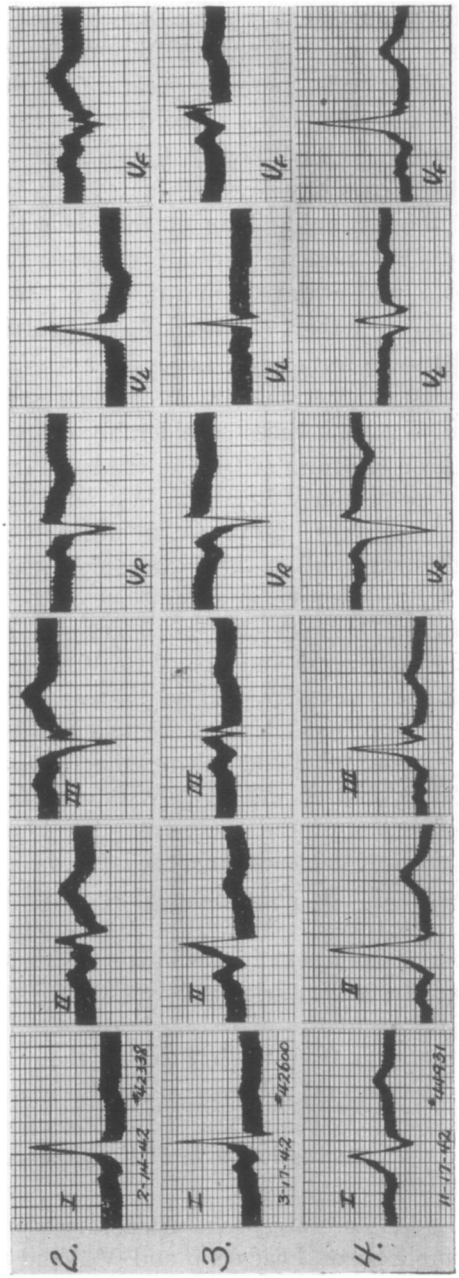

Fig. 5.—Cases 2, 3, and 4. Standard and unipolar limb leads.

QRS group is nearly vertical. It is clear, therefore, that the factors responsible for left axis deviation when the cardiac mechanism is anomalous are not the same as those that give rise to it when the cardiac

mechanism is normal. In Case 1 the anomalous QRS deflections of Lead V_F (Fig. 1) resemble those of the leads from the ventricular levels of the esophagus (Fig. 3), although the initial R wave (not present in the strip of Lead V_F reproduced, but well marked in tracings taken on other occasions) is much larger in the esophageal leads. In Case 8 the ventricular deflections of Lead V_F (Fig. 10) are like those of the leads from the right side of the back (Fig. 7), and, in Case 6 (Fig. 10), like those of esophageal Lead E_{47} (Fig. 8). In these same cases, the QRS deflections, although not the T waves, of Lead V_L are more like those of the leads from the left side of the precordium. It seems probable, therefore, that the occurrence of left axis deviation was due in these cases to abnormally early excitation of the more basal parts of the dorsal ventricular wall and the transmission of the potential variations of this region to the left leg as in posterior myocardial infarction. In those instances in which the limb curves do not display left axis deviation, the ventricular complexes of Lead V_F are like those of the leads from the left side of the precordium or those of the leads from the lowest levels of the esophagus (Case 4, compare Lead V_F, Fig. 5, and Lead E_{59}, Fig. 8). Whether these cases differ from the others because the long axis of the heart made a more acute angle with the frontal plane, or for some other reason, is not clear. It should be noted that there is no correlation between the inclination of the mean electrical axis of QRS in the limb leads and the form of the anomalous ventricular complex in the leads from the right side of the precordium; left axis deviation occurs in cases that belong to Group A (Case 1, Fig. 1), as well as in those that belong to Group B (Case 8, Fig. 10). Our observations suggest, however, that cases of the first group are more likely to display prominent S waves in Lead I and in the leads from the extreme left side of the precordium than are those of the second.

Additional Cases of Group A; Comparison With the Type Case.— Cases 2, 3, 4, and 7 are members of Group A, and may be compared with the type case of this group which has been discussed at length. The anomalous ventricular complexes in Case 2 differ from those in Case 1 in the following respects: there is no S deflection in either Lead I or Lead V_L (Fig. 5); the R wave of Lead V_1 is less distinctly bifid; there is no S deflection in any of the precordial leads except Lead V_6, and the R wave of this lead is very small (Fig. 6).

In Case 3 the limb leads do not show left axis deviation (Fig. 5), and the R wave of Lead V_1 has only one peak (Fig. 6). Two sets of curves were taken in this case, the first on March 17, 1942, before splanchnicectomy, and the second on Aug. 17, 1943, after this operation. The extremity and precordial leads reproduced here belong to the first set, and the other thoracic leads, to the second set. The differences between the two sets of tracings are of a minor kind. Compared to the first, the second set of precordial curves exhibits a larger S wave in Leads V_1 and V_F, smaller R deflections in Leads V_2 to V_6, inclusive, and upright in-

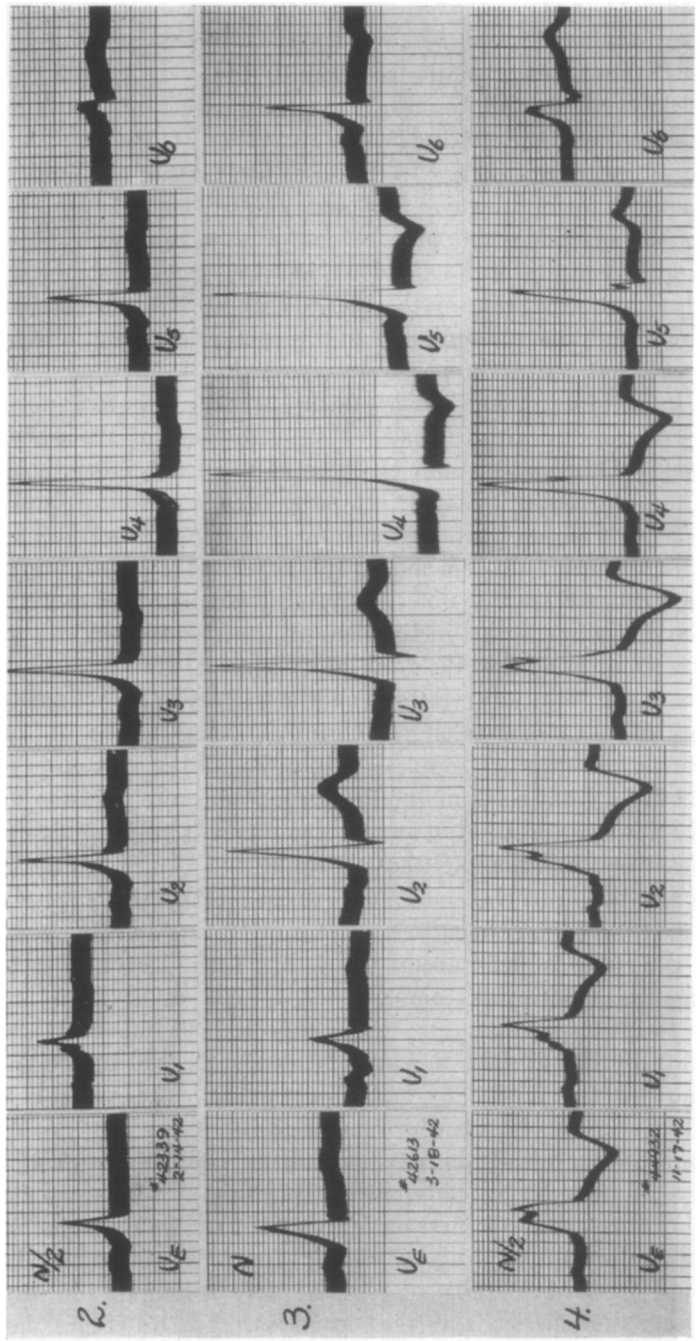

Fig. 6.—Cases 2, 3, and 4. Precordial leads.

stead of inverted T waves in Leads V_4, V_5, and V_6. The first set of leads from the back, the right axilla, and the right anterior chest wall differs from the second set in these particulars: there is a distinct S deflection in the lead from the left posterior axillary line, the T wave in this lead is inverted instead of upright, and there is no S in Lead V_{3R}.

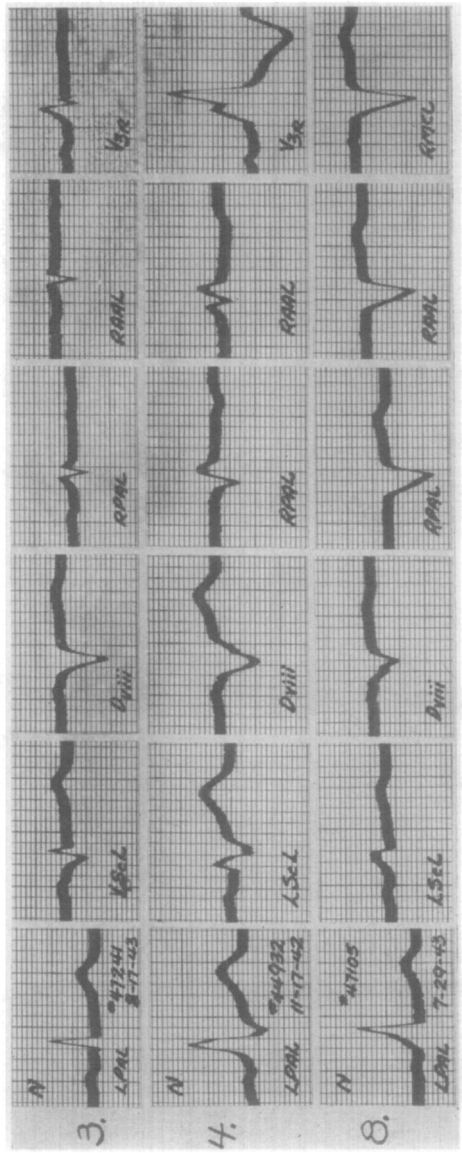

Fig. 7.—Cases 3, 4, and 8. Unipolar leads from the back and right anterior hemithorax. Compare with Fig. 4, in which the same symbols are employed.

In Case 7 precordial leads were taken on three occasions, June 19, 1934, July 9, 1934, and July 27, 1934. The second set of curves is reproduced (Fig. 11). In the others the ventricular complexes of Lead V_E have the same form, but those of Lead V_1 display a conspicuous S wave. In the third set this S is as large as the R wave, and there is some

doubt as to whether this case properly belongs in Group A. In the figures it has been placed with the cases of Group B.

In Case 4 precordial leads were taken Nov. 13, 1942, as well as on Nov. 17, 1942. There are no significant differences between the two sets of tracings. The form of the ventricular deflections of the standard leads, however, was quite variable, and could be greatly modified by forced respiration. There was always a prominent S wave in Lead I,

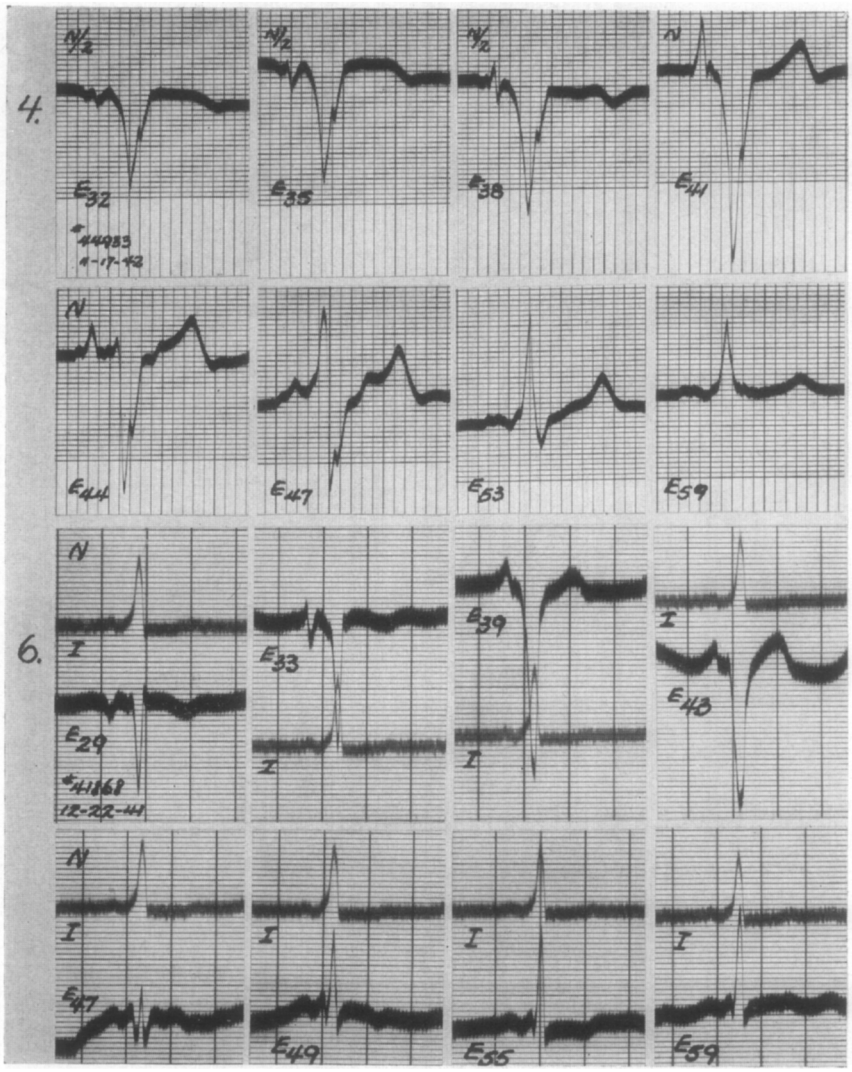

Fig. 8.—Cases 4 and 6. Unipolar esophageal leads. Compare with Fig. 3.

but in some records the voltage of R_1 is more than twice as great as in that reproduced (Fig. 5). The very long QRS interval, which measures at least 0.16 second, and the broad, deformed P waves of the limb leads raise the question as to whether anomalous atrioventricular excitation

was the only cardiac abnormality present. The ventricular complexes of the thoracic and esophageal leads differ from those of the corresponding leads in the type case in minor particulars only (Figs. 6, 7, and 8). The premature component of QRS is positive instead of negative in the leads from the right anterior axillary line, and there is a more conspicuous final R deflection in the leads from the right back (Fig. 7). In the leads from the lowest levels of the esophagus (Fig. 8) there is only one R wave and this component is much larger than in Case 1 (Fig. 3).

The Type Case of Group B.—Case 5 is a typical example of the cases of the second group. In this instance transitions from the normal to the anomalous cardiac mechanism could be induced by the Valsalva procedure. Strips of the standard extremity leads and the unipolar precordial leads which show both kinds of ventricular complexes are reproduced in Fig. 9. No other leads were taken. Both the normal and the anomalous ventricular complexes of the limb leads closely resemble the corresponding complexes of the same leads recorded in Case 1. It will be noted, however, that in Lead I there is no S component in either species of complex, whereas, in Case 1, there is a conspicuous S in both (Fig. 1). There are no significant differences between the type cases of the two groups as far as the deflections of the leads from the left side of the precordium are concerned, with one possible exception. In Case 1 there is a conspicuous S wave in the anomalous QRS group of Lead V_6; in Case 5 this component is absent. On the other hand, the two cases differ greatly as regards the form of the anomalous ventricular complexes of the leads from the right side of the precordium (V_1 and V_2). In Case 5 the premature component is diphasic in Lead V_1 and very small in Lead V_2. There is a notch on the descending limb of the deep S wave of these leads. There is no trace of the final positive com-

TABLE II

CASE 5. INTERVALS IN FIG. 9. MEASUREMENTS IN SECONDS

	1		2		3		4		5	
LEAD	a	n	a	n	a	n	a	n	a	n
I	.122	.160	-	.173	.203	.203	-	.227	.244	.238
II	.115	.155	-	.170	.204	.204	.230	.237	.256	.251
III	.121	.152	.170	.170	.196	.202	.216	.234	.250	.248
V_1	.116	.150	-	-	.144	.175	.206	.204	.256	.244
					.182*					
V_2	.116	.166	-	-	.146	.192	.216	.222	.261	.262
					.187*					
V_3	.127	.172	-	-	.216	.210	.251	.232	.268	.266
V_4	.129	.171	-	.171	.211	.215	.250	.231	.257	.253
V_5	.128	.163	-	.171	.212	.204	.246	.228	.258	.252
V_6	.119	.155	-	.166	.206	.200	-	.238	.252	.232

Key:
a—anomalous; n—normal.
Column 1—measurement from beginning of P to beginning of QRS.
Column 2—measurement from beginning of P to peak of Q.
Column 3—measurement from beginning of P to peak of R.
Column 4—measurement from beginning of P to peak of S.
Column 5—measurement from beginning of P to end of QRS.
*Measurement to submerged R peak on descending limb of S.

ponent which is such a prominent feature of the anomalous QRS of Lead V_1 in the type case of Group A. It may be pointed out, however, that in Leads V_1 and V_2 the net area of the anomalous QRS is algebraically larger than the net area of the normal QRS. This is clearly

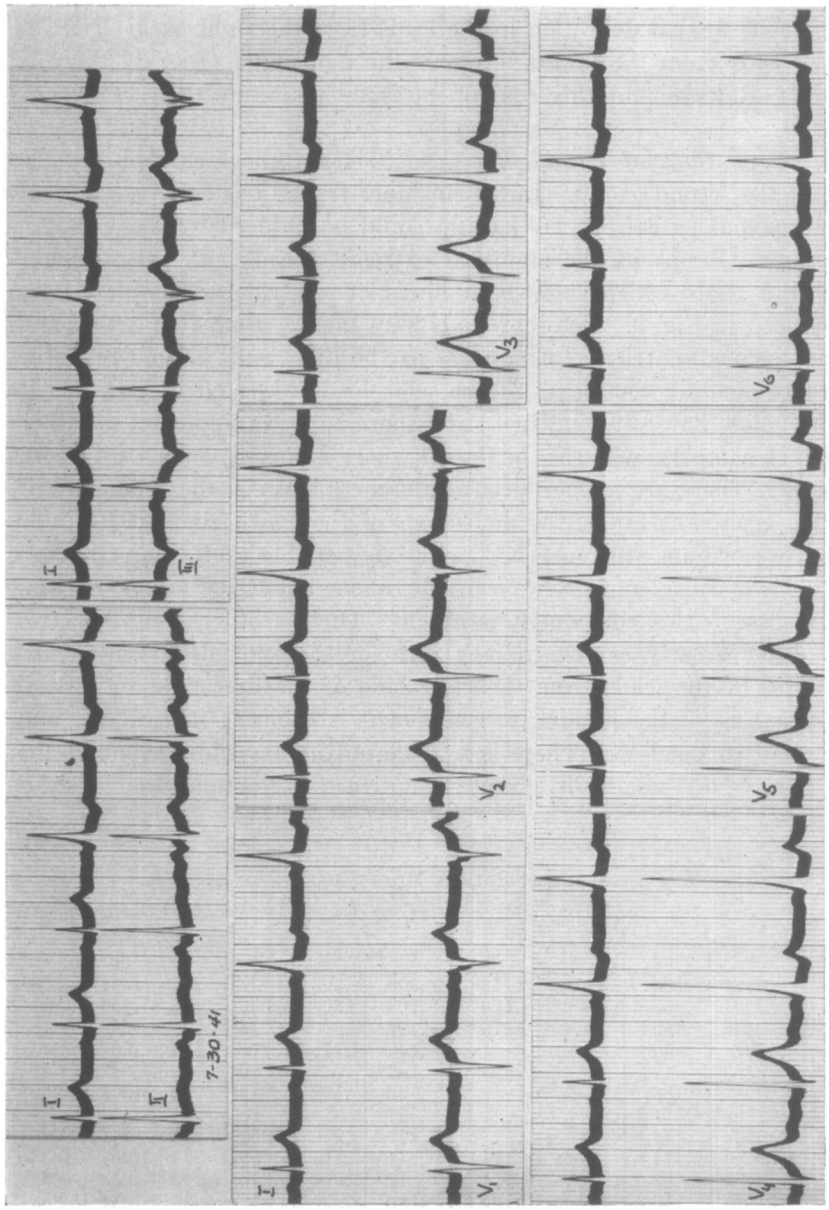

Fig. 9.—Case 5. Leads II and III and precordial Leads V_1 to V_6 inclusive, taken simultaneously with Lead I. In the records of the standard leads, the first three complexes are normal, the last three anomalous; in the precordial leads the first two are normal, the last two anomalous. Compare with Figs. 1 and 2.

indicated by the comparative size of the anomalous and the normal T waves. The difference between Case 5 and Case 1 in this respect is one of magnitude and not one of kind.

Table II gives, for each lead and for each type of complex, measurements of the interval from the beginning of the P wave to (1) the onset of the first QRS deflection, (2) the apex of Q, when this component is present, (3) the apex of R, (4) the apex of S, and (5) the end of the QRS complex. The P-R interval of the anomalous beats appears to be

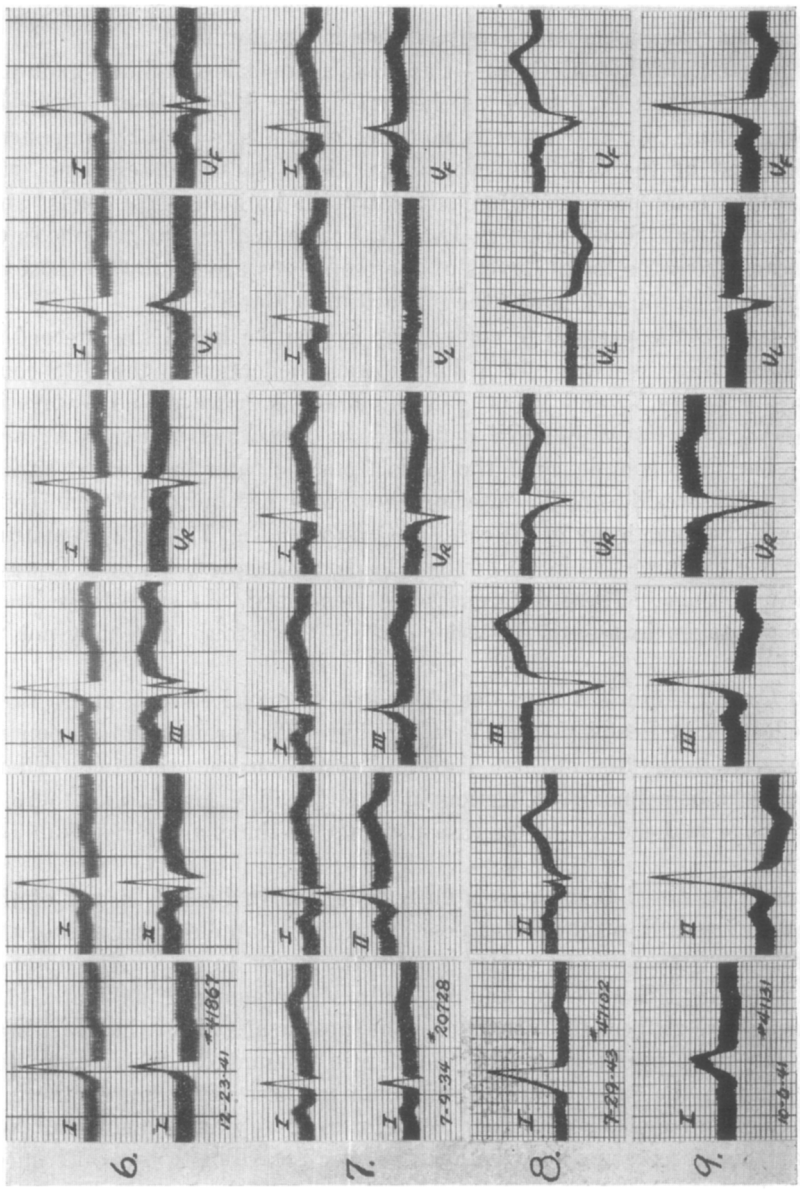

Fig. 10.—Cases 6, 7, 8, and 9. Standard and unipolar limb leads. Compare with Fig. 5.

roughly 0.04 second shorter than that of the normal beats, and the interval from the beginning of P to the end of the QRS complex is approximately the same for beats of both types. In the standard limb leads and in the leads from the left side of the precordium the R peak of the

normal, and that of the anomalous, complex bear the same relation to the P wave within a few thousandths of a second. In the leads from the right side of the precordium the notch on the descending limb of S in the anomalous complex corresponds in time to the peak of R in the normal QRS complex. These measurements, like those of Table I, support the view that the His bundle transmitted impulses when the cardiac mechanism was anomalous as well as when it was normal.

Group B; Additional Cases.—Case 6 is a much less striking example of Group B than that taken as the type. The S deflection is large in Leads V_1 and V_E, and there is no trace of a positive QRS component at the end of the QRS interval (Fig. 11). On the other hand, the premature component of QRS is positive in both of these leads, and there is a small S deflection in Lead V_6. The ventricular complexes of the leads from the lowest levels of the esophagus are similar to those of the leads from the left side of the precordium (Fig. 8).

In Case 8 the premature component is positive in all the precordial leads and there is a trace of a final upward deflection in the QRS complexes of Leads V_1 and V_2 (Fig. 11). There is, however, no S wave in Lead V_6, and the chief QRS deflection is downward in Leads V_1 and V_E. The QRS complexes of the leads from the right back, right axilla, and the right side of the anterior chest wall are quite different from those of the same leads in the cases of Group A (Fig. 7). The premature component is negative in all of these leads, and there is no final R wave in any of them. These differences are mentioned, but since these leads were not taken in any of the other cases of this group, they may not be significant.

In Case 9 there is a very deep QS deflection in Lead V_1, and a deep S deflection in Leads V_2 and V_3 (Fig. 11). The premature component is negative in the first of these leads and positive in the other two. There is no trace of a final R deflection in either Lead V_1 or V_2, and there is no S wave in Lead V_6. This case presents all the characteristics of the group.

It will be noted that there is no S deflection in Lead I in any of the cases of Group B, although the position of the mean electrical axis of the anomalous QRS complex varies greatly from case to case. The absence of a prominent S wave in Lead V_6 is also conspicuous (Figs. 9 and 11). Although we suspect that the muscle on the dorsal wall of the heart that was activated prematurely was smaller in amount or different in distribution in the cases of this group than in those of Group A, there is not enough evidence bearing on this point to justify any conclusion. We must, therefore, consider whether the electrocardiographic differences between the cases of these two groups are dependent upon differences in the order of ventricular activation or upon variations in the position of the heart. The magnitude of the differences, as regards the form of the anomalous ventricular complexes of the leads from the right side of the precordium, between Cases 1 and 4, on the one hand, and Cases 5

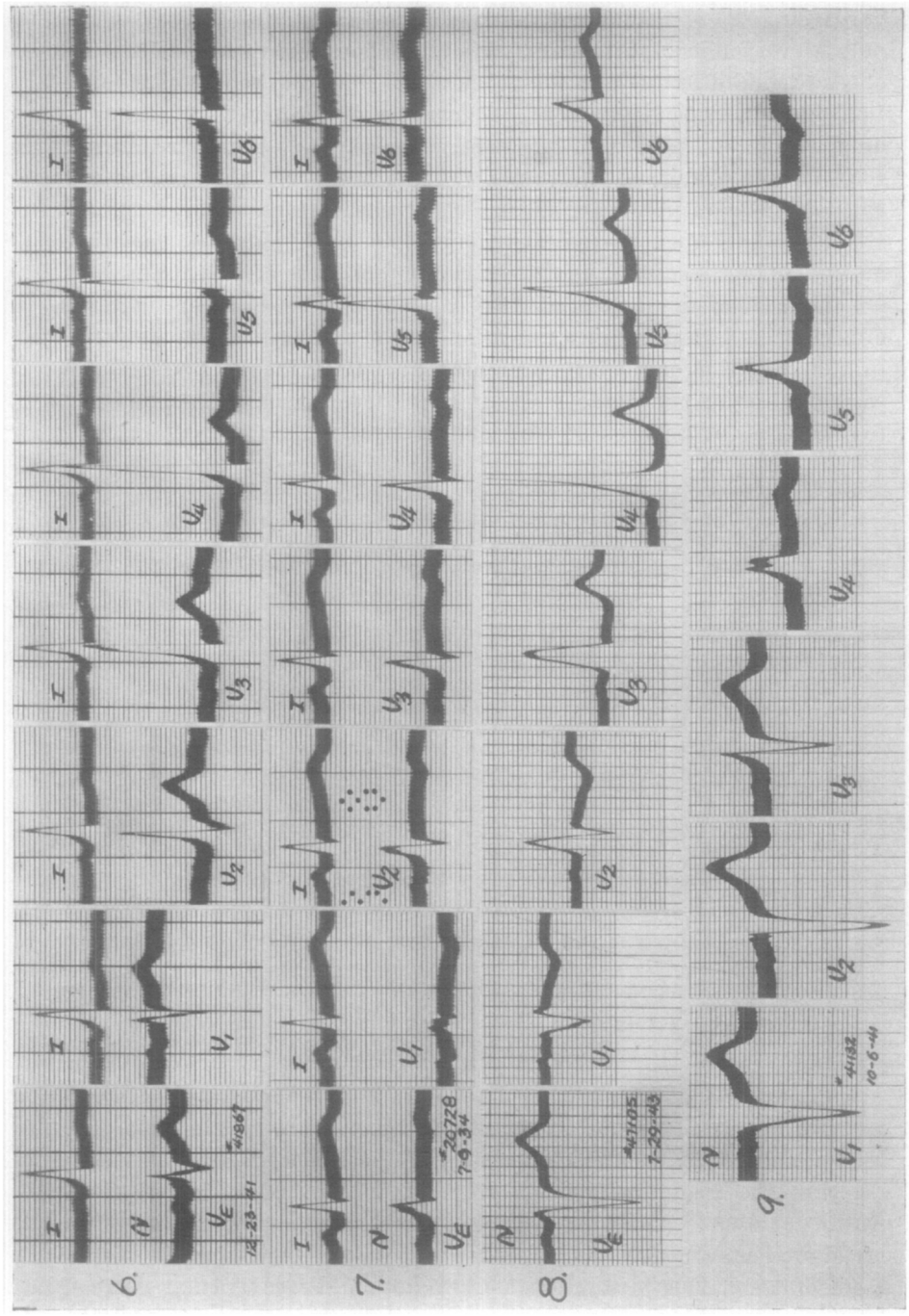

Fig. 11.—Cases 6, 7, 8, and 9. Precordial leads. Compare with Fig. 6.

and 9, on the other, is certainly opposed to the second supposition. This difference is particularly striking when it is borne in mind that, as regards the form of the normal ventricular complexes, Cases 1 and 5 differ only in very minor particulars. We must, however, remember that the effect produced by the position of the heart upon the ventricular deflection must be dependent upon the character of the potential variations on the different aspects of the ventricular surface, and, consequently, that a given change in the position of the heart may give rise to conspicuous changes in the ventricular electrocardiogram, or no changes at all, depending upon the epicardial distribution of potential variations of one kind, as compared to the distribution of those of an opposite sort. On the basis of the data presented, it is not possible to accept or reject either of the two possibilities mentioned, but the observations described in a later section of this article indicate that the differences between the cases of Group A and those of Group B are due, at least in some measure, to differences in the order of ventricular activation.

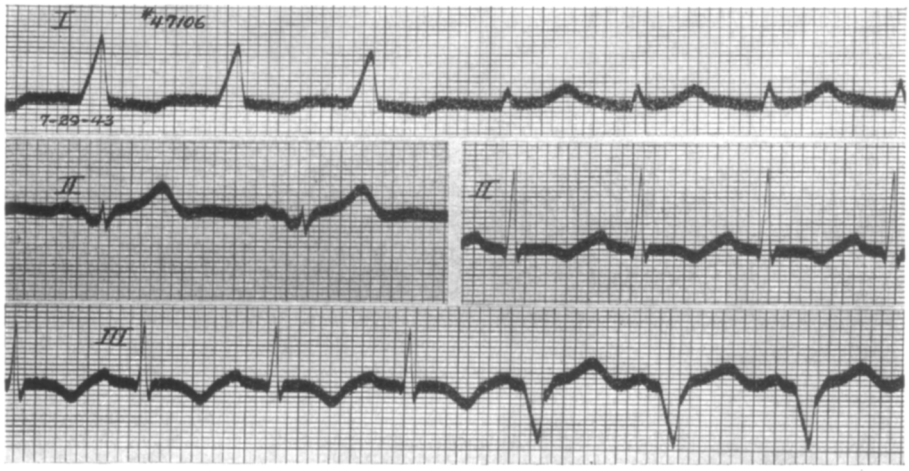

Fig. 12.—Case 8. Leads I, II, and III, showing both anomalous and normal complexes.

OBSERVATIONS RELATING TO THE EFFECT OF ATRIOVENTRICULAR RHYTHM UPON THE FORM OF THE VENTRICULAR COMPLEX IN ANOMALOUS ATRIOVENTRICULAR EXCITATION

An accessory atrioventricular bundle, if it is regarded as a separate and distinct structure, and in no sense as a part of, or as connected with, the specialized atrioventricular system of the normal heart, can hardly transmit the excitatory process from auricles to ventricles when the cardiac rhythm is under the control of a center in the lower levels of the atrioventricular node. Our working hypothesis, then, implies that in cases of anomalous atrioventricular excitation the ventricular complex must assume the normal form on the induction of atrioventricular rhythm of the kind in which ventricular excitation is simultaneous with, or precedes, auricular. We have not made an extensive search of the

literature, but two cases of anomalous atrioventricular excitation in which atrioventricular rhythm of this sort was observed have come to our attention. One of these was reported by Fox, Travell, and Molofsky,[14] and the other by Aixalá.[15] The ventricular complex was of the normal form during the atrioventricular rhythm in both of these cases. The authors who reported them did not comment upon the possible significance of their observations on this point.

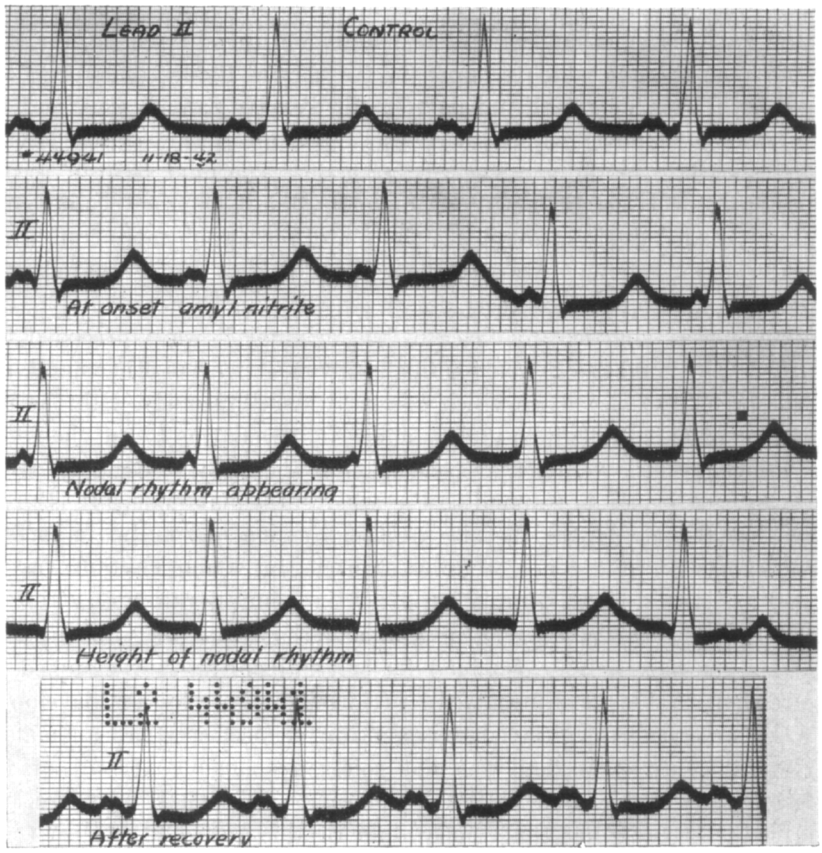

Fig. 13.—Case 4. The effect of atrioventricular nodal rhythm upon the form of the anomalous ventricular complex in Lead II.

In Case 4 of our series, atrioventricular rhythm of the kind in which the P wave is buried in the QRS complex was observed on two occasions. On the first, it appeared when the patient inhaled amyl nitrite approximately three hours after the administration of 0.6 Gm. of quinidine sulfate. Up to that time the latter had had no noticeable effect upon the cardiac mechanism. On the second occasion it was induced by carotid sinus stimulation approximately eight minutes after the hypodermic injection of 0.0013 Gm. of atropine sulfate. Thirty minutes after this injection the same procedure was no longer effective. The occurrence of atrioventricular rhythm after the inhalation of amyl nitrite is illustrated in Fig. 13. In the control record a broad, notched

P wave is followed by a slowly rising segment which is apparently part of the QRS complex. If this interpretation is correct, the QRS interval measures approximately 0.14 second, but if the segment in question is ignored, this interval does not much exceed 0.10 second. The abnormally long Q-T interval may be due to the administration of quinidine. The next two strips of record show a transition from sinus rhythm to atrioventricular rhythm. During this transition the ventricles were responding to the atrioventricular node, but some fraction of the auricular muscle was still responding to the sinus node, for that part of the P wave which remains visible retains its original form. In the fourth strip of record, no part of the P wave can be seen, and we may assume that when this record was taken all of the cardiac muscle was responding to the atrioventricular node. The final strip of record represents the re-establishment of sinus rhythm. The ventricular complexes recorded during the ectopic rhythm differ significantly from those of the control tracing in two respects: they display a somewhat shorter QRS interval and a definite Q deflection. It will be noted that Q did not appear as long as any trace of the original P wave preceded the QRS complex, and the reason for this is obvious. On the other hand, the reduction in the size of S which occurred simultaneously with the appearance of Q is difficult to understand. If it were due to the change in the location of the ventricular pacemaker it should have occurred when the ventricle began to respond to the atrioventricular node. It is probable that the change in the size of this component has no important significance, for it did not occur when atrioventricular rhythm was induced by carotid sinus stimulation after the administration of atropine.

The results of this experiment are somewhat equivocal, for the ventricular complex neither retained its original outline nor assumed an entirely normal appearance when atrioventricular rhythm developed. We have already mentioned reasons for suspecting that anomalous atrioventricular excitation was not solely responsible for all of the electrocardiographic peculiarities in this case. We do not, therefore, feel justified in concluding that the ectopic rhythm failed to abolish those that can be justifiably ascribed to it.

A most interesting case, the last of our series, and one of those studied by Hecht, at the William J. Seymour Hospital, remains to be described. The patient was under observation for a long period, and many electrocardiograms were taken. We shall describe and illustrate only the more significant.

The anomalous QRS complexes of the limb leads (Fig. 15, *a* and *b*) and those of the leads from the left side of the precordium (Leads V_4, V_5, and V_6, Fig. 14) have the same general contour in all records. The former exhibit pronounced left axis deviation, and the latter differ in no significant way from the QRS complexes of the same leads in the other cases of our series. On the other hand, the form of the QRS complexes of the leads from the right side of the precordium is very variable (Fig.

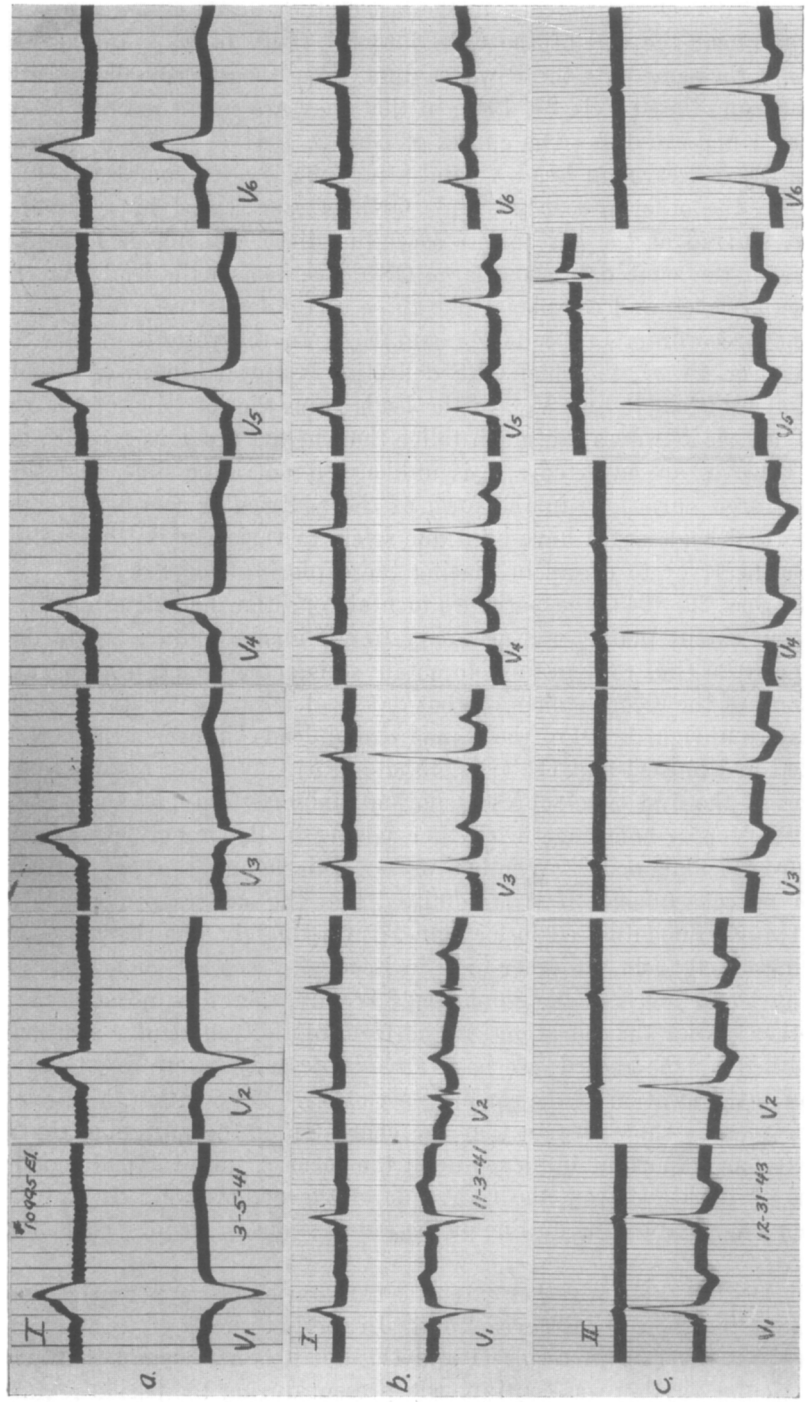

Fig. 14.—Three sets of precordial leads taken simultaneously with a standard limb lead. In *c* the limb lead was taken with the electrocardiograph at subnormal sensitivity. Note variation in the form of the ventricular complexes of the precordial leads.

14). In the tracing taken March 5, 1941, Leads V_1, V_2, and V_3 display large QS deflections, notched in the last of these leads by an embryonic R wave near the end of the QRS interval (Fig. 14, *a*). In the limb leads of the same date the P waves differ from those of the first electrocardiogram, dated Feb. 27, 1941, in that they are small in Lead II and inverted in Lead III. The curves of Nov. 3, 1941 (Fig. 14, *b*), show a large QS deflection in Lead V_1 and a polyphasic QRS complex in Lead V_2. In the other precordial leads QRS is represented by a broad R wave, slurred at the base of its ascending limb. Esophageal tracings taken on the same date show large QS deflections in the lead from the auricular level, and complexes like those of the leads from the left side of the precordium in Leads E_{40} and E_{50}. In the records of Dec. 31, 1943 (Fig. 14, *c*), the chief QRS deflection is upward in all of the precordial leads, including V_E; in the leads from the left side of the precordium the R waves are much taller than in the previous records, and the T waves are inverted. It is unlikely, if not impossible, that these pronounced variations in the form of the ventricular complexes of the precordial leads could have been due solely to variations in the position of the heart or to errors in placing the exploring electrode.

By Dec. 29, 1943, the patient's mental condition had deteriorated to such an extent that it was necessary to administer sodium amytal in a dose of 0.25 Gm. (3¾ grains) to obtain satisfactory electrocardiograms. In some of the records taken on that day, and subsequently, the P deflections are upright in all of the standard limb leads; in others they are inverted in Leads II and III (Fig. 15, *a* and *b*). We shall assume that P waves of the first type represent normal sinus rhythm, and those of the second type, a homogenetic rhythm arising in the upper levels of the atrioventricular node. Whether or not this assumption is justifiable is of no consequence, if it is admitted that the centers responsible for the two rhythms differed in location. Unfortunately, one rhythm can be distinguished from the other only in Lead II, Lead III, or a lead from the auricular levels of the esophagus. We are, therefore, not able to say whether or not the variations in the form of the ventricular complexes exhibited by the records we have already described were due to variations in the location of the cardiac pacemaker. We mention this because other records show clearly that the character of the auricular rhythm exerted an important influence upon the form of the ventricular deflections of the leads from the right side of the precordium. It had only minor effects upon the outline of these deflections in the other leads employed.

On Dec. 29, 1943, a rather extensive exploration of the potential variations of the thorax was carried out. In the limb leads of that date the P wave is inverted in Leads II and III, but it is not certain that atrioventricular rhythm was continuously present during the period required to take all of the many unipolar leads employed. In the leads from the left margin of the sternum, from the left midclavicular line, and from

a line halfway between the two (levels of the second, third, fourth, and fifth intercostal spaces), the QRS deflections are represented by a broad R wave which is slurred near the base of its ascending limb. In the leads from the right sternal margin, the right midclavicular line, and a line midway between the two (levels of the second, third, fourth, and fifth intercostal spaces), the QRS complex consists of a broad QS deflection, or of a Q followed by an R wave, with one notable exception. In

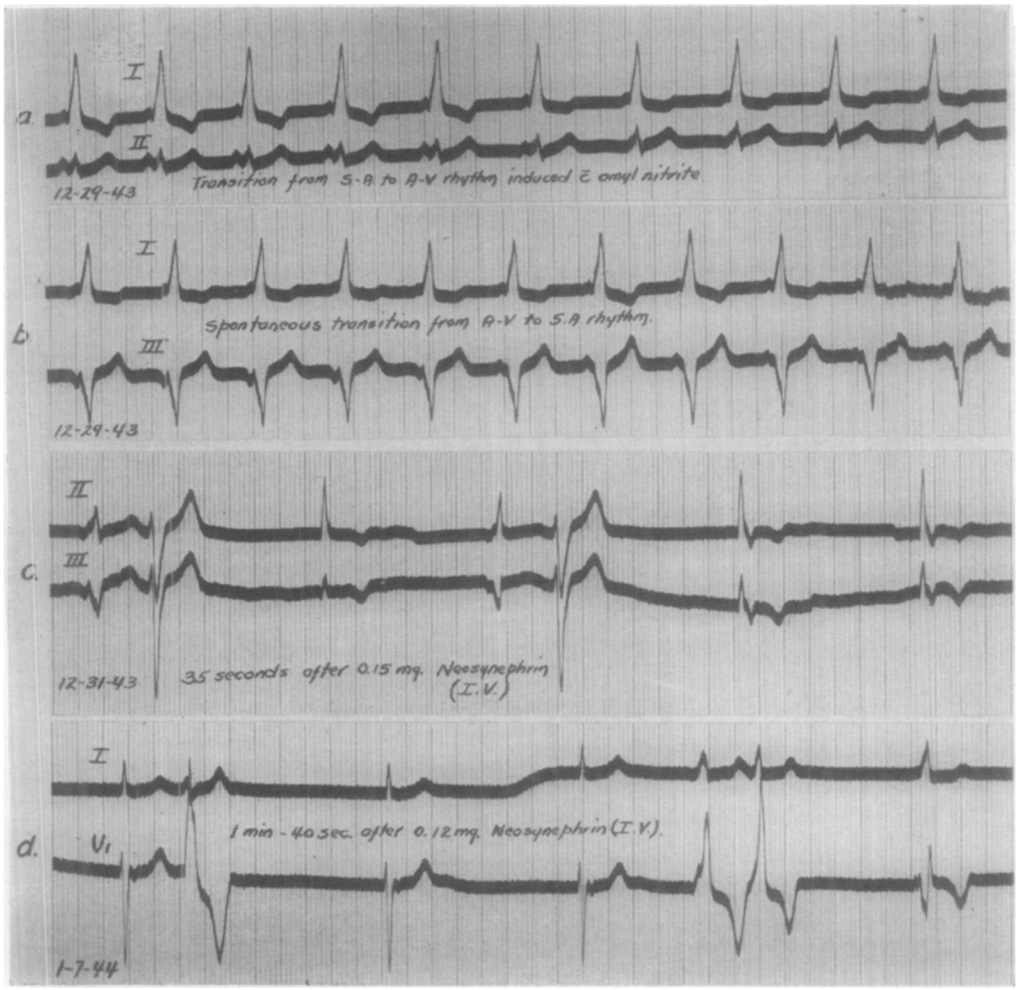

Fig. 15.—Case 10. *a*, Transition from sinus to atrioventricular rhythm arising in the upper levels of the atrioventricular node. *b*, Transition from atrioventricular rhythm arising in the upper part of the atrioventricular node to sinus rhythm. *c*, Taken after neosynephrin. Complexes 6 and 7 represent beats arising in the lower levels of the atrioventricular node; QRS is followed by an inverted P wave. Complex 3 is of the same type except that no P wave is visible. Complex 1 represents a beat arising in the higher levels of the atrioventricular node; the ventricular complex is anomalous. Complex 4 is transitional in form between Complex 1 and Complexes 3, 6, and 7. The remaining beats are ventricular extrasystoles. *d*, Complexes 1, 3, and 4 represent beats arising in the lower levels of the atrioventricular node; in the precordial lead (V_1), QRS is followed by an inverted P wave. Complexes 2, 5, and 6 represent ventricular extrasystoles. Complex 7 in V_1 resembles beats of questionable origin (see text), except that the QRS complex is triphasic instead of consisting of a broad, notched R. Note that the ventricular complexes of the atrioventricular beats are of the normal form.

the lead taken with the exploring electrode in the fifth intercostal space at the right sternal margin, the ventricular complex is of the same form as in the leads taken from points farther to the left. In the other leads the R component is largest in comparison with the Q deflection in those from the second intercostal space, and is either smallest or absent in those from the fifth intercostal space. In the leads from the right midaxillary line and right anterior axillary line (level of the fourth and

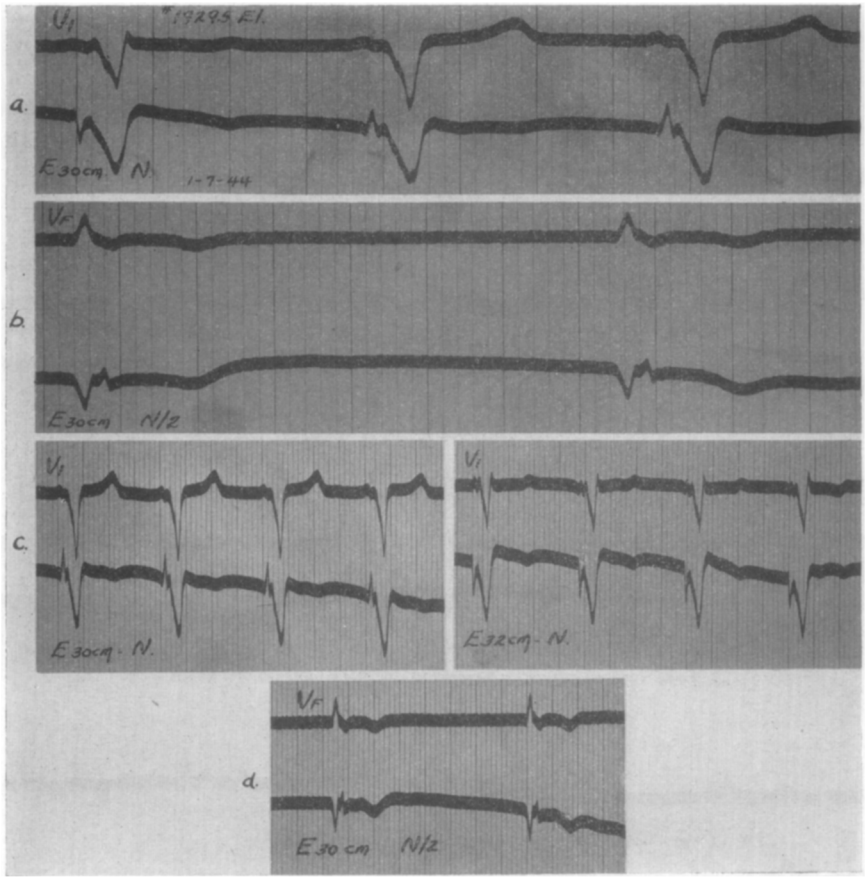

Fig. 16.—Case 10. *a*, Transition from atrioventricular rhythm arising in the upper levels of the atrioventricular node to sinus rhythm; precordial Lead V_1 taken simultaneously with a lead from the auricular level of the esophagus. *b*, Taken after neosynephrin on Jan. 7, 1944. Two beats which arose in the lower levels of the atrioventricular node (QRS followed by a P wave); lead from the auricular level of the esophagus taken simultaneously with Lead V_E. *c*, Lead from the auricular level of the esophagus taken simultaneously with precordial Lead V_1; the first strip shows sinus rhythm, the second a rhythm arising in the upper levels of the atrioventricular node. *d*, same as *b*, but with camera running at a slower speed.

fifth intercostal space), the QRS complex is represented by a QS deflection. In the leads from the left posterior axillary line and the left midscapular line (level of the fifth intercostal space), the QRS complexes are like those of the leads from the left side of the precordium. In the lead from the left paravertebral line, at the same level, QRS consists of a broad R, followed by a small S wave. In

the leads from the seventh dorsal spine and from the right paravertebral and right scapular line at the level of this spine, the QRS group is represented by a QS deflection of small voltage. Transitions from sinus to atrioventricular rhythm, or the reverse, are shown in Figs. 15, 16, 17, 18,

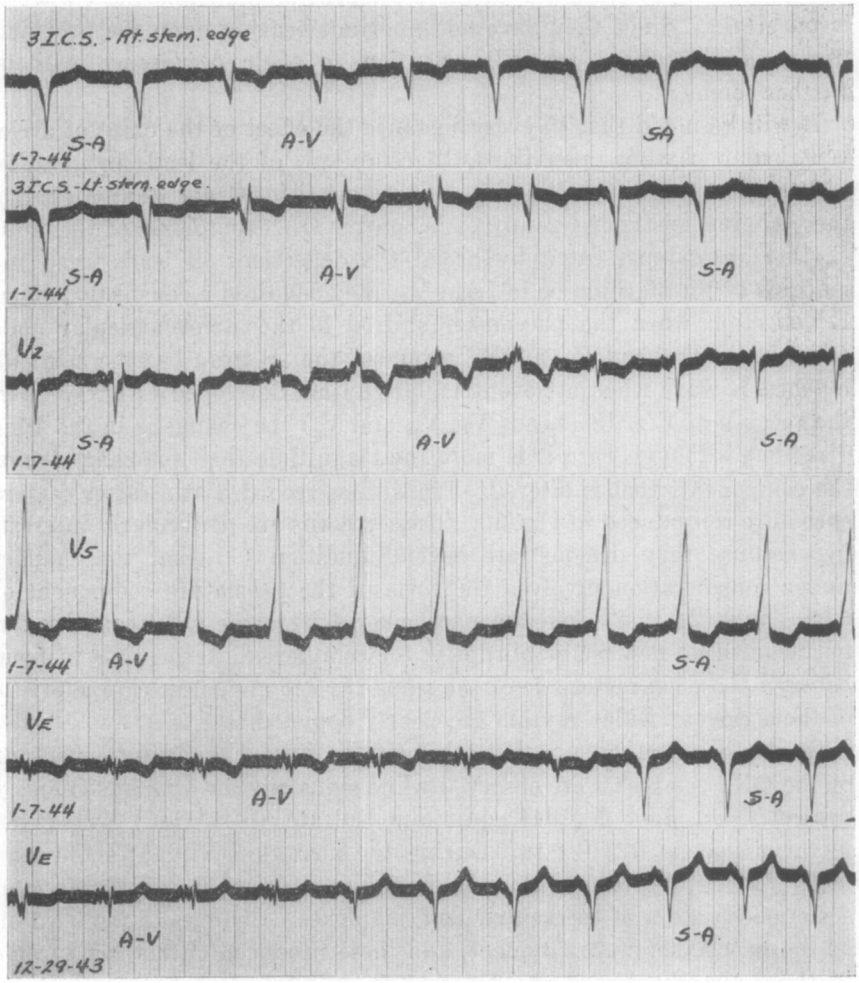

Fig. 17.—Case 10. Transitions from sinus rhythm to atrioventricular rhythm arising in the upper levels of the atrioventricular node, or vice versa. Precordial leads taken on Jan. 7, 1944.

and 19. In the limb leads (Fig. 15, *a* and *b*) the development of the ectopic rhythm was accompanied by a slight reduction in the voltage of the chief QRS deflection in Leads I and III; in the leads from the auricular levels of the esophagus it produced no noticeable change in the form of the QRS complex (Fig. 16). In Leads V_E, V_1, and V_2 (Figs. 17 and 18), however, its effect upon the character of the ventricular deflections was pronounced. It will be noted that the ventricular complexes of these leads varied in form independently of the location of the pacemaker; they were not of the same form on Jan. 11, 1944, as on Jan.

7, 1944, either when the auricular rhythm was normal or when it was ectopic (compare Fig. 18 with Fig. 17). Whether these apparently spontaneous changes in the form of the ventricular complex were produced by variations in the order of ventricular activation or by variations in the position of the heart or in the placement of the exploring electrode is uncertain. Since they have no important bearing upon the problems under consideration, we call attention to their occurrence without further comment.

It will be noted that in every instance the effect of the onset of atrioventricular rhythm upon the QRS complexes of the leads in question was to make them more like those characteristic of Group A and less like those characteristic of Group B. When the QRS complexes of the sinus rhythm were represented by broad QS deflections in leads from the margins of the sternum or in Lead V_E, they acquired a conspicuous final R deflection when the pacemaker shifted to the atrioventricular node (Fig. 17). In Lead V_2, a QRS group of the rS form became a broad, notched R wave when atrioventricular rhythm developed (Fig. 17). In the records of Fig. 18 (Leads V_E, V_1, and V_2) the change is in the same direction, although here it is more the magnitude than the character of the components that is altered. Transitions recorded at a faster camera speed are reproduced in Fig. 19. These records are particularly interesting because they demonstrate beyond question (1) that the changes under consideration involved the form of the premature component of QRS; and (2) that when the pacemaker shifted the QRS complex did not acquire its new shape abruptly, for complexes intermediate in form between those characteristic of the sinus rhythm and those characteristic of the atrioventricular rhythm are clearly depicted.

On one occasion, abrupt, but otherwise similar, changes in the contour of the QRS complex were observed while sinus rhythm was continuously present. On Jan. 7, 1944, quinidine sulfate (0.47 Gm.) was given intravenously at 12:10 P.M. During the next thirty minutes the limb leads showed a pronounced arrhythmia, an increase in heart rate, and some modification of the ventricular complexes. Changes in the location of the pacemaker were frequent, and these produced effects comparable to those already described. No changes in auricular rhythm were recorded in the chest leads taken during this period. At 12:40 P.M., however, a record of Lead V_1, taken simultaneously with Lead III, showed changes in the character of the ventricular complexes, even though sinus rhythm was continuously present (Fig. 18). At the beginning of this record the QRS group of the chest lead consists of a broad, notched R, but from time to time two or three QRS complexes in succession are represented by broad downward deflections, followed by a small R component. In later parts of the tracing the number of complexes of the second type rapidly increases, and those of the first type disappear.

On Dec. 31, 1943, and on Jan. 7, 1944, records were taken after the intravenous injection of neosynephrin (0.15 mg. on the first date and

0.12 mg. on the second). On the first occasion standard limb leads were employed. Before the drug was given, atrioventricular rhythm of the kind already described was present. The earliest effects of the drug were slowing of the heart rate, variations in the location of the pacemaker, and arrhythmia due to ventricular extrasystoles. There are

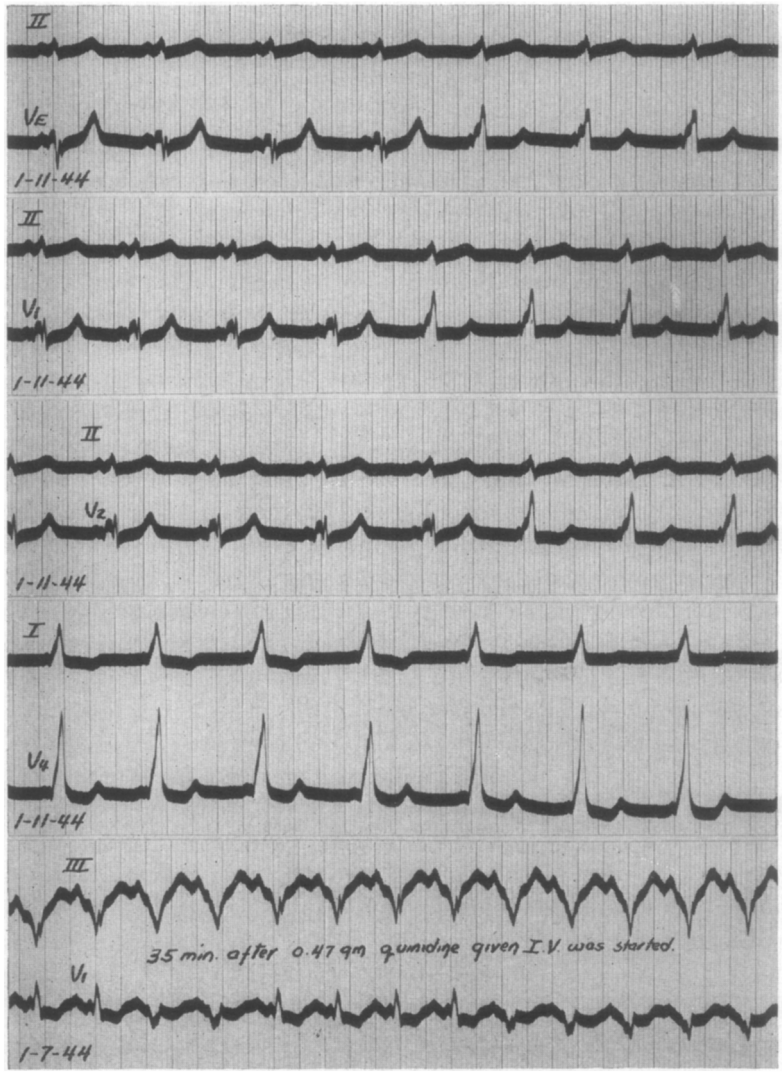

Fig. 18.—Case 10. *a*, *b*, *c*, and *d*, Transitions from sinus rhythm to atrioventricular rhythm arising in the upper levels of the atrioventricular node. Precordial leads taken simultaneously with a standard limb lead on Jan. 11, 1944. *e*, Variations in the form of the ventricular complex after the intravenous administration of quinidine when sinus rhythm was continuously present (Jan. 7, 1944).

short strips of record in which all of the beats, and others in which all of the beats other than those which are obviously ventricular extrasystoles, are represented by ventricular complexes of the normal form (Fig. 15, *c*). In the majority of instances the QRS deflections of these

beats are immediately followed by inverted P waves both in Lead II and Lead III. In other instances the initial QRS component is preceded by a conspicuous dip, which apparently represents the first limb of an inverted P wave which is partly superimposed upon the ventricular deflections. In still others no trace of a P deflection is visible. There is also one ventricular complex which is intermediate in form between those that represent anomalous, and those that represent normal, atrioventricular excitation (fourth complex, Fig. 15, *c*). The later sections of the record show short runs of extrasystoles represented by ventricular complexes of variable form, followed presently by the return of the original cardiac mechanism. On the second occasion, similar, but somewhat more complicated, changes in the cardiac mechanism followed the injection of the drug. Most of the records show Lead I and Lead V_1, taken simultaneously, but there is also a tracing of a lead from the auricular level of the esophagus, taken simultaneously with Lead V_E. At the beginning, sinus rhythm is present and the QRS complex of Lead V_1 is represented by a broad QS deflection. Some seconds later the pacemaker shifts to the upper levels of the atrioventricular node, and this complex displays a broad Q followed by a small R wave. Then extrasystoles begin to occur, and there are frequent transitions from atrioventricular rhythm to sinus rhythm and back again (Fig. 16, *a* and *c*). Presently we come to a strip of record in which two or more beats in succession are represented by ventricular complexes of the normal form, not accompanied by visible P waves. In this same strip of record there are beats which are similarly spaced, but represented by ventricular complexes of still another form. In Lead V_1 the QRS group consists of a broad, deeply notched R; the first peak of this deflection is less than half as high as the second. In Lead I the QRS complex is like that of the beats of sinus origin, but of smaller voltage, and no P wave can be made out. It may be that these beats were of ventricular origin, for, in parts of the record, they are irregularly spaced and occur in rapid succession. In other sections of the record there are beats represented by normal ventricular complexes which display, in Lead V_1, an inverted P wave immediately following the QRS complex (first, third, and fourth complexes, Fig. 15, *d*). Beats of similar origin were recorded in a lead from the auricular level of the esophagus (Fig. 16, *b* and *d*).

These observations demonstrate that the His bundle and its branches were capable of functioning, and that impulses arising in the lower levels of the atrioventricular node spread to the auricles and to the ventricles in the normal manner.

Do the observed effects of atrioventricular rhythm upon the form of the ventricular complex support our working hypothesis or are they in conflict with it? Let us examine certain implications of this hypothesis which we have not had occasion to consider heretofore. If an accessory atrioventricular bundle is present, the excitatory process may

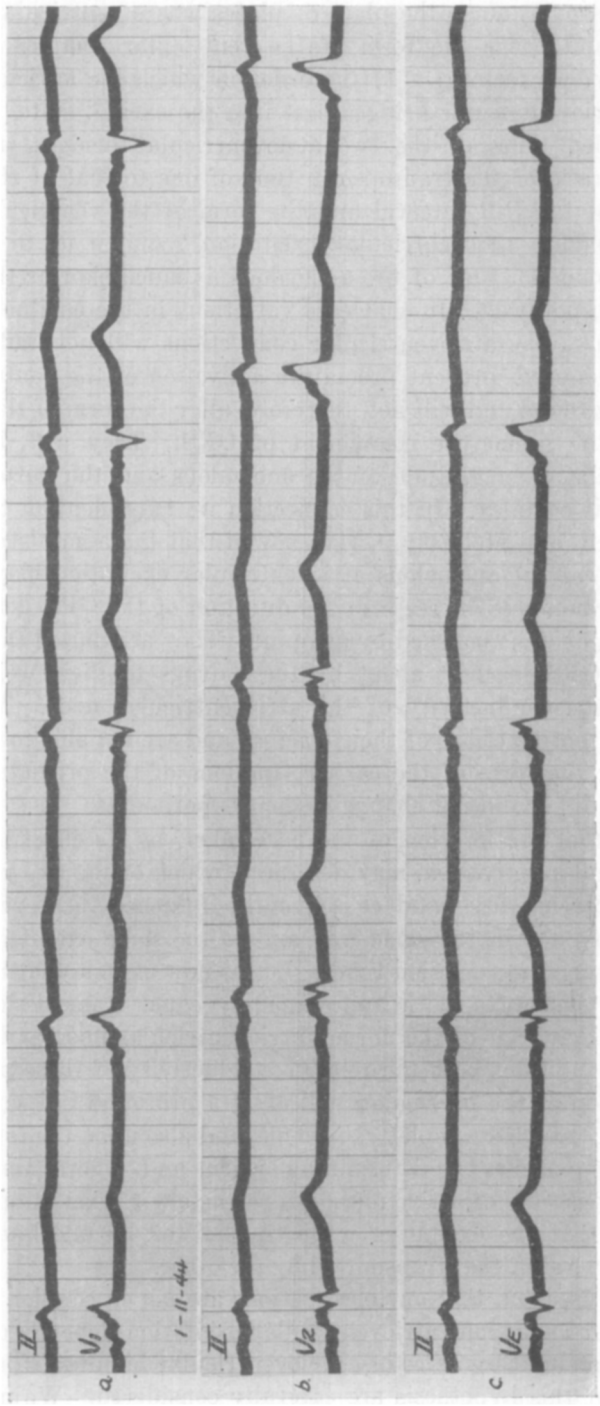

Fig. 19.—Case 10. Transitions from atrioventricular rhythm arising in the upper levels of the atrioventricular node (*a*), or vice versa (*b* and *c*), taken with the camera running at a speed of 75 mm. per second. Precordial leads taken simultaneously with Lead II. Note the transitional form of the third complex in *a* and of the corresponding complex in *c*.

spread to the ventricle both by the anomalous and by the normal route. Under these circumstances the place or places where ventricular excitation begins, and hence the form of the ventricular complex, will be determined by two factors: (1) the order in which the auricular ends of the two atrioventricular bridges pass into the excited state, and (2) the transmission times of the two atrioventricular bridges, and particularly the ratio of the transmission time of one to that of the other. The brevity of the P-R interval and the form of the ventricular complex in anomalous atrioventricular excitation requires us to suppose that the transmission time of the anomalous, is much shorter than that of the normal, pathway. In that case variations in the conductivity of either or both of the atrioventricular connections will not, unless they are very pronounced, prevent premature activation of the ventricles by the anomalous route, and will not, therefore, alter the form of the earlier fractions of the premature component of QRS. They will, however, change the relative magnitude of the anomalous and the normal components of this complex. In this connection we may mention the work of Fox, Travell, and Molofsky,[14] who showed that the administration of digitalis in a case of anomalous atrioventricular excitation may be followed by a pronounced increase in the duration of the QRS deflections, and that this effect is abolished by atropine. They attributed the alterations in the QRS brought about by these drugs to their well-known effects upon the conductivity of the atrioventricular node. We have examined the reproductions of their tracings and are not able to say with certainty that the form of the earlier fractions of the premature component either did or did not change in their experiments.

It is clear that the location of the pacemaker, by its effect upon the order of auricular activation, may determine which of the two pathways the impulse reaches first, and consequently influence the form of the ventricular complex in the same way and to the same extent as minor variations in their transmission times. Let us now assume that there are two accessory pathways, with approximately equal transmission times much shorter than that of the normal atrioventricular node and bundle. Under these conditions both the relative conductivity of these pathways and the location of the pacemaker will exert a profound influence upon the contour of the QRS complex as a whole, and also upon the form of its premature component by determining when and where ventricular excitation begins. Since active muscle is refractory to excitation, it may even happen that the excitation wave transmitted by one bundle will prevent the arrival of that transmitted by the other.

It seems to us, then, that our observations are not in conflict with the hypothesis that anomalous atrioventricular excitation depends upon the existence of one or more accessory atrioventricular bundles, if all of the implications of this hypothesis are carefully considered. We may suppose that, in Case 10, two bundles of this kind were present; that the changes in the form of the ventricular complex which accompanied the

onset of atrioventricular rhythm in this case were due to the effect of the order of auricular activation upon the time when excitation of the auricular end of each of these bundles began; and that the changes in the form of the ventricular complex which occurred independently of changes in the location of the pacemaker after the administration of quinidine were dependent upon unequal changes in the transmission times of the two pathways brought about by this drug.

It is possible that the changes in the ventricular complex that accompanied the onset of atrioventricular rhythm may be satisfactorily accounted for in another way. It is known that transitions from sinus rhythm to atrioventricular rhythm are sometimes accompanied by alterations in the ventricular complex even when the ventricles are activated in the normal manner. As far as we know, such changes are ordinarily of very small magnitude, and have been described only in connection with ectopic rhythms arising in the lower levels of the atrioventricular node. An isolated instance in which the onset of a rhythm of this sort was associated with very striking modifications of the ventricular deflections in standard limb leads has, however, been reported.[2] This phenomenon is apparently due to imperfect distribution of the excitation process to all of the fibers of the His bundle. The occurrence of reciprocal rhythm in association with a low atrioventricular pacemaker is further evidence that cross conduction in the His bundle may be limited, and that this structure and the atrioventricular node do not always function as a single, uncomplicated channel for the transmission of impulses. Nevertheless, we doubt very much whether faulty cross conduction in the His bundle played an important role in the production of the phenomenon under consideration. In the first place, it is difficult to understand why the His bundle and its subdivisions should conduct normally when the ectopic center was on the ventricular side of the junctional tissues, and abnormally when this center was in the upper part of the atrioventricular node.

The other evidence bearing upon this problem is of an indirect kind. We have pointed out that the differences between the anomalous ventricular complexes associated with the more common of the two atrioventricular rhythms observed, and those that represent responses to the sinus node are similar in character to the differences between the ventricles complexes recorded in the cases of Group A and those recorded in the cases of Group B. In both instances these differences were pronounced in the leads from the right side of the precordium, and slight in the leads from the left side of the precordium and from the auricular levels of the esophagus. It is evident that, in Case 10, they were dependent upon differences in the order of ventricular activation. We infer that the differences between Group A and Group B had a similar origin. On the other hand, there is no reason to suspect that the differences between these two groups of cases were in any way dependent upon faulty cross-conduction in the His bundle, and, consequently, it seems

unlikely that this played a part in determining the differences dependent upon the location of the pacemaker in Case 10. If these inferences are justifiable we must suppose that, in the cases of Group A, the ventricular end of the hypothetical accessory bundle was not in precisely the same location as in the cases of Group B, and, finally, that, in Case 10, there were two accessory bundles, one similar to that present in the cases of the first group and one similar to that present in the second.

PAROXYSMAL TACHYCARDIA IN ANOMALOUS ATRIOVENTRICULAR EXCITATION

Of the various types of paroxysmal rapid heart action that have been observed in cases of anomalous atrioventricular excitation, simple paroxysmal tachycardia of supraventricular origin is by far the most common. Its actual frequency is difficult to estimate, because relatively few cases of anomalous excitation in which cardiac symptoms are lacking are discovered.

Many years ago, Mines[16] produced circus contraction in rings of muscle cut from reptilian hearts in such a way as to include auricular and ventricular tissue and two atrioventricular junctions. The circulating excitation waves set up in these rings spread through the auricular and ventricular segments in succession, crossing one junction in the normal, and the other in the opposite, direction. It was suggested by the experimenter that paroxysmal tachycardia in man might be due to a similar mechanism.

The hypothesis that anomalous atrioventricular excitation is due to the presence of an accessory atrioventricular bundle has revived interest in Mines' conception of the nature of paroxysmal tachycardia. The hypothetical anomalous, and the normal, atrioventricular bundle provide the two atrioventricular junctions present in his experiments, and it has been postulated that, under proper conditions, an impulse might pass from the auricles to the ventricles by way of the atrioventricular node and bundle, and, by returning to the auricles via the anomalous bridge, initiate circus contraction. This is a plausible hypothesis, but as yet no direct evidence pointing to the existence of the postulated mechanism in an observed attack of paroxysmal tachycardia has appeared. We shall, therefore, call attention to some peculiarities of the paroxysms recorded in one of the cases of our series.

In some of the electrocardiograms taken in Case 7 there are extrasystoles represented by QRS complexes of normal outline which are immediately followed in Lead I by an inverted, and in Lead III by an upright, P wave (Fig. 20, *c*). The paroxysms of tachycardia appear to be made up of a rapid succession of beats of this type (Fig. 20, *c* and *d*). On two occasions the onset of a paroxysm was recorded (Fig. 20, *a* and *b*). In both instances the complexes of the beats of sinus origin which immediately precede the ectopic rhythm are of the anomalous type. The first ectopic beat is represented by ventricular deflections of more

normal outline, but the ventricular complexes of the first few paroxysmal beats differ considerably in form from those that follow them. The differences are of the kind usually attributed to aberrant intraventricular conduction. Since the first QRS complex of the abnormal rhythm is not preceded by a P wave, the ectopic rhythm must be ascribed to impulses arising in the lower levels of the junctional tissues. It is possible that the impulses responsible for the paroxysmal beats, as well as those that

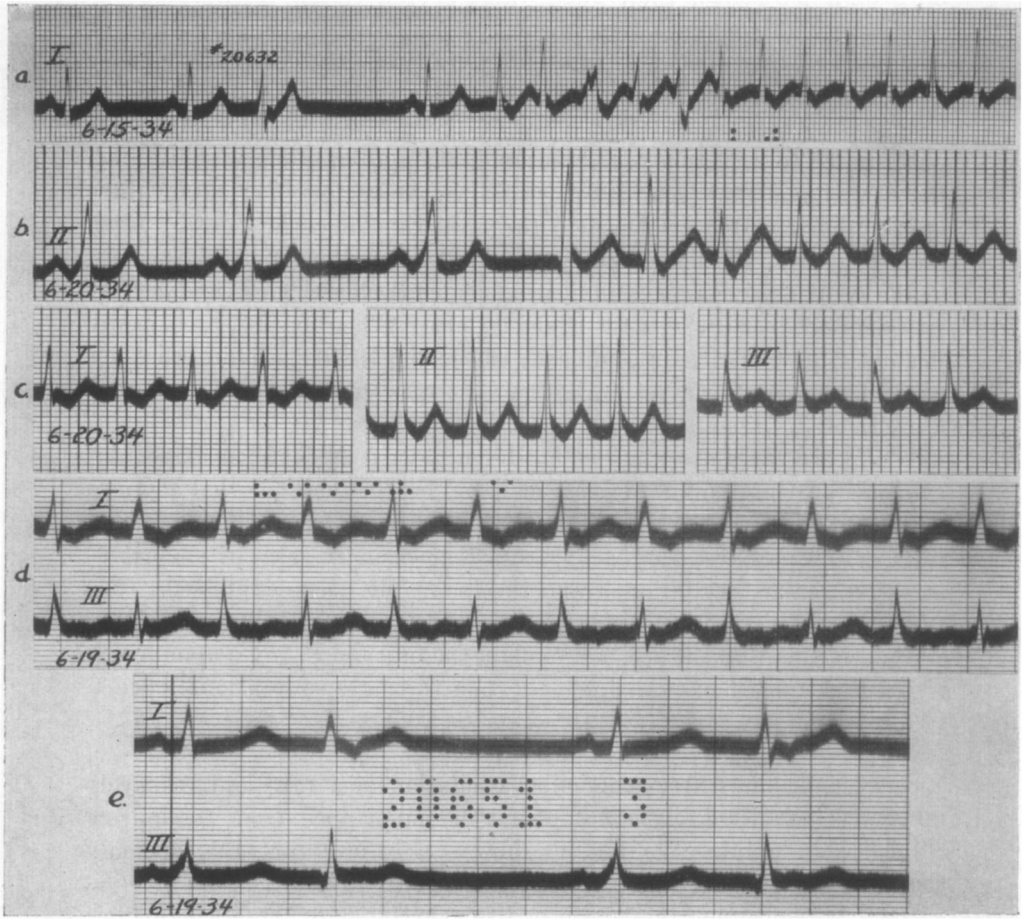

Fig. 20.—Case 7. *a* and *b*, Onset of paroxysmal tachycardia on two different occasions. *c*, Paroxysmal tachycardia on June 20, 1934; note negative P waves in Lead I and upright P waves in Lead III. *d*, Paroxysmal tachycardia on June 19, 1934. There is alternation of two types of ventricular complexes; compare with *e*, in which similar types of ventricular complexes represent extrasystoles of atrioventricular origin.

provoked the single extrasystoles, reached the auricles by the same route as in ordinary atrioventricular rhythm. The sole objection to this supposition is that it does not explain why the P waves are inverted in Lead I and upright in Lead III, and not the reverse, as is almost always, if not invariably, the case when the auricles are activated by the retrograde transmission of an impulse through the atrioventricular

node. The unusual character of the P waves led us to consider another possibility. Suppose that the path through the atrioventricular node was blocked when the ectopic impulse was liberated, and that the peculiar P waves represent auricular excitation by impulses conducted from the ventricles to the auricles via the anomalous bundle. In that case the excitatory process could return to the ventricle by the normal route, and thus initiate a paroxysm. It has been pointed out that, at the beginning of a paroxysm, there was evidence of aberrant intraventricular conduction. To explain this we may postulate that the junctional or ventricular tissues had not, as a rule, recovered completely at the time when the ectopic impulse was liberated. As a result, many extrasystoles, followed by retrograde stimulation of the auricles via the anomalous path, failed to initiate a paroxysm, and, when a paroxysm was initiated, there was aberrant conduction until the refractory period of these tissues shortened in response to the reduction in cycle length. Even during some of the longer paroxysms there was alternation of the form of the ventricular complex, indicating that intraventricular conductivity was depressed (Fig. 20, *d*). It should also be mentioned that in many instances the ventricular complexes of the single extrasystoles were not accompanied by P waves, indicating that retrograde stimulation of the auricles often failed.

We record these observations not because we consider them important evidence bearing on the problem at issue, but in the hope that those to whom the opportunity may come will make a careful study of the mechanism of the paroxysms of tachycardia that occur in cases of anomalous atrioventricular excitation from the point of view expressed. We frankly admit that the suggested interpretation of them is highly speculative, and that it does not explain the unusual P waves upon which it is based much more satisfactorily than the more conventional one.

FURTHER DISCUSSION AND CONCLUSIONS

Since anomalous atrioventricular excitation is a rare and relatively innocuous condition, it is not of major importance from the purely clinical standpoint. We do not, however, believe that this anomaly should be completely ignored by military and insurance examiners as without bearing upon the health or life expectancy of those who exhibit it. The paroxysms of tachycardia to which such persons are predisposed may certainly lead to death, and there is no reason to suppose that they may not also give rise on occasion to sudden giddiness, faintness, or syncope. In the pilot of an airplane, symptoms of this kind could result in disaster. Nor does the lack of a history of such paroxysms in the past necessarily mean that they will not occur in the future. It would seem wise, therefore, to look upon this condition as always involving such hazards as attacks of extremely rapid heart action entail.

The importance of this disorder and the interest it has aroused among cardiologists depend, however, not upon its clinical implications, but

upon its bearing on our conceptions of the mechanisms responsible for the normal sequence of auricular and ventricular contraction and the interval which separates them. We must ask ourselves whether it is possible to explain satisfactorily the electrocardiographic anomalies which characterize it without revising ideas concerning these mechanisms that seem to rest upon a solid experimental foundation. We refer particularly to the belief that in the normal mammalian heart the cardiac impulse is transmitted to the ventricles by the successive activation of the components of a specialized muscular or neuromuscular pathway, consisting of the atrioventricular node, the His bundle, and the subdivisions thereof. There is abundant experimental evidence that section of this bundle or of both its right and left branches produces complete atrioventricular dissociation. The action currents of these structures and of the node have, however, never been recorded, and there is no direct evidence available as to exactly what happens to the cardiac impulse in the latter.

We must suppose that the electrocardiographic peculiarities encountered in the syndrome under consideration depend upon an anatomic or a functional anomaly. Unless we abandon our present conceptions, any anomaly of the first sort must involve either (1) the existence of one or more muscular or neuromuscular accessory bridges extending from the auricular to the ventricular myocardium, or (2) some structural peculiarity of the atrioventricular node, bundle, or bundle branches. These two possibilities are not completely distinct, for it matters little whether the accessory channel for the transmission of impulses is widely separated from, or lies within, the same sheath as its fellow. With reference to the manner in which an accessory bundle might arise in the course of the heart's development, we may refer to observations on the junctional tissues of the embryonic mammalian heart and of the mature heart in lower orders of animals made by Keith and Flack,[17] Keith and Mackenzie,[18] Mackenzie,[19] and Mall.[20]

According to these authors, the mammalian atrioventricular bundle is derived from the invaginated portion of the auricular canal. Portions of this funnel atrophy as the lateral endocardial cushions, which form the parietal auriculoventricular valves, develop. In some fish the ring is interrupted at two points, so that the funnel is replaced by two strands. When the single ventricle of the lower forms is divided into two chambers, that part of the funnel which lies on the left side entirely disappears, so that the connection between auricular and ventricular muscle is present only on the right side. This reduction continues until, in the mammal, only the His bundle remains. In the monotreme echidna there is, in addition to an atrioventricular bundle similar to that of mammals, another leash of tissue which descends to the ventricles in the posterior angle between the parietal and septal valves on the right side. The sequence of changes by which the His bundle is de-

rived from the invaginated atrial canal was observed by Mall in human embryos.

It is logical to suppose, then, that any accessory bundle in the human heart must represent some remnant of the invaginated auricular canal. One difficulty with this supposition is that the electrocardiograms of fish, amphibians, and reptiles, and of the chick embryo display a conspicuous P-R interval[21] which, in comparison with the other intervals of the curve, is not very different from that of the normal human electrocardiogram. Why, then, should an accessory bundle derived from the invaginated auricular canal conduct the cardiac impulse with much greater speed than the normal atrioventricular bridge? Eckey and Schäfer[22] have ascribed the anomalous component of QRS in cases of the Wolff-Parkinson-White syndrome to the action currents of remnants of the original atrioventricular funnel persisting in the fully developed heart. Without considering other objections to this hypothesis, we may point out that the component in question seems much too large to be accounted for in this way.

Of the various hypotheses that have attributed anomalous atrioventricular excitation to a physiologic, rather than a structural, anomaly, the one that deserves most serious consideration is that which ascribes this disorder to the direct stimulation of ventricular fibers by the action currents of adjacent auricular muscle. Attempts to excite the ventricles to contraction in this way in experiments on animals have thus far failed, but if it should be proved possible, an anomaly of this kind would account as well as a hypothetical accessory muscular bridge for all of the phenomena observed. It should be noted, however, that observations pointing clearly to the presence of partial block in the accessory pathway would greatly strengthen the view that the anomaly is structural.

In our opinion, there has not yet been advanced any tenable hypothesis which ascribes electrocardiograms of the kind under consideration to anomalies that involve no part of the heart other than the normal junctional tissues. To be satisfactory, any hypothesis of this sort must explain the following observations relating to electrocardiograms of this type: (1) the QRS complex seems to be made up of a normal component, which begins at the normal time, and an anomalous component which begins several hundredths of a second earlier; (2) the precordial electrocardiogram is very different from the precordial electrocardiograms obtained in bundle branch block, complete or incomplete, right or left; (3) the form of the ventricular complex, including that of the premature component of QRS, is sometimes determined by the order of auricular excitation; (4) when two pacemakers, one in the sinus node and one in the upper levels of the atrioventricular node, are sending out impulses almost simultaneously, QRS complexes transitional in form between those characteristic of the sinus rhythm and those characteristic of the atrioventricular rhythm may occur. This observation implies that in one and the same cycle two supraventricular impulses may reach

the ventricles without interfering one with the other; (5) when the pacemaker shifts to the lower levels of the junctional tissues, the ventricular complex assumes the normal form. This suggests that anomalous atrioventricular excitation is impossible when ventricular excitation occurs simultaneously with, or precedes, auricular. It seems to us that any hypothesis that can satisfactorily account for these phenomena must provide more than one distinct channel for the transmission of impulses from auricles to ventricles.

CONCLUSIONS

The form of the ventricular complex in unipolar leads from the esophagus, precordium, and other parts of the thorax suggests that, in anomalous atrioventricular excitation, the dorsal wall of the ventricles is activated prematurely by impulses of supraventricular origin. There is evidence also that the normal atrioventricular node and bundle continue to function in this condition.

There are two types of cases, which differ as regards the form of the ventricular deflections in leads from the right sternal margin and adjacent parts of the right side of the thorax. The differences between these two types of cases are due, at least in part, to differences in the order of ventricular activation.

The anomalous ventricular complex assumes the normal form when the pacemaker shifts to the lower levels of the junctional tissues.

In some cases the location of the auricular pacemaker determines the form of the premature component of the QRS group, as well as that of the anomalous ventricular complex as a whole.

Our observations support the view that, in this disorder, impulses pass from the auricles to the ventricles not only by way of the atrioventricular node and His bundle, but by one or more additional channels.

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ADDENDUM

The monograph of Richard F. Öhnell, entitled *Pre-Excitation, A Cardiac Abnormality*, Stockholm, 1944, P. A. Norstedt & Söner, was not available when this article was written. In one of Öhnell's cases careful histologic examination of the atrioventricular junction disclosed an accessory atrioventricular bundle about 6 mm. long which connected the myocardium of the left auricular wall with the subepicardial myocardium of the left ventricle. This bundle was dorsal to the mitral orifice and about 4 cm. from the ventricular septum. An accessory bundle of this sort could explain the occurrence of electrocardiograms of the kind obtained in cases which we have placed in Group A.