Anomalous Atrioventricular Excitation Produced by Catheterization of the Normal Human Heart

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During the withdrawal of a catheter-electrode through the right ventricle of 2 normal subjects the electrogram displayed briefly a short P-R interval and an aberrant QRS. In the simultaneously recorded standard lead, the phenomenon simulated what is seen in the Wolff-Parkinson-White syndrome. The findings are presented in support of the physiologic, rather than anatomic, explanation for anomalous atrioventricular excitation.

SODI-PALLARES and his associates\(^1\) have reported on the nature of the intracardiac deflections when the standard electrocardiographic leads displayed the characteristics first described by Wolff, Parkinson, and White.\(^2\) As part of the study they were able to produce records of a similar type in dogs. This was accomplished by tapping the upper portion of the interventricular septum with a probe which had been pushed through the free wall of the right ventricle. Following such manipulation there were, occasionally, runs of a rhythm characterized by a normal P wave, a short P-R interval, and an aberrant QRS with a slurred initial ventricular deflection (delta wave, anomalous component\(^6\)) not unlike that seen in anomalous atrioventricular excitation\(^6\) in man.

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* Since the electrocardiogram under consideration may occur without the other features of the syndrome described by Wolff, Parkinson, and White,\(^2\) it is desirable to have some designation for it. Among those suggested have been: short P-R, bundle branch block syndrome,\(^4\) pre-excitation,\(^5\) and anomalous atrioventricular excitation.\(^6\) The evidence against a block of a bundle branch being the mechanism responsible for the record seems convincing enough to make the first name undesirable. Pre-excitation\(^1\) is a good term. It is brief, and embodies the concept of the mechanism involved without stating its specific nature. However, it fails to tell in what chamber the phenomenon is occurring, and whether the usual or other pathways of ventricular excitation are involved. Anomalous atrioventricular excitation is perhaps best, although the objection to this term is the fact that anomalous excitation of the kind it implies is usually, though not always,\(^4\) limited to the ventricles or a part of them.\(^5\) Despite this shortcoming it is believed to be the most desirable designation.

In the course of a comprehensive study of the intracardiac potentials developed during electrical activity of the human heart, we have on two occasions while manipulating the catheter seen what appeared to be anomalous atrioventricular excitation in normal subjects. It is the purpose of this report to describe the records obtained.

Case Summaries

Patient QWI was a 45 year old Negro admitted to the hospital because of a convulsive seizure, the second since he sustained a fracture of the skull six months before admission. The course and laboratory data, including an electroencephalogram and x-ray examination of the skull, confirmed the clinical impression of post-traumatic epilepsy. Cardiac examination, including an electrocardiogram (fig. 1) and a teleroentgenogram, gave entirely negative findings. There was no history of paroxysmal tachycardia. Cardiac catheterization was performed one week after the last convulsion.

Patient PAQ was a 49 year old Filipino waiter, resident in the United States for twenty-two years, admitted to the hospital because of intermittent chills and fever of two weeks' duration. Two days after admission a low-grade fever subsided. The subsequent course and laboratory data favored a diagnosis of grippe. Cardiac examination, including an electrocardiogram (fig. 3) and teleroentgenogram, was productive of negative findings except for a soft
systolic murmur heard at the apex. There was no
history of paroxysmal tachycardia. Although exam-
ination of the spinal fluid revealed no abnormality,
serologic reactions for syphilis (Wassermann and
Kahn) were positive. He was regarded as having
late latent syphilis. Cardiac catheterization was per-
formed one week after admission.

METHODS

Details of the methods will be reported else-
where. Briefly, two string galvanometers were used,
one, with an amplifier in circuit, to record an in-
tracardiac lead, the other to record in the usual way
a lead somewhere from the surface of the body. The
intracardiac exploring electrode was a solid woven
catheter* with a silver cylinder, 3 mm. in length, at

\[ \text{Fig. 1.—Electrocardiograms of Patient QWI. In the upper row are the standard leads (I, II, III), and the augmented extremity potentials (aV\\text{R}, aV\\text{L}, aV\\text{F}) recorded at normal sensitivity of the string (1 mv. = 1 cm.) The lower two rows are a continuous record of an intracardiac lead at 0.15 normal sensitivity made simultaneously with Lead I as the tip of the intracardiac catheter was slowly withdrawn from a point in the pulmonary conus (V\\text{RV3}) toward the lower part of the right ventricle near the apex (V\\text{RV4}). Withdrawal of the catheter was halted just before the standardization seen in Lead I of the lowest record. Just preceding this the shortening of the P-R interval (numbers under the intracardiac lead) and the considerable change in the QRS complex of both leads can be seen without significant change in the length of cycles (numbers in parentheses above the intracardiac lead). The new form continues to the end of the strip. Particularly to be noted is the slurred ascending limb of the R wave in Lead I, and the simultaneity of the peak of this deflection with the notch on the ascending limb of the aberrant QRS complex as well as with the nadir of the S wave of the earlier supraventricular complexes recorded from inside the heart.}

\[ \text{The simultaneous Lead I is distorted by somatic tremors. Its smaller size, compared to the Lead I in the top row, is probably explained on the basis of increased resistance across this lead which developed as the electrode jelly dried in the course of the experiment. This tended to increase the relative size of the short circuit through the central terminal because a common electrode was used on each extremity. Time lines occur every 0.2 sec.} \]

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\[ \text{* Manufactured by the U. S. Catheter and Instrument Co., Glens Falls, N. Y.} \]
making continuous tracings at any time that the catheter is being moved in the heart. In the first patient (PAQ), the change occurred while the tip was being moved from one point to another in the right ventricular cavity. Both locations were probably in the outflow tract of this chamber. In the second

patient (QWI), a short P–R interval and aberrant QRS complex developed on movement of the electrode from one location to another in the right pulmonary artery. It is likely that more prox-

Fig. 2.—Patient QWI. Two strips taken from a continuous record made as the electrode was withdrawn from just below the pulmonary valve into the pulmonary conus. The upper string shadow in each row (RV2A) is the intracardiac lead at 0.15 normal sensitivity of the string. The lower is Lead I (I).

In the upper row there is a ventricular premature systole. Within the heart it begins with a small summit, presumably because its focus of origin was not at the tip of the catheter but at some more proximal (or distal) point. In later parts of the record (not shown) ventricular premature systoles occurred in which there was no initial summit. In this record the appearance of positive S–T displacement in systoles after the premature one is due to development of an injury potential from endocardial pressure of the electrode.

In the lower row, which begins four seconds after the end of the first, the injury potential on the intracardiac electrode has varied and given rise to a low, slurred summit just after the QRS complex of all four intracardiac complexes. The first two complexes in this row differ from the last two but in the simultaneous Lead I the corresponding ventricular deflections are the same in all four. Further, although the P–R interval is shortened within the heart (0.136 and 0.148 second), this is not evident in Lead I (0.170 second). The beginning of the large, intracardiac, ascending ventricular deflection (ascending limb of QS or S) is simultaneous with the peak of the R wave of Lead I in both types. In the first two complexes, then, the rapid initial downward deflection followed by smaller, slower waves in both directions preceding the peak of the R wave, together constitute the anomalous component or the delta wave (representing ventricular muscle excited anomalously from an ectopic focus at the tip of the electrode superimposed upon and obscuring the earliest deflections resulting from normal supraventricular excitation). Unlike what is seen in figure 1, the delta wave is not premature enough or of sufficient magnitude to be reflected in Lead I either by a shortening or distortion of the P–R interval or segment, or by a distortion of the QRS complex. Further, this deflection varies somewhat in the first two intracardiac complexes as does the P–R interval. The numbers in parentheses above Lead I show the length of the cycles between beats.
mal parts of the catheter, particularly those just below the pulmonary valve where the curvature of the catheter is usually sharper than elsewhere, made pressure on the ventricular wall to cause the abnormal electrocardiogram which resulted.

The lower record in these lines is the standard Lead I. The standardization in the latter on the lowest line indicates the point at which withdrawal of the intracardiac electrode was discontinued.

Just before withdrawal was halted, both the electrogram and the electrocardiogram changed considerably. In the former, the R wave was replaced by a deep, broad, negative deflection with a notch on its ascending limb simultaneous with the peak of R in Lead I. The RS-T seg-

RESULTS

The intracardiac tracings, the standard leads, and the extremity potentials of the 2 patients are shown in figures 1 and 3. In the first (Patient QWI), the record obtained as the electrode was being withdrawn from the pulmonary conus to the lower part of the right ventricle near the apex is the upper tracing in the second and third lines labelled $V_{RV3}$ and $V_{RV4}$. The lower record in these lines is the standard Lead I. The symbols have the same meaning as in figure 2. The lowest row is a record of the potential within the right pulmonary artery ($V_{PA1}$) recorded simultaneously with Lead I(I) as the intracardiac electrode was withdrawn slowly to a more proximal point in the right pulmonary artery. The numbers on the intravascular lead give the duration of the P-R intervals; the numbers in parentheses above Lead I give the duration of the cycle lengths. The shorter cycle lengths preceding the aberrant ventricular deflections were probably fortuitous, for they occurred elsewhere preceding normal ventricular complexes. Sensitivity of the string was normal for all but the intravascular records where it was 0.4 normal (0.4 N).

In the simultaneously recorded leads the appearance of the two middle QRS complexes preceded by shorter P-R intervals are to be noted. The P wave varies somewhat in the pulmonary artery, as is usual with movement of the electrode. No change is seen in this deflection in Lead I. It is believed that the QRS complex of the last cycle in both leads is slightly widened as a result of impaired conduction in the right bundle branch caused by pressure of the catheter. Subsequent ventricular deflections in the strip were similar to those of the first complex shown.
ANOMALOUS ATRIOVENTRICULAR EXCITATION

ment became elevated and the T wave positive. This was in contrast to the normal intracardiac deflections in which the T wave was inverted, and the initial positive deflection, R, was gradually becoming larger and the S wave slightly shallower as the electrode was withdrawn. As might be expected in anomalous atrioventricular excitation, the S wave of the normal complex was not much altered, and could be identified as the notch on the ascending limb of the abnormal QRS complex. Also, as might be expected, the duration of the P-J interval* was the same in both types of systoles. In the simultaneous electrocardiogram (Lead I) the ascending limb of the R wave became slurried (anomalous component, delta wave), and the T wave almost isoelectric, simulating what is seen in the Wolff-Parkinson-White syndrome. The P-R interval, measured with greater accuracy in the electrogram, shortened from 0.149 second to 0.116 second, and was quite constant in all of the anomalous complexes. It also became shorter in Lead I. The P wave did not change. The cycle lengths with either type of ventricular excitation did not vary significantly or according to any recognizable pattern. The anomalous ventricular excitation persisted for twelve beats, at the end of which time recording was discontinued. When it was begun again, less than a minute later, the anomalous excitation had ceased.

Earlier in this experiment as the catheter was being withdrawn from one point to another in the conus itself the record shown in figure 2 was obtained. The upper intracardiac record in this figure shows simply a ventricular premature systole. The lower record differs from figure 1 in that the simultaneous standard Lead I does not show a delta wave even though the form of the initial parts of the intracardiac QRS complex in the first two complexes is quite different from the last, more normal two. Also, the P-R interval of the anomalous beats is slightly shorter than the normal in the lead from within the heart, and more so in the first (0.136 second) than in the second (0.148 second), but it is not shorter in the external lead.

These data suggest that in this instance the myocardium excited anomalously was of such small extent that the phenomenon was not reflected in the QRS complex of Lead I. Even though ventricular excitation was slightly premature, no shortening of the P-R interval occurred in Lead I.

If one were to grade the three different types of anomalous beats shown, on the basis of the extent of the anomalously excited muscle, the completely aberrant record within the heart and in the standard lead (premature systole) would be first (fig. 2, upper record); the partially aberrant record found both within the heart and in Lead I would be second (fig. 1); and the aberrant record found within the heart but not in Lead I would be third (fig. 2, lower record). It would appear that prematurity of the ectopic beat, whatever its cause, was the factor responsible for the absence of or different nature of anomalous deflections when they occurred.

In the second patient (PAQ, fig. 3), the catheter was moved only a few centimeters while the tip was in the right pulmonary artery. At the end of this exploration it was still well to the right of the bifurcation of the pulmonary stem as determined on a frontal fluoroscopic projection. During the withdrawal, a record (VPAQ) was made simultaneously with Lead I (lowest line in fig. 3). In the figure the first beat may be regarded as the normal. The middle two are abnormal. In these the slurried ascending limb of R, the inverted T, and the short P-R interval in Lead I accompanied in the intravascular lead by a large, early, positive deflection rather than a late one, are easy to see. P waves are all the same in the standard lead but vary somewhat in the arterial lead as expected when the electrode is moved in the pulmonary artery. Cycle lengths vary as shown by the numbers in parentheses above Lead I. Variations of this degree were found elsewhere in the record and were not considered to be significant.

The last QRS complex in this figure differs from the first in that it is wider and this widening affects principally the R wave in the pulmonic lead and the S deflection in Lead I. The T wave in the latter is also slightly taller than

* The interval measured from the beginning of the P wave to the junction of the QRS complex and the RS-T segment.
in the normal control. The precise cause of these variations, seen in one other systole of the record, is unknown although impaired conduction through the right bundle branch produced for one beat by pressure of the catheter is a possibility.

**DISCUSSION**

The data presented may be interpreted in one of three ways: (1) the catheter increased the rhythmicity of a center in the ventricular muscle which then fortuitously discharged at a rate similar to but preceding that of the sinoatrial node; (2) the catheter itself, extending as it does through the right atrium, was moved by contraction of this chamber to a sufficient degree to make more distal parts of it in the ventricle stimulate the latter prematurely, probably in the region of the upper right side of the interventricular septum; (3) the catheter increased the irritability of a ventricular center which was then discharged prematurely by atrial systole, either electrical or mechanical.

The first is unlikely because the cycle lengths preceding either a normal or an anomalous beat varied considerably (figs. 1 and 3). That an ectopic focus should discharge fortuitously under such circumstances just before and in relatively fixed relation to normal supraventricular excitation of the ventricles is not probable. The second is possible although the experimental observations on dogs make it clear that a phenomenon similar to anomalous atroventricular excitation may occur after the mechanical stimulus to the upper septum has been withdrawn. On the basis of this finding it is believed that the third interpretation, namely, an increase in irritability of a ventricular focus produced by the catheter, is most likely, and that the discharge of this center depends on either electrical or mechanical events in the atria.

Contraction of the atria as a stimulating factor seems a good possibility, since the "a" wave of the atrial sphygmogram in man begins on the average 0.09 second after the beginning of the P wave. This causes a slight rise in intraventricular pressure which under certain circumstances of heightened irritability of the ectopic focus, such as could be caused by a foreign body (catheter) in the heart, may result in its premature discharge and the development of a short P-R interval and aberrant QRS complex. The time of discharge will depend in part upon the degree of irritability. All varieties of anomaly of the QRS complex, from a simple delta wave in direct leads with no change in the P-R interval or in the QRS complex of semidirect or indirect leads, to considerable abbreviation of A-V conduction time and complete premature excitation of the ventricles from the ectopic focus, are conceivable under such circumstances.

A question of considerably longer standing than these observations, is whether a mechanism similar to the one reported is responsible for the electrocardiogram seen in the Wolf-Parkinson-White syndrome. In the voluminous literature which has appeared on this type of electrocardiogram, two principal mechanisms for its production have been suggested: one anatomic, the other physiologic. Among the anatomic tracts indicted have been: those similar to bundles described by Kent, the paraspecific connections between the common bundle, the left bundle branch, and the upper septum as described by Mahaim and Winston, and direct connections between the right atrium and the ventricular septum. Since the papers by Holzmann and Scherf and by Wolferth and Wood appeared, suggesting an anatomic basis for the electrocardiogram and indeed for other features of the syndrome, a possible physiologic basis has been almost completely ignored.

In retrospect, one of the fallacies of the anatomic concept has been to ascribe a function of conduction to a tract simply because it has been found to exist at necropsy in a patient known to have shown anomalous A-V excitation during life. It would appear that adequate attention has not been paid to control observations; it is not known what number of subjects will display such tracts at necropsy who never displayed the syndrome during life. If these should turn out to be numerous, and the observations of Glomset and Glomset suggest that they are, the cause of the organic theory

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*a* See the article by Segers, Lequime, and Denolin and that by Öhnell for a complete bibliography up to 1944.
should be seriously weakened. Further, and in addition to many other aspects, it is most difficult to explain on an anatomic basis the presence of the electrocardiographic features of the syndrome at times and their absence at others when the location and the rate of the pacemaker is constant.

Without giving all the pros and cons of the controversy, it seems just as easy to explain most of the features of the Wolff-Parkinson-White syndrome on the basis of an ectopic focus in the ventricles as on the basis of an accessory conducting pathway. It is also just as difficult to explain certain phenomena by the former as by the latter. The evidence presented here does not prove the one concept or disprove the other, but it should revive interest in the physiologic approach to the final solution of the problem.

Summary and Conclusions

1. In 2 patients with normal hearts, electrocardiograms with features usually ascribed to anomalous atrioventricular excitation were produced as an intracardiac electrode was slowly withdrawn through the right ventricle during a continuous electrocardiographic recording.

2. Contact of the catheter with the endocardial surface of the right ventricle resulted in increased irritability of a ventricular center most likely in the septum. During anomalous excitation of the ventricles this center was stimulated and discharged prematurely, possibly by the slight rise in intraventricular pressure ordinarily caused by atrial contraction.

3. The time of discharge of the new center determined the appearance of the simultaneous standard lead. When discharge was slightly premature, as determined from an early, minor, anomalous appearance of the QRS complex in the intracardiac lead, no change in one standard lead, and by inference in the other standard leads, could be seen.

4. Spontaneous physiologic alterations of a similar kind in a ventricular center would explain most of the features of the Wolff-Parkinson-White syndrome, without assuming the existence of one or more accessory anatomic pathways of conduction between the atria and the ventricles.

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