Epicardial Excitation of the Ventricles in a Patient with Wolff-Parkinson-White Syndrome (Type B)

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SUMMARY
Epicardial excitation was explored by means of an exploring electrode during operation on a patient with a large atrial septal defect of the secundum type, whose ECG indicated a Wolff-Parkinson-White syndrome of type B. Very early excitation occurred, 10 msec after the end of the P wave, at the right lateral border, near the atrioventricular sulcus, an area which is located a relatively large distance from the atrioventricular node. Because the epicardial region closest to this node did not show early excitation, it was concluded that in this heart the node was not involved, but that a muscular bypass between the right atrial muscle and the closely adjacent right ventricular surface was responsible. The location corresponds with that described in several extensive anatomic studies of hearts with a WPW syndrome. The right ventricle was activated predominantly or completely by an excitation wave originating in this area; the excitation pattern of the left ventricle did not differ significantly from the normal.

Additional Indexing Words:
Pre-excitation    Atrial septal defect

The Wolff-Parkinson-White (WPW) or pre-excitation syndrome\(^1\) may be diagnosed on the electrocardiographic criteria of a short PR-interval (0.12 sec or less) and increased duration of the QRS complex (0.11 sec or more) because of widening in its initial portion by a slow-rising, slurred deflection (delta wave).

It is impossible to discuss all theories advanced, but most authors\(^2\) accept the view that pre-excitation of a part of the ventricles occurs in this condition, the normal atrial impulse being divided between the normal, slowly transmitting atrioventricular node and an accessory pathway with a much smaller delay, which connects a part of the atria with a closely adjacent basally located region of the ventricles. Acceleration of conduction through an abnormal part of the node is advanced as another explanation.\(^3\) As early as 1932 Holzmann and Scherf\(^4\) indicated two possibilities: bypass of the atrioventricular node and activation of an abnormal ventricular focus by the contraction of the atrium.

The form of the complexes in the unipolar leads from the right precordium is used for differentiation of two types of WPW syndrome.\(^5,6\) In type A with predominantly positive QRS complexes, it is assumed that an accessory muscular connection is located at the posterior side of the ventricles, whereas in type B, where the QRS complexes are mainly negative in these leads, a more anterior location is postulated. Many attempts have been made to study the genesis of the WPW complex. Direct proof of premature activation of a part of the ventricles and the location of this area using exploration of epicardial excitation is still lacking, and also the excitation pattern of the remainder of the ventricles is unknown.

In this study, exploration of the epicardial surfaces of a heart was possible during surgical intervention in a patient with an atrial septal defect of the secundum type, having

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ECG abnormalities, fulfilling the criteria for the diagnosis of a WPW syndrome.

Report of Case

Patient W., a 21-year-old unmarried woman, was short of breath during exertion. Several times in the preceding years she had experienced attacks of tachycardia, but she did not remember the duration of these attacks and how they began or ended.

Physical examination of this well-developed woman revealed a slightly enlarged heart. The auscultatory findings consisted of a protosystolic murmur with ejection sound in the third left intercostal space, a split second sound, and a mid-diastolic murmur at the right lower sternal border. The electrocardiogram (fig. 1) revealed a sinus rhythm of 72 per minute, the P-R time of 0.12 sec, and the duration of QRS complexes of 0.16 sec. A delta wave was clearly present in many complexes. In the right precordial leads, predominantly negative complexes were recorded. The electrocardiographic findings, therefore, may be classified as those of type B of the WPW syndrome. After operation for the atrial septal defect the QRS complexes in all leads were unchanged.

Roentgenological examination of the thorax revealed a slightly enlarged heart; the pulmonary artery was dilated. At catheterization increased oxygen concentration in the blood obtained at the right atrial level was found. The pressures in the right ventricle (27/0 mm Hg) and in the pulmonary artery (27/10 mm Hg) were normal. The relation between the lung and body circulation was 2:1. During operation a large-sized defect of the secundum type was found in the atrial septum, with normally formed and located tricuspid and mitral valves.

Exploration of Epicardial Excitation

Unipolar complexes were recorded immediately after opening the pericardium, using a small-tipped exploring electrode; the reference electrode was a Wilson central terminal. The WPW complexes were present throughout the duration of the exploration.

For reference the right ventricular cavity complex was recorded from an electrode introduced through the middle part of the free wall into the central part of the right ventricle, and the beginning of this complex was used as zero for all measurements of epicardial excitation. A drawing of the exposed surface of the heart was made by an artist during operation, the position of the electrode was indicated by a mark, and checked by a physician also during the operation. It was not possible to explore the lateral left ventricular surface adequately. All complexes were photographically recorded from a high-fidelity machine. Other technical details are described in previous papers. The rapid part of the intrinsic deflection (i.d.) signaled the arrival of the excitatory wave.

Figure 1

Electrocardiogram recorded with a photographic four-channel Hellige apparatus showing sinus rhythm with a frequency of 72 per minute. The P wave has a duration of 0.12 sec and is immediately followed by a broad QRS complex with a duration of 0.16 sec, with prominent delta wave in several complexes. The complexes in V₄R and V₅ are predominantly negative. Therefore, a Wolff-Parkinson-White syndrome, type B, is present.
in the subepicardial muscle layer in contact with the exploring electrode.

**Results**

The unipolar right ventricular cavity reference complex showed an initially positive deflection with a duration of 30 to 35 msec, followed by a deep negative deflection. This positive deflection began to rise immediately after the end of the P wave. All epicardial complexes of the right ventricle except one showed initial positivity too. This exceptional complex with an intrinsic deflection of 79 msec was located at the posterior side of the right ventricle. The downstroke of the QS complex began about 70 msec after the beginning of the right cavity complex. The epicardial complexes from the anterolateral part of the right ventricular surface and the anteroseptal part of the left ventricular wall are shown in figure 2; numbers indicate the time of occurrence of the intrinsic deflection of these complexes.

**Discussion**

The type of epicardial excitation pattern present and the form of the epicardial complexes in our case were completely different from those recorded from hearts with normal conduction in the atroventricular bundle and its main branches.

**Normal Epicardial Time Relations**

In the normal heart, the earliest excitation of the subepicardial muscle occurs near the attachment of the anterior papillary muscle of the right ventricle, at about 30 msec after the beginning of the QRS complex in the reference tracing from the right or left ventricular cavity. From this area epicardial excitation in the human heart spreads in a more or less radial way across both ventricular surfaces, with the posterobasal area of the left ventricle activated last (at 70 to 80 msec). The region near the lateral right atroventricular sulcus is excited at 50 to 60 msec. Excitation of the left anteroseptal and anterior regions occurs at 30 to 40

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**Figure 2**

Unipolar ventricular complexes recorded from several regions of the anterior and posterior surfaces of the ventricles. The complexes are placed on the regions from which they were recorded. The figures indicate the time of occurrence of the intrinsic deflection in the adjacent complex, using the beginning of the reference complex from the right ventricular cavity as zero.
msec, and of the lateral region at about 50 msec. 7 8

Epicardial Excitation in the WPW Heart

In this heart with WPW syndrome, the earliest excitation of a subepicardial muscle layer was found near the right lateral part of the anterior atrioventricular sulcus, 10 msec after the beginning of our reference, which closely followed the end of the P wave. The form of the complex recorded from this region is abnormal and looks like a large diphasic P wave, immediately followed by a slower, less pronounced deflection. This suggests the possibility that the complex was recorded from the atrial surface. In figure 3 this complex and the reference complex recorded simultaneously are reproduced. In both complexes synchronous and shallow P waves are present. The initial positive deflection of this complex begins to rise immediately after the end of the P wave. The fast intrinsic deflection occurs 10 msec after termination of the P wave during the first part of the positive deflection of the ventricular complex in the right ventricular cavity tracing.

It has to be concluded that the right ventricular surface near the lateral part of the atrioventricular sulcus was activated early in the WPW heart. We did not record normal beats without pre-excitation which would have facilitated the estimation of the degree of prematurity in the excitation present. If we assume the presence of normal atrioventricular conduction in this patient, the QRS complex should begin about 0.20 sec after the beginning of a sinus P wave. Because here the P wave has a duration of 0.12 sec, the beginning of the QRS and probably that of the cavity potentials occur about 0.08 sec after the end of the P wave. In the WPW heart the end of the P wave coincides with the very beginning of the cavity complex.

Normal excitation and WPW excitation, therefore, may be compared by adding 0.08 sec to the values found in normal hearts obtained by using the beginning of either the right or the left cavity complexes as zero point. The degree of prematurity of excitation of the earliest point activated in the WPW heart is about 120 msec. In the two adjacent points this amounts to 90 to 100 msec because excitation in normal hearts occurs here 40 to

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**Figure 3**

Unipolar complex from the earliest ventricular front activated, and recorded simultaneously with the unipolar reference complex from the central part of the right ventricular cavity. A small P wave preceding the high positive deflection is clearly visible and occurs synchronously in both complexes.

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50 msec after the beginning of the cavity complex.

Epicardial excitation as indicated by the figures of occurrence of the intrinsic deflection of the ventricular complexes at the anterior and posterior surfaces spreads in a "radial" way from this area toward the anterior and posterior attachments of the ventricular septum, a pattern compatible with that present in ventricular premature beats originating there. With normal conduction in the right bundle branch, the presence of two colliding excitation fronts at the right epicardial surface may be expected, one originating from the pre-activated region, the other one from the area trabecularis, arriving there by way of the right bundle branch. The large degree of pre-excitation is the reason that this pattern was not present in the WPW heart, for the main part of the right ventricle was activated anomalously. It is possible that complete or incomplete right bundle-branch block was present in this patient, but this could account only for an increase in size of the pre-excitated area.

In the postoperative period, runs of nodal premature beats were recorded with QRS complexes of 0.08-sec duration. Therefore normal excitation in the right bundle was possible.

Epicardial excitation times at all parts of the left ventricle in our patient are apparently delayed, but the overall epicardial excitation pattern, showing a movement from the anterior region toward the posterobasal region, resembles the pattern found in normal hearts. There appeared to be a delay in epicardial excitation of about 60 to 70 msec. This figure agrees with the one given above for the difference between the zero points used in the measurements of this WPW heart and the normal hearts.

In the dog stimulation of a region corresponding with the early activated region results in a left ventricular excitation pattern resembling that present during left bundle-branch block: there is double envelopment of the anterior and posterior left ventricular surfaces toward the latest activated area located at the lateral surface of the left ventricle. This pattern is not present here. In view of these considerations it is permissible to assume normal excitation or nearly so in the left ventricular wall.

**Form of the Epicardial Complexes**

The form of the complexes recorded at the early activated region is unusual, replacing the R's commonly found in normal hearts, and explained by the abnormal excitation pattern present. The large positivity of the early activated region beginning immediately after reference occurs because the anomalous excitatory front in the right ventricular myocardium close to the exploring electrode is opposed. The complex with intrinsic deflection at 33 msec also begins immediately after reference and has the same form.

The other complexes, recorded from the right and left ventricular surfaces except one, show a gradually increasing more or less pronounced positive deflection, a delta wave, often followed by a R wave, rapidly increasing in size, sometimes with a notch between the delta wave and the R wave. The QS complex at the posterior surface of the right ventricle begins 70 msec after reference, proving the presence of an "isoelectric" part. For reasons we cannot unravel, the pre-excitation forces did not influence this electrode. In contrast to normal hearts no Q waves were recorded from the left ventricular surface. The excitatory forces, originating at the right lateral border of the right ventricle and progressing in a mainly leftward direction, were responsible for its disappearance.

The initial positivity recorded from the central area of the right ventricular cavity is also caused in the excitatory forces in the free wall of the right ventricle. During cardiac catheterization, identical complexes from the central part of the right ventricular cavity were recorded but no systematic exploration of right ventricular cavity potentials was done.

It is probable that the excitation pattern of the subjuncardial muscle reflects in a general way the time course of excitation in the subendocardial muscle, as recently demonstrated.
in an isolated normal human heart. We are, however, completely ignorant of the excitation pattern in the ventricular septum, especially in the region surrounding the atrioventricular node. However, septal excitatory forces did not play a role in the genesis of the very first part of the ventricular complex in this instance. The relatively large distance between pre-excited area and the atrioventricular node and the absence of early excitation of subepicardial muscle lying closest to the atrioventricular node at the posterior side near the atrioventricular sulcus, make it unlikely that in this instance the atrioventricular node was involved. Therefore we assume that in this patient with type B of the WPW syndrome a pathway capable of conducting an excitatory wave was present which ran from the lower lateral part of the right atrium toward the adjoining part of the right ventricle. This location is in agreement with the findings of several anatomic studies in hearts with a WPW syndrome in which a muscle bundle was found. In the reconstruction of Truex and associates, the bundle was situated in close proximity to the atrioventricular node.

The first right ventricular region activated was not detected, because the spacing of the epicardial points explored was too wide. Therefore, it is not possible to picture the sequence of ventricular excitation in the very first period of ventricular excitation accurately and draw conclusions about the dimensions of the bundle and the intramural location where the transition toward the ventricular myocardium takes place. The sharp positivity with intrinsic deflection at 10 msec, caused by depolarizing of right ventricular muscle, does not prove subendocardial termination with outward spread of the intramural excitatory wave, but may be caused, though less likely in our opinion, by oncoming excitatory forces from a subepicardially located earliest activated region, lying at some distance.

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References


100 Years Ago—Amyl Nitrite for Angina Pectoris

Few things are more distressing to a physician than to stand beside a suffering patient who is anxiously looking to him for that relief from pain which he feels himself utterly unable to afford . . .

Perhaps there is no class of cases in which such occurrences as this take place so frequently as in some kinds of cardiac disease, in which angina pectoris forms at once the most prominent and the most painful and distressing symptom . . .

As I believed the relief produced by the bleeding to be due to the diminution it occasioned in the arterial tension, it occurred to me that a substance which possesses the power of lessening it in such an eminent degree as nitrite of amyl would probably produce the same effect, and might be repeated as often as necessary without detriment to the patient’s health . . .

On pouring from five to ten drops of the nitrite on a cloth and giving it to the patient to inhale, the physiological action took place in from thirty to sixty seconds; and simultaneously with the flushing of the face the pain completely disappeared . . .—T. Lauder Brunton (Life span: 1844-1916): On the Use of Nitrite in Angina Pectoris. Lancet 2: 97, 1867. (See also Sir Thomas Lauder Brunton, F.R.S. 1844-1916. St. Bartholomew’s Hosp Rep 52: 1, 1916.)
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