References
6. Eggleton RC: Ultrasonic visualization of the dynamic geometry of the heart. Presented at the Second World Congress on Ultrasound in Medicine, Rotterdam, June 1973

An Echocardiographic Study of Interventricular Septal Motion in the Wolff-Parkinson-White Syndrome

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SUMMARY Echocardiographic studies of interventricular septal motion were performed in 26 consecutive patients with the Wolff-Parkinson-White (WPW) syndrome and in ten normal subjects. All patients with types A or B pre-excitation were subclassified into groups I to IV on the basis of their echocardiograms utilizing the method of Boineau and associates. In all 14 patients with type A (Group III or IV) pre-excitation, the motion of the interventricular septum and posterior left ventricular wall motion were normal. However, in 11 patients with type B (Group I) WPW an abnormal septal movement was noted. This was characterized in ten patients by an early systolic posterior motion, a subsequent anterior movement in mid systole, and the usual posterior septal motion beginning in late systole. In eight patients, including the one without early systolic posterior movement of the septum, the late systolic posterior movement was interrupted by a prominent septal notch. One patient with type B (Group II) WPW was studied and exhibited normal septal and posterior wall motion.

In one patient with a spontaneous change in the QRS complex from normal to a type B (Group I) WPW pattern, the septal motion was initially normal and abruptly changed following the first WPW beat. The onset of abnormal interventricular septal motion with type B pre-excitation QRS complexes strongly suggests that abnormal septal movement may be related to an altered sequence of ventricular depolarization during right ventricular pre-excitation.

THE ANOMALOUS CONDUCTION and ventricular depolarization occurring in the Wolff-Parkinson-White syndrome has been studied electrophysiologically, but no good correlation has been established between the electrophysiological abnormalities and a mechanical disturbance of contraction. Demonstration of such an electro-mechanical association would be useful in understanding the effects of various types of pre-excitation on ventricular contraction, and could be useful in identifying the site of pre-excitation. Echocardiography provides a reliable noninvasive method for characterizing interventricular septal and posterior left ventricular wall motion.1 2 Abnormal septal motion has been demonstrated echocardiographically in patients with right ventricular volume overload,3 coronary artery disease,4 and in association with conduction abnormalities such as left bundle branch block.5 6 It was the purpose of the present study to characterize interventricular septal motion and left ventricular posterior wall movement in patients with the Wolff-Parkinson-White syndrome.

Methods

Ten normal subjects and 26 patients with typical electrocardiographic and vectorcardiographic patterns of the WPW syndrome were studied. All of the patients were clinically free of cardiac disease and were referred for study solely on the basis of the conduction abnormality. One patient had a history of atypical chest pain, but selective coronary cineangiograms were normal. All patients were subclassified on the basis of their twelve-lead electrocardiogram according to the method of Boineau and associates8 as follows:

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Group I: positive delta wave in I, aVL, V1 and V6; predominant positive QRS forces in I, II, aVF and V5; and predominant negative QRS forces in V1. The site of pre-excitation is in the anterior right ventricle.

Group II: positive delta wave in I, aVL, and biphasic in V1; predominant negative QRS forces in II, III, and aVF, and predominant positive QRS forces in aVL and V5. The site of pre-excitation is in the anterior right ventricle.

Group III: negative delta wave in II, III, and aVR and positive in V1; negative QRS forces in II, III, and aVR, but not as negative as in group II; predominant positive QRS forces in V1. The site of pre-excitation is in the posterior wall.

Group IV: negative delta wave in I and aVL; and predominant positive QRS forces in V1 and V5. The site of pre-excitation is in the posterior left ventricle.

Group V: predominant positive QRS forces in II, III, and aVR, with biphasic QRS forces in I and V1. The site of pre-excitation is in the anterior left ventricle.

Fourteen patients had a dominant upright QRS deflection in the right precordial leads and were classified according to the initial delta wave as having Group III (13 patients) or Group IV (1 patient) pre-excitation. Ten of these 14 type A WPW patients were male and four were female. Their ages ranged from 7 to 57 years with a mean age of 30. Eleven patients, eight males and three females, had negative QRS deflections in the right precordial leads and were classified as having type B (Group I) pre-excitation. The age range in this group was 16 to 51 years, with a mean age of 39. One patient, an asymptomatic 23-year-old man, had an ECG pattern consistent with type B (Group II) pre-excitation and no patients with Group V WPW were available for study.

Echocardiograms were performed with the use of a commercially available ultrasound continuous recording device (Picker, Echoview X interfaced with a Honeywell 1856 strip chart recorder). The recording paper was calibrated with time-distance markers separated by 1 cm in anterior-posterior distance and by 0.5 sec in time. All calculations were made on tracings recorded at 50 mm/sec paper speed. Patients were studied in the supine or left lateral decubitus position with the transducer (2.25 mHz with a focal depth of 5 cm) positioned in the third or fourth left intercostal space. Care was taken to record septal motion below the level of the mitral valve where the inferior portion of the interventricular septum normally moves in a posterior direction during systole. All patients were studied in sinus rhythm with heart rates which ranged from 60 to 86 beats per minute (average 75). There was no significant difference in heart rates among the three groups of patients studied.

The time from the beginning of the QRS complex to the peak amplitude of each septal and posterior wall movement was hand calculated in each of the ten normal subjects, in 14 patients with type A pre-excitation, and 11 patients with type B (Group I) WPW (table 1). The average values obtained from these calculations were then used to draw the composite septal motion and left ventricular posterior wall motion for each of the three groups of patients (fig. 1). Statistical analysis was performed with a Sigma III computer using the Student’s nonpaired t-test.

### Results

An echocardiogram from a typical patient with type A pre-excitation is depicted in figure 2. During ejection the posterior motion of the septum and the anterior motion of the posterior left ventricular wall are nearly synchronous and are no different from those found in normal subjects. Following left atrial contraction there is a slight anterior

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**Table 1. Time Course and Amplitude of Septal and Posterior Wall Motion**

<table>
<thead>
<tr>
<th></th>
<th>Normal Subjects</th>
<th>Type A WPW (Groups III &amp; IV)</th>
<th>Type B WPW (Group I)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Δ-ESM (msec)</td>
<td>—</td>
<td>56 ± 12†</td>
<td>—</td>
</tr>
<tr>
<td>Δ-peak ESM (msec)</td>
<td>—</td>
<td>100 ± 25</td>
<td>—</td>
</tr>
<tr>
<td>Δ-SFM (msec)</td>
<td>110 ± 20</td>
<td>122 ± 26</td>
<td>160 ± 25*</td>
</tr>
<tr>
<td>Δ-end SFM (msec)</td>
<td>378 ± 45</td>
<td>420 ± 75</td>
<td>380 ± 50</td>
</tr>
<tr>
<td>ESM amplitude (mm)</td>
<td>—</td>
<td>3 ± 1</td>
<td>—</td>
</tr>
<tr>
<td>SFM amplitude (mm)</td>
<td>6 ± 2</td>
<td>8 ± 2</td>
<td>4 ± 1*</td>
</tr>
<tr>
<td>SN amplitude (mm)</td>
<td>2 ± .5</td>
<td>3 ± 1**</td>
<td>4 ± 1*</td>
</tr>
<tr>
<td>Δ-PWM (msec)</td>
<td>105 ± 11</td>
<td>98 ± 13</td>
<td>150 ± 38*</td>
</tr>
<tr>
<td>Δ-end PWM (msec)</td>
<td>414 ± 47</td>
<td>485 ± 73</td>
<td>469 ± 75</td>
</tr>
</tbody>
</table>

*Significantly different (P < 0.05) from both normal and type A WPW patients.

**Significantly different (P < 0.05) from both normal and type B WPW patients.

†Mean values = ± 1 se.

Abbreviations: Δ = onset of QRS complex; SN = septal notch; ESM = early systolic posterior wall motion; SFM = systolic septal posterior motion during ejection; PWM = posterior wall motion during ejection.
motion of the septum and a slight posterior motion of the left ventricular wall similar to that found in normal subjects without conduction abnormalities. Normal motion of the interventricular septum and posterior left ventricular wall was recorded in each of the 14 patients with type A (Group III or IV) pre-excitation. Early anterior movement of the posterior left ventricular wall during systole was not seen in any of our patients with type A WPW, although this finding has been described recently by DeMaria and associates in five of seven patients with type A pre-excitation. However, we did not specifically scan the entire left ventricular posterior wall for premature contraction of the endocardium as was done by DeMaria and his coworkers.

In contrast, an echocardiographic recording from a patient with type B (Group I) pre-excitation is illustrated in figure 3. The septal movement is clearly abnormal and shows an early posterior motion of the septum (ESM), a subsequent midsystolic anterior movement, and the usual posterior septal motion in late systole. This finding was present to a greater or lesser extent in ten of the 11 patients with type B (Group I) pre-excitation evaluated in this study.

An echocardiogram from another patient with type B (Group I) WPW is shown in figure 4. In this echocardiogram the early systolic posterior septal motion is less prominent, but an additional feature of the abnormal septal motion found in type B (Group I) WPW patients is illustrated. The usual late systolic posterior movement of the septum is interrupted by a prominent septal notch, which coincides with the peak anterior motion of the left ventricular posterior wall and begins before the opening of the mitral valve (fig. 4). This finding was present in eight of 11 type B (Group I) WPW patients, but has also been observed, to a lesser degree, in normal subjects.

An echocardiogram from a patient whose QRS complex changed from normal to a type B (Group I) pre-excitation pattern during the continuous echocardiographic recording is depicted in figure 5. During normal conduction the septal and posterior wall motion are normal, and there is no septal notching, while each WPW beat is associated with an accentuated septal notch (N) beginning in late systole and continuing into early diastole. The appearance of the prominent septal notch following the onset of pre-excitation indicates that it may result from an alteration in the sequence of ventricular depolarization.

The average time course from the beginning of the QRS complex to each change in the movement of the subsequent septal and posterior wall motion in the three groups of patients is included in table 1, and composite drawings of the average septal and posterior wall motion utilizing the average values for each of the three patient groups is depicted in figure 1. There was no early systolic posterior
motion of the interventricular septum in any of the ten normal subjects, or in any of the 14 patients with type A WPW. However, in ten of 11 patients with type B (Group I) pre-excitation there was an early systolic posterior septal motion which began at an average of 56 ± 12 (SD) msec after the beginning of the QRS complex, and peaked at an average of 100 ± 25 msec after the onset of the delta wave. This was followed by a subsequent mid systolic anterior movement which averaged 60 msec in duration and was followed by the usual late systolic posterior septal motion. The amplitude of the early systolic abnormal septal posterior motion averaged 3 ± 1 mm and that of the second normal posterior septal motion during later systole 4 ± 1 mm. The amplitude of the late systolic posterior movement in patients with type B pre-excitation was significantly reduced (P < 0.02) as compared to its amplitude in normals (6 ± 2 mm) and in type A WPW patients (8 ± 2 mm). In addition, this posterior septal motion began significantly later in patients with type B (Group I) pre-excitation (160 ± 25 msec, P < 0.01) compared to normal subjects (110 ± 20 msec) and type A WPW patients (122 ± 26 msec).

A single patient with type B (Group II) WPW was evaluated in this study and both septal motion and posterior wall motion were normal. No patient with the ECG pattern of WPW subgroup V could be identified for this study.

There was no significant difference in the time of the peak systolic anterior motion of the left ventricular posterior wall in the three patient groups. However, in patients with type B (Group I) pre-excitation, the onset of posterior wall anterior motion occurred significantly later (150 ± 38 msec, P < 0.01) as compared to normal subjects (105 ± 11 msec) and type A WPW patients (98 ± 13 msec) as indicated in table 1.

Discussion

Previous studies concerning the correlation between electrophysiologic and mechanical events in patients with the Wolff-Parkinson-White syndrome have been inconclusive. Using electrokymography to study aortic and pulmonary artery pulsations, Samet and associates were unable to demonstrate an abnormal sequence of ventricular contraction in four of seven patients with type A WPW and in six of eight patients with type B pre-excitation. Subsequently, Bandiera and Antognetti used roentgenkymography to demonstrate sites of early ventricular contraction involving the left ventricle in patients with type A WPW and the right ventricle in patients with type B WPW. These authors were able to demonstrate abnormal ventricular contraction in eight of 11 patients studied, but did not specify which patients had abnormal wall motion.

In our study, abnormal septal motion characterized by an early systolic posterior movement, a subsequent mid systolic anterior motion, and a delay in the usual late systolic septal movement was recorded in patients with type B (Group I) pre-excitation. In the majority of these patients the second posterior septal movement was interrupted by a prominent septal notch. However, the septal motion did not differ from normal variations in any of the 14 patients with type A WPW. This may be due to the difference in the site of pre-excitation, which is in the anterior right ventricle in patients with type B (Group I) WPW, and in the left ventricle in patients with type A (Group III or IV) pre-excitation.

The abnormal interventricular septal motion in type B (Group I) WPW is similar to that recorded in some patients with left bundle branch block. In the latter conduction abnormality, an early posterior movement of the septum beginning 50 msec and peaking 110 msec after the beginning of the QRS complex has been described. The mechanisms responsible for abnormal septal motion in these two conditions may be similar, although in neither case are the exact mechanisms known. However, in both conditions, abnormal septal motion appears related to the altered sequence of ventricular depolarization, since normalization of the QRS complex results in normal septal motion.

The septal notch, which was prominent in the majority of our type B WPW patients, has been noted in some normal subjects and occurs prior to the dicrotic notch of the simultaneously recorded indirect carotid pulse tracing. As indicated in figure 4, it occurs prior to mitral valve opening. The subsequent septal motion continues into early diastole. Whether or not the accentuated septal notch in patients with type B pre-excitation is due to abnormal ventricular depolarization cannot be answered from this study.

In our patients with type B (Group I) pre-excitation there was considerable variation in the amplitude of the early posterior septal movement which was absent in one patient, and in the degree of septal notch. Variability in the extent of abnormal septal motion is to be expected in individual patients on the basis of: 1) different transmission times through the normal pathways; 2) variations in the distance from the sinus node to the location of the accessory bundle;
3) different sites of entrance of the excitatory impulse from the anomalous pathway; and 4) different patterns of ventricular depolarization.

Recently Boineau and coworkers have studied the electrocardiographic variations in patients with the Wolff-Parkinson-White syndrome and demonstrated that dynamic changes in the QRS configuration occurred as a result of changes in atrial activation and the relative input times to the normal and accessory A-V connection. Patients with right ventricular pre-excitation (type B WPW) demonstrated predominantly anomalous excitation and long QRS duration while those with left ventricular pre-excitation (type A WPW) had a greater component of normal depolarization and a shorter QRS duration. In patients with left ventricular pre-excitation, there was epicardial fusion of wave fronts arriving over the normal accessory connections while in right ventricular pre-excitation there was such fusion and excitation spread entirely from the pre-excited depolarization forces. Thus, the more pronounced alteration of depolarization in right ventricular pre-excitation might result in a more marked alteration in ventricular contraction. In our study, definite anomalies of septal motion were found in patients with type B WPW and right ventricular pre-excitation but not in patients with type A WPW and left ventricular pre-excitation. In the latter patients fusion between the normal and aberrant pathways is present. Furthermore, depolarization of the septum should be more profoundly affected by conduction abnormalities when depolarization occurs from right to left rather than from left to right. Kanakis and coworkers have described asymmetric septal hypertrophy in patients with pre-excitation, and Chandra et al. have described mitral valve prolapse and cardiomyopathy in patients with Wolff-Parkinson-White syndrome. However, only patients with no associated clinical heart disease were selected for this study so the effect of the conduction disturbance on septal wall motion could be evaluated independently of other pathologic variables.

The exact mechanism for the alteration of septal motion in patients with type B (Group I) pre-excitation is unknown, but may result directly from the altered sequence of ventricular depolarization or from changes in the timing of hemo-dynamic events. These data indicate that echocardiography is a useful method for identifying abnormal septal contraction patterns in patients with various sites of pre-excitation.

Recent physiologic studies by Gallagher and associates in patients with the WPW syndrome have provided important information concerning the mechanism of arrhythmias, the usefulness of drug therapy and the site for potential surgical intervention. The addition of echocardiography to epicardial mapping techniques for identifying sites of pre-excitation may be useful in the diagnosis and treatment of patients with various subgroups of the Wolff-Parkinson-White syndrome.

References

An echocardiographic study of interventricular septal motion in the Wolff-Parkinson-White syndrome.
G S Francis, P Theroux, R A O'Rourke, A D Hagan and A D Johnson

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