SUMMARY  Echocardiograms of 52 patients with the Wolff-Parkinson-White (WPW) syndrome were investigated. Abnormal patterns of motion were observed in the left ventricular posterior wall (LVPW) and the interventricular septum (IVS). The abnormal LVPW motion was presumably specific for the syndrome and included early onset of the anterior motion which preceded the first heart sound (S1) and a premature peak formation of the anterior displacement which occurred before the second heart sound. The latter was usually followed by a second lower peak. These findings seem to suggest that both contraction and relaxation of LVPW begin earlier than normal due to ventricular pre-excitation initiated at or near the LVPW. The IVS abnormality characteristic of the syndrome was a posterior protrusion, the onset of which preceded S1. Subsequent motion of IVS was either paradoxical or hypokinetic during ejection. These IVS abnormalities were quite similar to those observed in left bundle branch block. The abnormal LVPW motion was observed in all 20 Type A patients and 10 of the 32 Type B patients. The abnormal IVS motion was observed in 10 Type B patients. We feel that echocardiography could be used as an auxiliary noninvasive means of locating the pre-excitation site in patients with WPW.

IT IS WIDELY ACCEPTED that the QRS complex characteristic of the Wolff-Parkinson-White (WPW) syndrome is a fusion beat produced by an atrial impulse conducted via two (or more) different atrioventricular pathways, of which one is the normal specialized conducting system and the other an anomalous pathway. Such altered sequences of ventricular activation should be accompanied by abnormal sequences of ventricular contraction, which are expected to be reflected in echocardiographic patterns of ventricular motion. There have been a few studies reporting the echocardiographic features of the syndrome, but their sample sizes seem to be too small to draw a definite conclusion. The aim of the present report is to describe the specific features of the echocardiogram in the WPW syndrome and to correlate them with the electrocardiographic classification of the syndrome in a relatively large patient population.

Material and Methods

Fifty-two patients without any clinical evidence of cardiovascular diseases except for the electrocardiographic abnormalities characterized by a short P-R interval and a wide QRS complex with a definite delta wave were studied. In all of them Frank vectorcardiograms were recorded, which showed characteristic QRS deformities associated with a delta loop. There were 31 males and 21 females, who ranged in age from six to 53 years, the average being 26 years. On the basis of Rosenbaum's classification the electrocardiograms were classified as Type A in 20 patients and as Type B in 32. The control group consisted of 20 normal males, who ranged in age from 18 to 34 years, the average being 28 years.

The echocardiographic examinations were performed with a commercially available ultrasonoscope (Aloka SSD-90), using a nonfocused transducer of 10 or 13 mm in diameter (2.25 MHz). Echocardiograms were photographed from an oscilloscope screen with a Polaroid camera. With patients in the supine or left lateral position the transducer was placed in the third or fourth intercostal space at the left sternal border. The ultrasonic beam was directed so that the left ventricular posterior wall (LVPW) and the interventricular septum (IVS) were recorded simultaneously with both the anterior and posterior mitral valve leaflets or with the anterior leaflet and the chorda tendinea. Careful attention was paid to the direction of the beam since the echo pattern of IVS varies significantly with the beam direction even in normals. However, slight modification of the beam direction in a lateral or medial direction was permitted to cover as large an area of LVPW as possible. Lead II electrocardiograms and phonocardiograms were recorded simultaneously with echocardiograms.

Three patients were given ajmaline intravenously in order to block the anomalous atrioventricular bypass for comparative analysis of the echocardiographic patterns during normal atrioventricular conduction and WPW aberration. The transducer was held manually through this procedure so that the direction of the ultrasonic beam was kept as constant as possible.

Results

Echo Patterns of LVPW Motion

Abnormal echo patterns of LVPW motion (fig. 1) were seen in 30 of the 52 patients studied: all of the 20 Type A patients and ten of the 32 Type B patients (table 1). Following a slight posterior displacement of short duration due to atrial contraction, which was not recorded in some cases, the LVPW began to move in an anterior direction 40 to 140 msec (73 ± 4 msec, mean ± SEM) after the onset of the delta wave but 10 to 100 msec (52 ± 3 msec) before the first heart sound (S1). In contrast, the onset of the anterior LVPW motion consistently occurred after S1 in normals (fig. 2 and table 2). This early anterior motion was soon interrupted, resulting in a small step or hump formation. The LVPW resumed anterior motion immediately after S1 and reached the peak in late systole 340 to 460 msec (399 ± 6 msec) after the onset of the delta wave but 10 to 60 msec (37 ± 2 msec) before the second heart sound (S2). In normal controls the peak either occurred simultaneously

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with, or following $S_2$ (fig. 2 and table 2). In 17 of the patients who showed LVPW motion abnormalities, a second lower peak appeared shortly after $S_2$, thus the anterior heave showing a bifid apex. In the remaining 13 cases, a single peak was observed.

**Echo Patterns of IVS Motion**

The onset of posterior motion of the IVS consistently followed $S_1$ in normals (fig. 2 and table 2). Ten of our patients, however, all of whom showed Type B electrocardiograms (table 1), exhibited abnormal echo patterns of IVS motion (fig. 3). The IVS began to move posteriorly 10 to 80 msec (50 ± 9 msec) after the onset of the delta wave but 60 to 110 msec (83 ± 5 msec) before $S_2$ and soon reversed the direction of motion, forming a rather sharp posterior protrusion nearly coincident with $S_1$. In three patients it still continued to shift slowly in an anterior direction until $S_2$, showing the pattern of paradoxical motion. However, in the remaining seven cases it moved in a posterior direction and reached the first nadir in late systole. In all ten patients the IVS moved posteriorly after $S_2$ and formed a deeper nadir in early diastole.

**Changes in Echo Patterns after Ajmaline**

Normalization of atrioventricular conduction was attempted with intravenous ajmaline in three patients. In two Type A patients who showed LVPW abnormalities, the pre-ejection step or hump disappeared as soon as the delta wave was abolished (fig. 4). At the same time the peak of the anterior displacement shifted from late systole to early diastole and became unimodal. In one Type B patient with abnormal IVS motion the pre-ejection posterior protrusion disappeared simultaneously with abolition of the delta wave, while the pattern during ejection and early diastole remained nearly unchanged (fig. 5).

**Discussion**

**Abnormal Echo Patterns of LVPW Motion**

Following a slight posterior displacement due to atrial contraction, which is also noted in normals, the LVPW initiated anterior motion before $S_1$ in many patients with the
WPW syndrome. This finding contrasts strongly with the fact that the anterior motion of LVPW invariably begins after isovolumetric contraction in normals, and suggests the presence of abnormally early contraction of LVPW probably activated by the pre-excitation impulse. The subsequent small dip or step formation is considered to be due to the change in the shape of the left ventricle during isovolumetric contraction. The occurrence of the peak anterior displacement in late systole and also the succeeding notching seem to suggest early relaxation and backward bulging of LVPW in the presence of still contracting myocardium of the rest of the left ventricle. DeMaria and colleagues echo-cardiographic pattern after ajmaline-induced normalization of atrioventricular conduction in a patient with Type A WPW syndrome. The beginning and the peak of anterior motion of LVPW are indicated by arrows. The former shifted from before to after S1, and the latter from before to after S2.

**TABLE 2.** Time Intervals from the Onsets of the QRS Complex and Heart Sounds to Echocardiographic Movements

<table>
<thead>
<tr>
<th>WPW</th>
<th>Time intervals (msec)</th>
<th>Normal (N = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Q-LVPWo</td>
<td>Q-LVPWp</td>
</tr>
<tr>
<td>range</td>
<td>40 → 140</td>
<td>340 → 460</td>
</tr>
<tr>
<td>mean ± SEM</td>
<td>73 ± 4*</td>
<td>399 ± 6</td>
</tr>
</tbody>
</table>

*P < 0.001.
†A negative sign was given to the time interval when the echocardiographic event preceded the respective heart sound, and a positive one in the reversed condition.

In the WPW group measurements of LVPW were obtained in 30 patients with LVPW abnormalities and measurements of IVS in 10 with IVS abnormalities.

Abbreviations: Q = the onset of the QRS complex; S1 and S2 = the first and second heart sounds; LVPWo and LVPWp = the onset and peak of anterior motion of LVPW; IVSo = the onset of posterior motion of IVS.

**FIGURE 4.** Changes in echocardiographic pattern after ajmaline-induced normalization of atrioventricular conduction in a patient with Type A WPW syndrome. The beginning and the peak of anterior motion of LVPW are indicated by arrows. The former shifted from before to after S1, and the latter from before to after S2.

**FIGURE 5.** Changes in echocardiographic pattern after ajmaline-induced normalization of atrioventricular conduction in a patient with Type B WPW syndrome. The pre-ejection protrusion indicated by the arrow in the upper panel is not noted in the lower one, in which the hypokinetic motion of IVS might be due to negative inotropism of ajmaline.
cardiographically demonstrated early onset and premature occurrence of the peak of anterior LVPW motion in patients with the WPW syndrome by using electrocardiograms as time references. In the present study, heart sounds were used as time references. This procedure enables the examiner to identify early contraction and relaxation of LVPW simply by knowing whether the echocardiographic movements precede heart sounds or not. Bandiera and Antognetti6 roentgenkymographically demonstrated a premature onset of contraction of a localized ventricular area in patients with the WPW syndrome. Prinzmetal and colleagues7 studied ventricular motion by means of high-speed cinematographic records and showed an early onset of contraction and a late systolic outward protrusion of the experimentally pre-excited ventricular myocardium of dogs. Their observations provide supportive evidence for our explanation of the mechanisms of echocardiographic LVPW abnormalities. The present study also showed that abrupt normalization of LVPW motion occurred simultaneously with ajmaline-induced abolition of the delta wave. This finding seems to further support the above described explanation.

Abnormal Echo Patterns of IVS Motion

In one-fifth of the patients the IVS began to move posteriorly before S1 and formed a sharp posterior protrusion during the pre-ejection phase. These findings were consistent with those made by DeMaria and colleagues1 in one patient with Type B WPW. The subsequent IVS motion tended to be either hypokinetic or paradoxical during ejection. These observations closely resemble those reported on left bundle branch block10-12 and right ventricular pacing.11 The echocardiographic similarity among these three conditions seems to afford an important clue to the genesis of abnormal septal motion in the WPW syndrome. There have been two possible explanations proposed for the pre-ejection posterior motion in left bundle branch block: 1) earlier contraction of IVS which is unopposed by forces generated by contraction of the left ventricular free wall,10, 11 and 2) depolarization of IVS from right to left.12 We consider that similar mechanisms, especially the latter, may be responsible in the WPW syndrome. The hypokinetic or paradoxical motion during ejection might be explained by delayed left ventricular contraction.16

Correlation between Abnormal Echo Patterns and the Electrocardiographic Classification

Abnormal LVPW motion was noted in all of the Type A patients studied. This observation is compatible with the widely accepted concept that, in Type A, pre-excitation occurs at the posterolateral base of the left ventricle.13,14 However, abnormal motion of LVPW was also noted in one-third of the Type B patients. A recent report15 suggests the possibility that Type B electrocardiograms may result from pre-excitation at the right ventricular free wall or IVS. Two possible explanations may be offered for the combination of a Type B electrocardiogram and echocardiographic LVPW abnormalities: 1) pre-excitation occurs near the LVPW at the right ventricular posterior wall or the posterior portion of the IVS, thus inducing earlier activation of the LVPW than the rest of the left ventricle; and 2) pre-excitation of mild degree starts at LVPW, with the resultant QRS complex being predominantly negative in the right precordial leads. The latter possibility seems unlikely because echocardiographic abnormalities are presumed to be unrecognizably slight.

Abnormal IVS motion was observed in one-third of typical Type B patients, in whom pre-excitation is generally thought to occur in the right ventricle. It was reported that only three of ten patients with right ventricular pacing showed abnormal IVS motion.11 This indicates that patients with pre-excitation in the right ventricle do not necessarily have IVS abnormalities. An alternative explanation for the absence of abnormal IVS motion in the Type B WPW syndrome is that pre-excitation occurs in the left ventricle in these patients.

Abnormal motion in both the IVS and the LVPW were observed in a few Type B patients. These cases may be explained by pre-excitation at the right-sided IVS near the LVPW or simultaneous occurrence of pre-excitation at more than one site.

No echocardiographic abnormalities were noted in either IVS or LVPW in 14 Type B patients, who might have pre-excitation somewhere in the right ventricle far from both the IVS and LVPW, or in the left ventricle remote from the LVPW.

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