Alterations in Ventricular Contraction Pattern in the Wolff-Parkinson-White Syndrome

Detection by Echocardiography

ANTHONY N. DEMARIA, M.D., ZAKAUDDIN VERA, M.D., ALEXANDER NEUMANN, AND DEAN T. MASON, M.D.

SUMMARY The effects of abnormal ventricular activation upon the contractile pattern of the ventricles in patients with the Wolff-Parkinson-White syndrome (WPW) remain uncertain. Therefore we compared the motion of the anterior right ventricular wall (RV), the interventricular septum (IVS), and left ventricular posterior wall (LVPW) on echogram in nine patients with WPW and one patient with a coronary sinus pacemaker (CSP) to 20 normal subjects. Normal subjects manifested posterior RV motion which began and reached maximal excursion at 175 and 366 msec (group mean), respectively, after the onset of the QRS complex; posterior movement of the IVS which started and peaked at 90 and 350 msec, respectively; and anterior contraction of the LVPW which began and peaked at 159 and 406 msec, respectively. Five of seven patients with Type A WPW demonstrated a localized area of premature contraction of the LVPW occurring during the initial 100 msec interval following the onset of the QRS complex which was accompanied by paradoxic anterior motion of the IVS. Thereby in Type A patients initial and maximal posterior motion of the IVS occurred later, 230
\( P < 0.001 \) and 400 \( P < 0.05 \) msec, and anterior motion of the LVPW occurred earlier, 75 \( P < 0.001 \) and 367 \( P < 0.05 \) msec as compared to normal. The amplitude and duration of early contraction could be related to the prominence of the delta wave during atrial pacing. Similar premature contraction was also observed in the patient with CSP during paced beats. One Type B WPW patient exhibited abnormal IVS motion while the additional patient manifested premature LVPW contraction similar to that seen in Type A patients. The contractile pattern of the right ventricular anterior wall was recorded in five of seven Type A Wolff-Parkinson-White patients and manifested prolongation of the interval from the onset of the QRS complex to the initial posterior movement (group mean 234 msec, \( P < 0.05 \)) as compared to normal. Thus echocardiography can be used to confirm the diagnosis and to improve understanding of the pathophysiology of the Wolff-Parkinson-White syndrome.

THE WOLFF-PARKINSON-WHITE SYNDROME (WPW) is a disorder of cardiac conduction which causes premature electrical activation of a segment of the ventricular myocardium. Activation of the ventricular myocardium, therefore, proceeds as a fusion of the ectopic impulse conducted via an accessory tract with the normal propagation of depolarization conducted via the atrioventricular node, the interaction of which may vary depending on the predominant pathway. The effects of premature ventricular activation upon the contractile pattern of the ventricles have long intrigued cardiologists, both in reference to the alterations that might be induced in cardiac performance, and with regard to how such knowledge might contribute to the understanding of the underlying mechanism of the disorder. Thus, attempts have been made to assess the influence of pre-excitation upon the temporal sequence of ventricular contraction by means of high-speed motion pictures of the epicardium, \(^6\) roentgenkymography \(^4\) and electrokymography, \(^4\) intraventricular pressure recordings, \(^5\) \(^6\) and phonocardiography with external venous and arterial pulse recordings. \(^5\) \(^6\) \(^8\) \(^10\) However, these studies have been inconclusive and have yielded conflicting data.

Echocardiography is a noninvasive technique which is capable of providing high-fidelity recordings of the motion of the interventricular septum and left ventricular posterior wall, and in many patients, of the anterior right ventricular wall as well. Ultrasound recordings have been useful in detecting abnormalities of ventricular contraction in the presence of left bundle branch block \(^11\) \(^14\) right ventricular volume overload states, \(^15\) \(^16\) cardiomyopathy, \(^17\) \(^18\) and coronary artery disease. \(^19\) \(^20\) The present study was undertaken to evaluate the ability of echocardiography to detect, localize, and quantify abnormalities in the contractile pattern of the ventricles introduced by premature ventricular excitation in patients with the Wolff-Parkinson-White syndrome.

Methods and Materials

The patient population consisted of nine individuals, seven males and two females, ranging in age from 18 to 28 years (mean, 24) with electrocardiographic and vectorcardiographic documentation of the Wolff-Parkinson-White syndrome. \(^22\) Evidence of organic heart disease other than ventricular pre-excitation was absent on physical examination, chest roentgenogram, and echocardiogram in all patients. In addition, echocagrams were analyzed in a 59-year-old male in whom a coronary sinus pacemaker was demonstrated, by analysis of scalar electrocardiograms and vectorcardiograms, to result in initial ventricular posterior wall stimulation. Echographic data from these ten patients were compared to those of 20 normal subjects, 12 male and eight female, ranging in age from 21 to 28 years (mean, 25).

Scalar electrocardiograms (ECG) and vectorcardiograms (VCG) were carried out in all patients with ventricular pre-excitation at rest, and were also obtained with His bundle electrograms during atrial pacing in seven of these patients.

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From the Section of Cardiovascular Medicine, Departments of Internal Medicine and Physiology, University of California at Davis, School of Medicine and Sacramento Medical Center, Davis and Sacramento, California. Supported in part by a Research Grant from the Central Valley California Heart Association and Research Program Project Grant HL 14780 from the National Heart and Lung Institute, NIH, Bethesda, Maryland.

Address for reprints: Anthony N. DeMaria, M.D., Assistant Professor of Medicine, Section of Cardiovascular Medicine, University of California, School of Medicine, Davis, California 95616.

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Vectorcardiography and intracardiac electrography were recorded on a multichannel photographic oscilloscopic recorder. His bundle electrography, by the method of Scherlag and coworkers, and atrial stimulation were performed utilizing a bipolar electrode catheter. The diagnosis of Wolff-Parkinson-White syndrome was established by a short P-R interval (less than 120 msec) and prolongation of the QRS complex (greater than 80 msec) due to distortion of the initial phase of this deflection by a delta wave on ECG, and by demonstration of a reduced conduction velocity during early ventricular activation on VCG. Findings on His bundle electrogram indicative of accelerated ventricular activation consisted of a decrease in the interval from the His bundle potential to the onset of the QRS complex of the surface electrocardiogram. Patients with WPW were classified as Type A when the initial vector resulting from ventricular depolarization was directed anteriorly and Type B when this electrical potential was directed posteriorly.

Echocardiography was performed in the standard fashion in the supine or 30° right anterior oblique position using a commercially available echograph interfaced to either an Electronics for Medicine photographic or Honeywell fiberoptic recorder. Ultrasound recordings were obtained in all WPW patients and normal subjects at rest, and echograms were also obtained in five of the WPW patients during atrial pacing. During echographic examination care was taken to obtain simultaneous recordings of the interventricular septum and left ventricular posterior wall in a section of the left ventricle below the maximal deflection of the mitral leaflets, to scan as large an area of the posterior wall as possible, to obtain signals from the anterior right ventricular wall, and to have all echograms appear in the unretracted condition.

Echocardiograms obtained in the above fashion were then systematically analyzed for the sequence of left ventricular contraction (fig. 1). Thus, an echogram in which both the interventricular septum (IVS) and left ventricular posterior wall (LVPW) were simultaneously recorded in an area below the maximal deflection of the mitral leaflets was selected and the position of these ventricular walls at the onset of the QRS complex was determined. Serial movements of the endocardial septal and posterior wall surfaces were then analyzed at 50 msec intervals for 600 msec in three consecutive cardiac cycles. Motion of either ventricular wall away from the opposite wall with reference to its position at the onset of the QRS complex was designated as a negative amplitude while motion toward the opposite wall was designated as a positive amplitude (fig. 1). Mean values were then obtained for the intervals from the onset of the QRS complex to both the initial and maximal amplitudes of positive motion for the IVS and LVPW and these values in the pre-excitation patients and the normal subjects were compared. In addition, the period from the onset of the QRS to the onset of the initial and maximal amplitude of motion of the anterior right ventricular (RV) wall was compared in the WPW patients and normals. Although the exact onset of RV anterior wall contraction was often difficult to discern due to the amorphous nature of the echoes obtained, the initial definite posterior motion was taken to represent the onset of systolic contraction.

All results were evaluated by statistical analysis utilizing Student's t-test.

Results

Figure 2 demonstrates the values obtained in the normal subjects and Type A WPW patients relating to the time intervals from the onset of QRS to onset and peak contraction of the LVPW, IVS, and RV anterior wall.

Normal Subjects

Following the onset of the QRS complex, the left ventricular posterior wall remained stationary or exhibited a posterior movement for a period of 100 to 200 msec (159 ± 9 [SEM] msec) at which time the initial anterior, or positive, motion of this structure was manifested (figs. 1 and 2). Anterior movement continued until 300 to 450 msec following the onset of the QRS deflection (406 ± 10 msec) at which time maximal contraction amplitude was reached and a gradual posterior movement followed as relaxation of the posterior wall occurred. The interventricular septum also exhibited stationary or a negative anterior motion in the interval immediately following the QRS complex, but this period

*Model DR 8, Electronics for Medicine, White Plains, New York
†Cordis Corporation, Miami, Florida
‡Ekoline 20A, Smith-Kline Instruments, Inc., Palo Alto, California
§Model 1856, Honeywell Instruments, Denver, Colorado

![Figure 1](http://circ.ahajournals.org/)

**Figure 1** Representative echogram of a normal subject (panel A) showing the appropriate posterior motion of the interventricular septum (IVS) and anterior motion of the left ventricular posterior wall (LVPW). Panel B is a schematic illustration taken from panel A depicting the method of quantitative analysis of the inward contractile pattern of the IVS and LVPW in the normal subject. The solid lines designate 50 msec intervals.
was of shorter duration, varying from 50 to 200 msec: 90 ± 11 msec. Subsequently the IVS manifested a sustained backward movement approaching the posterior wall which demonstrated maximal excursion at 300 to 450 msec: 350 ± 7 msec. Thus positive interventricular septal movement began earlier and reached maximal amplitude sooner than the left ventricular posterior wall. In addition, a notching of the interventricular septum in the latter part of systole, attributed by MacDonald and coworkers to anterior movement of the entire heart, was noted in all normal subjects.

Motion of the anterior wall of the right ventricle was recorded in 14 of the 20 normal subjects (figs. 2 and 3A). The anterior right ventricular wall remained stationary for 120 to 200 msec following the onset of the QRS complex (175 ± 7 msec) at which time this structure manifested gradual posterior motion for 310 to 450 msec, (366 ± 11 msec) whereupon it reached maximal excursion.

Type A Wolff-Parkinson-White Syndrome

Seven patients manifested electrocardiographic and vectorcardiographic evidence of Type A WPW. The most striking echographic feature noted in these patients was the presence of a 2 to 3 mm discrete early systolic anterior movement of the posterior left ventricular wall during the initial 100 msec following the onset of the QRS complex of the electrocardiogram, which was noted in five patients at rest or during atrial pacing (fig. 4). This posterior wall motion characteristically occurred coincident with inscription of the delta wave of the ECG and was interpreted to represent a prematurely contracting segment of the left ventricle secondary to early activation. One of the two remaining Type A patients manifested a delta wave during ultrasound recording only during atrial premature beats, and although early contraction appeared to be present, it was difficult to be certain of this observation because of the superimposition of ventricular contraction upon the relaxation of the posterior wall from the previous sinus beat. Thus, only one Type A patient revealed no evidence of an early contractile “bump” of the posterior wall. Thus the initial anterior or positive movement of the posterior wall for the group of Type A WPW patients occurred from 50 to 100 msec following the onset of the QRS complex, (75 ± 15 msec) which was significantly earlier (P < 0.001) than that of the normals (fig. 2).

The effect of premature contraction of the left ventricular posterior wall upon the contractile pattern of the interventricular septum was to induce a prolonged anterior or negative motion of the left endocardial septal surface during the initial portion of systole (fig. 4). Thus the initial posterior or positive motion of the IVS occurred at 150 to 350 msec following the onset of the QRS complex, (230 ± 25 msec) which was 140 msec later (P < 0.001) for the group than that recorded in normals (fig. 2). In addition, peak anterior motion of the left ventricular posterior wall occurred earlier (350 to 400 msec, mean 367 ± 7; P < 0.05) and maximal excursion of the IVS occurred later (300 to 500

![Figure 2](image-url)

**Figure 2** Comparative values obtained in the normals and Type A WPW patients of the interval from the onset of the Q wave of the ECG to the onset of contraction of the LVPW, IVS, and right ventricular anterior wall (RV) (top panel); and of the interval from the onset of the Q wave of the ECG to the peak of contraction of the LVPW, IVS, and RV (bottom panel).

![Figure 3](image-url)

**Figure 3** Representative echogram of a normal subject (panel A) demonstrating systolic posterior motion (arrow) of the right ventricular endocardial wall (RV). Panel B shows the delayed onset of systolic downward movement of the right ventricular wall (arrow) during contraction in a representative patient with Type A WPW. The time from the onset of the QRS to beginning of right ventricular motion is indicated in both panels. In this and subsequent figures the solid vertical lines depict one second intervals.
msec, mean 400 ± 31; P < 0.05) for the group of Type A pre-excitation patients than the group of normals (fig. 2).

Echocardiography was performed in five Type A WPW patients during atrial pacing at rapid rates (figs. 5, 6, and 7). Four of these five patients manifested definite prematurely contracting areas of the posterior wall at rest. Right atrial pacing produced an increase in the delta wave of the electrocardiogram in all five of these patients which was accompanied by an augmentation in both the amplitude and duration of the precontractile bump of the posterior wall in four of these cases. Thus right atrial pacing resulted in a greater proportion of ventricular activation via the accessory pathway which produced a more prominent area of prematurely contractile posterior wall.

It was possible to obtain a record of the contractile pattern of the right ventricular anterior wall by echogram in five of the seven Type A patients (fig. 3B). The initial posterior motion of this structure was delayed as compared to normal, occurring from 200 to 285 msec following the onset of the QRS complex (234 ± 15; P < 0.05) (fig. 2). In addition, RV wall movement reached maximal amplitude at 375 to 420 msec, (395 ± 8 msec) which was also not similar (P < 0.05) to that of the normal subjects (fig. 2).

Type B Wolff-Parkinson-White Syndrome

Two patients exhibited electrocardiographic and vectorcardiographic findings consistent with Type B WPW. One patient manifested a normal contractile pattern of the left ventricular posterior wall but was noted to have a peculiar pattern of interventricular septal contraction (fig. 8). Thus, IVS motion in this patient was found to consist of two discrete posterior movements which were followed by two similar anterior thrusts in an area of the left ventricle substantially below the mitral leaflets in the direction of the cardiac apex. Interventricular septal motion similar to this pattern was not noted in this section of the left ventricle in any of the normal subjects and therefore septal contraction was considered abnormal in this patient.

FIGURE 4 Representative echocardiogram in a patient with Type A WPW during normal sinus rhythm. The arrow indicates typical premature contraction of the LVPW. The IVS shows initial paradoxic anterior movement and delayed posterior motion during systole.

FIGURE 5 Representative echocardiogram during atrial pacing in the Type A WPW patient shown in figure 4. Arrows A, B, and C depict progressive increase in amplitude and duration of the prematurely contracting LVPW as the aberrancy of the QRS increases with greater atrial pacing rate. In addition, the IVS shows initial paradoxic anterior motion and delayed posterior movement during contraction.
FIGURE 6 Echocardiogram obtained from a Type A WPW patient during atrial pacing at varying heart rates, as indicated by the measured cycle lengths. Arrow A demonstrates prominent premature contraction of the LVPW at the most rapid rate, while arrow B shows decreased amplitude and arrow C exhibits nearly complete disappearance of the early bump as the paced cycle length widens. The typical abnormal motion of the IVS is also observed. S = electrical pacing artifact.

The second Type B WPW patient was interesting in that he manifested a small anterior contraction of the posterior wall just following the inscription of the delta wave of the ECG similar to that noted in the Type A patients. This posterior wall bump was only 1 mm in amplitude, and although the initial posterior motion of the interventricular septum did not occur until 200 msec after QRS onset in this patient, a similar delay in IVS contraction was noted in one normal subject.

The motion of the anterior wall of the right ventricle was able to be recorded only in the initially described Type B patient and was similar to that noted in the normal subjects. Atrial pacing was not available in either Type B patient.

Coronary Sinus Pacing

Echocardiography was performed in one patient with sick sinus syndrome without additional cardiac abnormalities with a permanent demand transvenous pacemaker. The pacemaker electrode was positioned in the coronary sinus so as to result in initial stimulation of the left ventricular pos-

FIGURE 7 Echocardiogram and simultaneously recorded precordial lead V1 from another Type A WPW patient obtained during atrial pacing. While no premature LVPW contraction is noted prior to atrial pacing, a prominent early bump corresponding to the delta wave of the QRS is clearly evident (arrow) during each of the aberrant QRS complexes induced by rapid atrial pacing. The IVS was noted to reach delayed peak maximal downward excursion following the T wave.
terior wall. The cardiac rhythm in this patient consisted of beats which originated from this pacemaker as well as impulses which were conducted through the atrioventricular node. Ultrasound examination in this patient demonstrated premature contraction of an area of the LVPW occurring with each pacemaker initiated beat, a motion similar to that observed in the Type A WPW patients (figs. 4–7). In contrast, ventricular activation which resulted from an impulse conducted via the A-V node did not produce evidence of early posterior wall movement on echogram (fig. 9).

Echocardiograms were also evaluated for abnormalities of the mitral, aortic, and tricuspid valves. No abnormalities were observed in any WPW patient in this study. Thus premature contraction does not appear to result in the development of sufficient ventricular pressure to alter valvular position.

Discussion

The electrocardiographic constellation of a short P-R interval with slurring and prolongation of the initial phase of the QRS complex occurring in patients prone to episodes of supraventricular tachycardia was initially described by Wolff, Parkinson, and White in 1930.21 Although this syndrome has attracted considerable attention since that time,
the exact anatomic and electrophysiologic correlates of the electrocardiographic findings remain uncertain. Traditionally, patients with ventricular pre-excitation have been divided into two groups: Type A, in which early activation is thought to occur in the area of the left ventricular posterior wall; and Type B, in which the lateral right ventricular myocardium is believed to undergo initial excitation. The electrocardiographic and vectorcardiographic manifestations of this disordered ventricular activation consist of an initial QRS vector which is directed anteriorly in Type A patients, and oriented posteriorly in Type B patients. Currently it is believed that the accelerated ventricular activation is the result of accessory atrioventricular conduction pathways which circumvent the normal conduction delay encountered in the atrioventricular node. While the existence of such accessory tracts from atria to ventricles across the lateral atrioventricular sulcus has been established in several cases, the possibility remains that pre-excitation may also occur via atrioventricular pathways in the interventricular septum. Although recent studies in which epicardial mapping has been performed in WPW patients have generally reported findings consistent with these concepts, the wide variation in the anatomical location of initial ventricular activation and the resultant diversity of electrocardiographic patterns have suggested intermediate forms of the conventional Type A and B classification. Measurements in another modality such as ultrasound which reflect the sequence of ventricular activation may provide the key in distinguishing these variations in the WPW syndrome.

Considerable attention has been directed toward the evaluation of alterations of cardiac contraction induced by ventricular pre-excitation but the results of these studies are inconclusive. The conflicting data is most likely due to the limitations inherent in the techniques used. Echocardiography, in contrast, although limited by the restricted expanse of left ventricle which may be examined and the width of the ultrasound beam, records the movement of the interventricular septum and posterior left ventricular wall at a frequency of 1000 times per second and thus may detect even a subtle area of ventricular premature contraction.

The pattern of contraction of the left ventricular posterior wall on echogram was initially described by Kraunz and Kennedy in 1970. Subsequently, MacDonald and associates performed a detailed analysis of the movements of both the interventricular septum and left ventricular posterior wall by ultrasound. These latter investigators assessed wall motion in an area of the left ventricle below the maximal excursion of the mitral leaflets and noted that the motion of the posterior wall consisted of an anterior excursion shortly following the onset of systole which resembled an inverted ventricular volume curve, while the interventricular septum demonstrated posterior motion following the onset of the QRS complex which was interrupted by a notch during the terminal phase of systole due to movement of the entire heart anteriorly in the thorax. Recently Hagan and associates have demonstrated that the contractile pattern of the IVS may be heterogeneous in normal subjects, varying from no motion or even paradoxical motion in the superior segments, to the backward motion approximating the posterior wall in the IVS segment below the mitral leaflets. Results of the analysis of left ventricular motion in our normal subjects was in agreement with these previously reported studies (fig. 1). Moreover, previous studies in our laboratories have shown no abnormality of ventricular contractile pattern in patients with right bundle branch block or with left axis deviation. This same investigation demonstrated that patients with left bundle branch block had normal motion of the LVVP but a characteristic abnormality of IVS movement consisting of an early peaking posterior motion followed by a sustained paradoxical anterior movement, unlike that observed here in Type A WPW patients. Finally, in order to insure uniformity of septal motion, all measurements in the present study were obtained from a section of the left ventricle below the mitral leaflets.

Previous investigators have alluded to the difficulty entailed in utilizing the electrocardiogram to time mechanical events in patients with the Wolff-Parkinson-White syndrome. Thus, the presence of a short delta wave in the ECG of a WPW patient may account for some alterations in the temporal sequence of ventricular contraction when compared to a patient with a normal ECG. In this regard, premature posterior motion of the LVVP as seen in WPW patients would be even more premature if encountered in a patient with normal P-R interval and QRS complex, while delayed contraction of the anterior RV wall in Type A WPW patients would be normal in relation to a normally inscribed ECG. However, since the echogram is traditionally timed with reference to the electrocardiogram, and since the purpose of this study was to correlate electrical and mechanical events, it was elected to measure all echographic movements from the onset of the QRS complex.

The most striking finding in our study was the presence of a prematurely contracting segment of the left ventricular posterior wall which was manifested on echogram as a small, distinct bump of the endocardial surface of this structure in early systole in the Type A Wolff-Parkinson-White patients (figs. 4–7). Definite prematurely contracting segments were noted in five of the seven patients with Type A ventricular pre-excitation, and a movement suggestive of early systolic anterior bulge of the posterior wall was noted in another of these patients when a delta wave appeared during atrial premature beats. Thus six of seven Type A WPW patients manifested evidence of premature contraction of the posterior wall. The peculiar contractile pattern of the LVVP observed in these patients with Type A early ventricular activation was interpreted to represent the contraction of the localized prematurely activated area of myocardium, manifested as a bump, which was superimposed upon the sustained anterior movement of the entire posterior wall following complete activation of all areas of this structure. It should be pointed out that the prematurely contracting segment of the left ventricle was localized in a small area in most patients and could be recorded only with a single transducer position. Therefore the early systolic bump of the posterior wall echogram could be easily overlooked unless the entire surface of the LVVP capable of being recorded by echogram were examined by ultrasound. Although maximal premature contraction seemed to occur in an area of the posterior wall just below the mitral leaflets along the medial or septal aspect, in several patients early systolic motion could be observed more laterally as well.

In addition, echocardiography was performed during
atrial pacing in five patients with Type A WPW, four of whom manifested premature contraction of the posterior wall at rest. In each instance atrial pacing resulted in an increased proportion of the ventricular myocardium which was activated via the accessory pathway as demonstrated by an exaggeration of the delta wave and increased QRS complex duration on the electrocardiogram (figs. 5, 6, 7). Although it is possible that the increased QRS duration could be related to aberrant conduction of an impulse transmitted via the A-V node, the predominant involvement of the initial portion of the QRS complex, combined with the maintenance of the basic QRS configuration, rendered it more likely that the electrocardiographic distortion provoked by atrial pacing represented increased conduction via the accessory pathway. Atrial pacing also resulted in an increase in both the amplitude and duration of anterior movement of the prematurely contracting area of myocardium. Thus atrial pacing provided further evidence that the early anterior bump of the posterior wall echogram represented a prematurely contracting segment of the left ventricle in response to accelerated ventricular activation. Further, the response of the asynchronously moving ventricular area to atrial stimulation indicated that the proportion of myocardium excited abnormally via accessory pathways is proportional to the prominence of the delta wave of the electrocardiogram.

The response of the contractile pattern of the posterior wall on echogram to coronary sinus stimulation in the patient in whom this examination was performed (fig. 9) provided additional confirmation that the early systolic bump represented premature ventricular contraction secondary to premature activation. Thus, ventricular stimulation via the pacemaker in the coronary sinus would be expected to result in early contraction of the left ventricular posterior wall similar to Type A WPW. Echocardiography in the patient with the coronary sinus pacemaker revealed an early posterior bump during coronary sinus paced beats identical to that seen in WPW patients, while a normal contractile pattern was observed during normal ventricular conduction as indicated by the ECG. Thus it appears highly likely that the early posterior wall contractions noted on echogram in patients with Type A WPW represents a prematurely contracting ventricular segment.

The effect of ventricular pre-excitation upon the contractile pattern of the left ventricle was to reduce the interval between the onset of the QRS complex and the initial anterior motion of the posterior wall in Type A WPW patients as compared to normals (figs. 1–3). This early posterior wall movement occurred unopposed by septal contraction and thereby resulted in a paradox anterior motion of the interventricular septum during early systole, which was manifested as a prolongation of the period between the onset of the QRS complex and the initial posterior motion of the IVS. Although it is possible that the temporal proximity of atrial contraction to the onset of the QRS complex may have resulted in the anterior motion of the interventricular septum in early systole observed in patients with accelerated ventricular conduction, it is unlikely that ventricular filling secondary to atrial contraction alone could account for such a sustained paradoxic movement. The net effect of the premature ventricular activation was therefore to reduce the interval between the onset of the QRS complex and initial and maximal posterior wall contraction, while increasing the period to initial and maximal IVS contraction in the group of Type A WPW patients compared to normals. Thus, it appears that Type A WPW should be added to the growing list of causes of abnormal septal motion.

In contrast to the patients with Type A WPW, no characteristic manifestation of ventricular pre-excitation could be detected on echogram in the contractile pattern of the ventricles in the two patients with Type B WPW. However, some disorder of left ventricular wall motion was observed in both patients. One patient was found to have a peculiar pattern of alternating posterior and anterior motion of the interventricular septum by ultrasound (fig. 8). Motion of the IVS similar to this patient was not observed in this section of the left ventricle in any of our normal control subjects although it has been found in other sections in normal subjects. The early systolic posterior peak of the IVS observed in this patient resembles that motion described in patients with left bundle branch block and is consistent with the activation of the right ventricle prior to that of the left ventricle, reported in patients with Type B WPW. Although the explanation of this abnormal septal motion remains uncertain, it is possible that the accessory atrioventricular pathway in this patient consisted of fibers which were located in the interventricular septum, as in cases described by James and Mahaim. The second patient with Type B WPW was particularly interesting in that he exhibited an early contraction motion in the posterior wall on echogram identical to that recorded in the Type A WPW patients. Although the etiology of this abnormal movement of the posterior wall has not been established, it is possible that two accessory pathways of atrioventricular activation were present in this patient; double accessory pathways have been described by Boineau, Coumel, and Ramachandran. Our observations upon the contractile pattern in Type B WPW patients are limited in number and do not enable any definite conclusions at this time. However, it does appear that the evaluation of ventricular contraction by echocardiography warrants further investigation in Type B WPW patients.

Evaluation of the contractile pattern of the anterior wall of the right ventricle by ultrasound is more difficult than assessment of the motion of the left ventricular endocardial surfaces. Thus the anterior right ventricular wall is frequently represented as a cloud of echoes upon which contraction and relaxation are superimposed. Nevertheless an effort was made to determine whether abnormalities of right ventricular movement could be detected in patients with either Type A or Type B WPW (fig. 3) by utilizing the first definite posterior motion of the right ventricular endocardium to represent the onset of contraction. The finding of delayed right ventricular anterior wall motion in patients with Type A WPW is consistent with the concept of premature activation of the left ventricle in these patients. Accordingly the RV anterior wall may actually be activated late, or may only appear so when considered in relation to the delta wave of the ECG. Right ventricular wall motion was able to be recorded in one Type B WPW patient and was found to be normal. However, it is again recognized that an identical interval from the onset of the QRS complex to
RV contraction in WPW patients may actually represent early wall motion, since RV anterior endocardial contraction would be expected to be delayed in relation to the delta wave if depolarization occurred in a normal sequence. Thus, although measurements of right ventricular contraction must be viewed with caution, it appears likely that abnormalities of right ventricular contractile pattern also result from ventricular pre-excitation.

In summary, echocardiography has documented prematurely contracting areas of ventricular myocardium in the posterior wall in at least five of seven patients with Type A WPW and one patient with Type B WPW. Unopposed early systolic contraction of the posterior wall resulted in abnormal anterior motion of the interventricular septum in early systole in Type A WPW. One patient with Type B WPW also exhibited abnormal IVS motion. However, it should be recognized that definite precontracting areas of the LVVPW were recorded in only four of the seven patients with Type A WPW at rest, and that premature excitation may be only intermittently present in patients with the Wolff-Parkinson-White syndrome. While echocardiography may be of limited value in some individuals with WPW, the technique offers the promise of aiding in the diagnosis and improving the understanding of the pathophysiology of the Wolff-Parkinson-White syndrome.

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References

33. Willens HJ, Lanse MJ, Van Dam RT, van Capelle FL, Meijne NG, Mellink HM, Durrer D: Epicardial mapping and surgical treatment in Wolff-Parkinson-White Syndrome Type A. Am Heart J 88:69, 1974
38. Ramachandran S: Wolff-Parkinson-White Syndrome, Conversion of Type A to Type B electrocardiographic changes. Circulation 45:529, 1972
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A N DeMaria, Z Vera, A Neumann and D T Mason

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