Echocardiography in Wolff-Parkinson-White Syndrome

MICHAEL S. CHANDRA, M.D., RICHARD E. KERBER, M.D., DONALD D. BROWN, M.D., AND DAVID C. FUNK, M.D.

SUMMARY Twenty-six patients with Wolff-Parkinson-White (WPW) syndrome were studied by echocardiography. They were classified into the following WPW types: anterior right ventricular pre-excitation (Type I) — six patients; posterior right ventricular pre-excitation (Type II) — six patients; posterior left ventricular pre-excitation (Type III) — fourteen patients. Twenty-three patients were in WPW at the time of study. Four patients with Type I WPW had normal systolic motion of the interventricular septum: three paradoxical and one flat. Patients with Type II and Type III WPW had no septal motion abnormalities related to pre-excitation. Three patients had intermittent WPW, Type III; in all three only minor changes in normal septal motion were apparent on WPW beats. Associated cardiac abnormalities were evident in six patients: two mitral prolapse (one Type II WPW and one Type III); one idiopathic hypertrophic subaortic stenosis (Type III); one congestive cardiomyopathy (Type III); one hypertrophic nonobstructive cardiomyopathy (Type I); and one atrial septal defect (Type II). We conclude that abnormal interventricular septal motion may occur with Type I WPW abnormality. Other abnormalities are detectable by echocardiography in a high proportion of WPW patients, but do not appear to be associated with any single Wolff-Parkinson-White type.

WITH THE ADVENT of echocardiography, the examination of interventricular septal motion has assumed an important role in the diagnosis of cardiac abnormalities. There are many conditions which are known to affect interventricular septal motion. These include right ventricular volume overload, interventricular septal ischemia or fibrosis and left bundle branch block. In Wolff-Parkinson-White (WPW) syndrome abnormal ventricular depolarization occurs, but whether this results in abnormal septal motion is unknown.

Since its recognition in 1930 there have been numerous reports of cardiac abnormalities associated with WPW. These include Ebstein's anomaly, tricuspid atresia, ventricular septal defect, atrial septal defect, coarctation of the aorta, corrected transposition of the great vessels, tetralogy of Fallot and idiopathic hypertrophic subaortic stenosis. The true incidence of these conditions associated with WPW is unknown.

The purposes of this study were to utilize echocardiography to determine the motion of the interventricular septum in WPW, and to assess the incidence of cardiac abnormalities associated with the WPW syndrome.

Methods

Patients were identified by reviewing the ECG files at the University of Iowa Hospital and Veterans Administration Hospital in Iowa City. Preliminary ECG criteria for identification were a P-R interval of less than 0.12 sec and a delta wave. No attempts were made to select patients by age, sex or cardiac diagnosis. All patients with at least one ECG meeting these preliminary criteria were contacted by letter and telephone; 26 responded and these were the subjects of the study.

All 26 patients underwent physical examination, 12 lead scalar electrocardiography, Frank system vectorcardiography, phonocardiography, chest X-ray in PA and left lateral positions and echocardiography. Vectorcardiograms were recorded on either polaroid film or an Electronics-for-Medicine photographic recorder. All patients displayed slowing of the initial 30 msec forces on the vectorcardiographic recordings.

The patients were classified by use of precordial leads of the scalar ECG and by vectorcardiography into three types using criteria proposed by Boineau et al.: Type I — a right ventricular anterior pre-excitation where the onset of pre-excitation depolarization is presumed to be localized adjacent to the anterior atrioventricular groove within the right ventricle. On scalar electrocardiography (fig. 1) there is a positive delta wave in lead V6 and a negative delta wave in V1 and V2. The initial 30 msec vector in the horizontal plane lies between 300 and 360°. Type II — a right ventricular posterior pre-excitation where the onset of depolarization occurs at the posterior atrioventricular groove in the right ventricle resulting in a positive delta wave in V2-V6 and a negative delta wave in V1 (fig. 1). The initial 30 msec vector in the horizontal plane lies between 0 and 60°. Type III — a left ventricular posterior pre-excitation where the onset of depolarization occurs in the left ventricle along the posterior atrioventricular groove. The electrocardiogram shows a positive delta wave in V1-V6 (fig. 1). The initial 30 msec vector in the horizontal plane lies between 60° and 90°.

Echocardiograms were performed with a Smith-Kline Ekoline 20 ultrasonoscope using a 7.5 cm focused 2.25 MHz transducer, and recorded either on a Honeywell 1856 fiberoptic strip chart recorder or an Electronics-for-Medicine photographic recorder. The examination was performed with the patient in supine and/or slight left lateral decubitus positions; the transducer was placed in the fourth or third intercostal space at the left sternal border, and the beam was directed inferiorly to the mitral valve echo in order to assess the systolic motion of the interventricular septum. Systolic septal motion was classified as normal, paradoxical (anterior during systolic ejection) or flat. Echocardiographic diagnosis of mitral prolapse, idiopathic hypertrophic subaortic stenosis, congestive cardiomyopathy and atrial septal defect was made according to previously published criteria. Eight patients underwent...
right and left heart catheterizations and two had coronary arteriograms.

**Results**

The results are summarized in table 1. Twenty-three patients demonstrated pre-excitation at the time of the study; in three patients previous ECGs and vectorcardiograms had shown WPW, but no WPW was present on any recordings made on the day of the echocardiographic exam. Six patients had Type I WPW, six patients Type II and fourteen patients Type III. Of the Type I patients, five were in WPW at the time of the study. Four of these five showed abnormal septal motion, three with paradoxical systolic motion (figs. 2, 3) and one with flat systolic motion. The latter patient had a hypertrophic nonobstructive cardiomyopathy and a calcified mitral annulus; the remainder of these Type I WPW patients had no known associated cardiac lesions. The fifth Type I WPW patient had normal septal motion. The sixth patient was not in WPW at the time of the study and had normal septal motion. The remaining eighteen patients who were in WPW Types II and III at the time of the study did not demonstrate any abnormal septal motion (fig. 4). Three of the Type III patients had intermittent WPW, and in all minor alterations in systolic septal motion were noticeable when WPW beats were compared to normally conducted beats, but the overall systolic motion of the septum remained within normal limits (fig. 5).

Three patients were not in WPW at the time of the study. One had Type I WPW previously and the echocardiogram was normal. The two other patients who were not in WPW at the time of the study had abnormal IVS motion. One had Type III WPW, flat septal motion and a normal right ventricular dimension; no cause for the septal motion abnormality was evident. The other had Type II WPW with paradoxical systolic septal motion and a large right ventricular dimension. This patient had an atrial septal defect which was considered the probable cause of her abnormal septal motion.

Associated cardiac abnormalities were evident on echocardiographic examination in six patients: two had mitral prolapse (Type II and Type III), and one each had idiopathic hypertrophic subaortic stenosis (Type III), congestive cardiomyopathy (Type III), hypertrophic nonobstructive cardiomyopathy with a calcified mitral annulus (Type I) and atrial septal defect (Type II).

**Discussion**

This study demonstrates that some patients with WPW have abnormal septal motion on echocardiography. This was encountered in WPW Type I where the anomalous bun-

---

**Figure 1.** Scalar ECG patterns in the three types of WPW. The ECGs have been retracted from recordings obtained in this study. In Type I (right ventricular anterior pre-excitation) the delta wave is negative in V1 and V2. In Type II (right ventricular posterior excitation) the delta wave is negative in V1, but positive in V2. In Type III (left ventricular posterior pre-excitation) the delta wave is positive in V1.

**Table 1.** Echocardiographic Studies on WPW Patients

<table>
<thead>
<tr>
<th>WPW type</th>
<th>Number of patients</th>
<th>Septal motion</th>
<th>Associated abnormality</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Patients in WPW at echo study</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>5</td>
<td>Paradoxical – 3</td>
<td>Hypertrophic nonobstructive cardiomyopathy with calcified mitral annulus</td>
</tr>
<tr>
<td>II</td>
<td>5</td>
<td>Normal – 1</td>
<td>Mitral prolapse (1)</td>
</tr>
<tr>
<td>III</td>
<td>13</td>
<td>Normal – 3</td>
<td>Mitral prolapse (1)</td>
</tr>
<tr>
<td>(3 intermittent)</td>
<td></td>
<td></td>
<td>IHSS (1)</td>
</tr>
<tr>
<td>B. Patients not in WPW at echo study</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>1</td>
<td>Normal – 1</td>
<td>Congestive cardiomyopathy (1)</td>
</tr>
<tr>
<td>II</td>
<td>1</td>
<td>Paradoxical – 1</td>
<td>Atrial septal defect</td>
</tr>
<tr>
<td>III</td>
<td>1</td>
<td>Flat – 1</td>
<td></td>
</tr>
</tbody>
</table>
Normally depolarization of subendocardial myocardium in both ventricles is rapid and essentially simultaneous. In left bundle branch block the sequence of depolarization is abnormal, resulting in activation and contraction of the right ventricle before the left ventricular free wall. This may allow the unopposed right ventricular contraction to draw the septum anteriorly toward the chest wall, resulting in the paradoxical septal motion seen by echocardiography in LBBB.

A similar mechanism may be producing the abnormal motion seen in our Type I WPW patients, since right ventricular depolarization precedes left ventricular depolarization in this abnormality. It is noteworthy, however, that we did not encounter in our WPW patients early systolic posterior motion of the septum which is characteristic of LBBB and has been emphasized as a distinguishing feature from the paradoxical septal motion seen in conditions of right ventricular volume overload (the posterior septal motion evident in fig. 2 is presystolic, not early systolic). McDonald believes this motion is due to early and directionally abnor-

**Figure 2.** Echocardiographic recording in WPW, Type I. The interventricular septal motion is paradoxical (anterior) in systole. IVS = interventricular septum. LVPW = left ventricular posterior wall.

**Figure 3.** Echocardiographic recording in another patient with WPW, Type I. The septal motion is paradoxical (anterior) in systole.

**Figure 4.** Echocardiographic recordings of patients with Type II (left) and Type III (right) WPW. Septal motion is normal in both patients.
normal septal contraction in LBBB unopposed by forces generated by a contracting free wall and thereby producing an inward (posterior) movement. In WPW abnormal depolarization is believed to occur via accessory atrioventricular bridges.11 His bundle electrocardiography shows, however, that the His bundle area is depolarized normally, so that at least the upper part of the interventricular septum is activated normally and simultaneously with the abnormal right or left ventricular activation via the accessory pathway.12 The morphology of the QRS complex in most WPW patients is a consequence of this fusion.13 Thus, the preservation of normal septal activation in WPW may explain the absence of the early systolic posterior motion frequently encountered in LBBB. On the other hand, the contribution of each pathway to septal and total ventricular activation may vary within and between patients, and in some cases the septal area might depolarize abnormally (i.e., primarily via the accessory pathway). In such cases an LBBB type early systolic posterior septal motion might occur.

There is no obvious explanation for the normal septal motion seen in one of our five Type I WPW patients. Although we have arbitrarily classified our patients into three types, many intermediate forms are known to exist in WPW;14 it may be that within Type I abnormal septal motion will be characteristic of only a subgroup of patients.

Septal motion was normal in Type II and Type III WPW. In Type III WPW the depolarization of the ventricles is from left to right. Although Type II is a form of right ventricular pre-excitation, the initial posterior site of depolarization is closely adjacent to that seen in Type III, and the sequence of activation of ventricles and septum is probably similar to Type III and to normal patients.

Our study also demonstrates that six of the 26 patients had associated cardiac abnormalities detectable by echocardiography. All these abnormalities have been previously described15,16,17 as associated with WPW. We did not encounter any cases of Ebstein's anomaly, another condition known to be frequently associated with WPW and easily demonstrated by echo.18 The overall incidence of these conditions in WPW remains unknown, but in this study the incidence was 23%. The cardiac abnormalities were not confined to any particular type of WPW but seemed to be randomly distributed among all three types.

Many of these cardiac lesions produce no or confusing symptoms. Since most can be easily demonstrated by echocardiography, a noninvasive and safe technique, we suggest that all patients with WPW syndrome undergo echocardiographic examination as part of a full cardiac evaluation.

Acknowledgment

We wish to acknowledge the dedicated assistance of Ms. Phyllis Shellady, Ms. Linda Rath and Ms. Marianne Hansen.

References

Echocardiography in Wolff-Parkinson-White syndrome.
M S Chandra, R E Kerber, D D Brown and D C Funk

Circulation. 1976;53:943-946
doi: 10.1161/01.CIR.53.6.943

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1976 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on
the World Wide Web at:
http://circ.ahajournals.org/content/53/6/943

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally
published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the
Editorial Office. Once the online version of the published article for which permission is being requested is
located, click Request Permissions in the middle column of the Web page under Services. Further
information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/