The Mechanical Consequences of Anomalous Atrioventricular Excitation (WPW Syndrome)

By Harold W. March, M.D., Arthur Selzer, M.D., and Herbert N. Hultgren, M.D.

Since its description as a clinical entity in 1930, the Wolff-Parkinson-White (WPW) syndrome has been the object of much study and speculation. The essential feature of the disorder is early anomalous activation of at least a portion of the ventricular myocardium as indicated by the "delta" deformity of the QRS complex. Shortening of the P-R segment to 0.12 second or less is accompanied by varying degrees of prolongation or aberration of the QRS complex. Evidence has been adduced in support of the hypothesis that preactivation occurs via a functioning accessory atrioventricular conduction pathway.

The mechanical consequences of this unique abnormality have received less attention. A number of technics have been employed in previous studies. Prinzmetal and co-workers used a high-speed movie camera during electrical stimulation of dog ventricles, and observed a localized area of contraction preceding the normal contraction wave in the same ventricle. Pick and Katz have questioned, however, whether true WPW complexes were produced. Bandiera and Antognetti, employing an improved method or roentgenkymography, reported precontracting areas high in the ventricle, on the left side when the electrocardiogram exhibited predominantly positive QRS complexes in right precordial leads (type A), and on the right side when predominant S waves were recorded in the same leads (type B). Curiously, the same aberrant border motion persisted when the WPW morphology was replaced by normal complexes.

A different point of view has been presented by Ferrer and co-workers. The intervals from the Q wave of the electrocardiogram to the onset of pressure rise in the right ventricle (Q-RVs), and from Q to brachial artery pressure rise (Q-BA) were measured in two patients with WPW undergoing cardiac catheterization. The authors concluded that in each case systole was delayed in both ventricles, and that neither ventricle was normally activated. But in the presence of the characteristic initial deformity of the QRS complex whose origin, time course, and relationship to the main process of activation are not established, it would appear more logical to relate mechanical activity to a portion of the electrocardiogram that is normal, namely the P wave. Measured in this way, i.e., P-RVs, activation of the right ventricle in the first case would be on time, whereas the distinct possibility remains that it would be delayed in the second case. Similarly, the argument for delayed BA would be stronger if it could be shown that the delay occurred with relation to the onset of atrial activity.

Samet et al. studied both types of WPW syndrome by roentgenographic records of aorta and pulmonary artery motion. Movement of the aortic shadow preceded that of the pulmonary artery in three of seven type-A cases, and the reverse was true in two of six type-B cases, but regardless of the presence of asynchrony, ejection was delayed on both sides of the heart in all cases studied. Dack et al., using a similar technic, could not establish significant asynchrony in any of four patients. But in roentgenograms there may be difficulty in distinguishing between intrinsic border motion as distinct from movements of the heart generally, especially in

From the Department of Medicine, Stanford University School of Medicine, Stanford, California.
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Figure 1
Case 1. Twelve-lead electrocardiogram showing WPW conduction, type B. The fine time lines indicate 0.04-second and the heavy lines 0.20-second intervals in this and subsequent figures.

curves taken from the ascending aorta or arch.12

Wolferth and Wood2 recorded the jugular phlebogram in a patient during WPW conduction and observed a bifid "c" wave in which the peaks were separated by a 0.09-second interval.2 The second summit was simultaneous with the carotid upstroke and the first peak was ascribed to early pressure rise in the right ventricle. Unfortunately no control data were available. Moia and Inchauspe14 and Cossio et al.15 have also studied the jugular pulse, submitting as evidence of asynchrony either that the "a"-"c" interval was shortened or that the "c" wave crest was early with WPW beats. The illustrations were not clear, and again no control information was offered. Scherf and Schonbrunner16 reported a longer Q-carotid upstroke interval with WPW beats compared with the patient's normal beats. But the onset of P to onset of carotid pressure rise appeared to remain constant, suggesting that ejection time is unchanged with respect to the start of atrial activation.

Kossmann and Goldberg17 published the phonocardiogram and carotid pulse of a case in which WPW and normal beats alternated, concluding from their material that both ventricles were anomalously excited, because the "intrinsicoid" deflection was delayed in all precordial leads. The second sound was up-split with WPW beats, but with normal beats, pulmonic closure was distinct from and later than aortic closure. Although P-CA was shorter with WPW beats, Q-CA was longer with anomalous conduction as was the Q-T interval of the electrocardiogram. It was concluded that ejection from the left ventricle was delayed in a manner that simulated left bundle-branch block. But this view appears inconsistent with the type of second sound splitting described, which suggests only that with WPW beats, pulmonic valve closure comes earlier. Öhnell18 included two phonocardiograms in his long monograph, showing impurity of the second sound with WPW beats but carotid pulses were not inscribed and the sequence of valve closure could not be determined.

Material and Methods

Since it has been questioned whether experimentally induced fusion beats of the WPW type really pertain to the mechanism of the human disorder, it would seem that further study by the clinical technics of phonocardiograms, pulse recordings, and cardiac catheterization would be useful, especially when the subject can act as his own control by furnishing both normal and anomalous complexes.

When the atrioventricular values are normal and heart failure is not present, the first sound is practically simultaneous with the onset of ventricular systole. This event usually occurs earlier on the left side, and distinct mitral and tricuspid components of the first sound may be identified,
but the separation does not exceed 0.02 to 0.03 second. A wider separation of these components strongly suggests abnormal ventricular asynchrony. In this regard it is important not to mistake an atrial gallop or an ejection sound for a component of a truly split first sound. When the second sound is not a single deflection, the end of systole on each side may be timed from the insura of the carotid pulse, which identifies the component due to aortic valve closure. In phonocardiograms recorded during expiration, pulmonic closure follows aortic by 0.03 second or less, and a prolongation of this interval suggests delayed ejection on the right, early completion of ejection on the left, or both. These considerations are true in reverse when the splitting is paradoxical and pulmonic closure precedes aortic. For reasons already discussed, it is here considered preferable to use the normal onset of the P wave as a reference point for the following intervals: P-1st sound (P-S1), or (P-S1m and P-S1s) when mitral and tricuspid components can be identified, P-2nd sound (P-S2), or (P-S2a and P-S2p) when aortic and pulmonic components are identifiable, P-carotid artery upstroke (P-CAa), and P-carotid artery incisura (P-CAi). A delay of 0.01 to 0.03 second between the aortic closure sound and the carotid incisura is usually present and represents the transmission time along the carotid artery and through the recording apparatus. The latter element is negligible.

Twelve patients with WPW syndrome have been studied in this manner. The 12-lead electrocardiograms were recorded with the Sanborn direct writer. Phonocardiograms and carotid sphygmograms were recorded mainly with the Sanborn "Twin-Beam" but a few of the older studies were done on the Sanborn Tribeam. The sphygmograph transducer was of the crystal type. Apex cardiograms and jugular pulses were usually available but were generally noncontributory. Collateral information from cardiac catheterization was available in one of these patients.

Results

Three patients had phonocardiograms recorded both during WPW and during normal conduction, and in one of them alternation of both types of conduction was present while cardiac catheterization was being performed. Their data are presented first.

Case 1

E. G., was an 8-year-old girl with congenital disease of the mitral valve. A probable history of paroxysmal tachycardia was elicited. The 12-lead electrocardiogram in figure 1 exhibited WPW conduction. The P-R interval was 0.08 second, the QRS complex was aberrant, and its duration was 0.14 second. Prominent delta waves were evident in the left precordial leads, and in V1 a small qR deflection was followed by a deep S wave. The configuration was suggestive of type-B WPW, in which early excitation was thought to occur on the right side.

While the phonocardiogram was being recorded, it was noted that the type of conduction was alternating. The first and third complexes of figure 2 were similar to lead II in figure 1, representing WPW conduction. In the alternate complex, however, the P-R interval was prolonged to 0.18 second and the QRS complex was 0.06 second. Conduction was more normal in these complexes but there was actually persistence of the delta wave. On another occasion a completely normal qR wave was recorded in lead II.

Nevertheless, distinct changes occurred in the phonocardiogram. In figure 2 recorded at the left sternal border, the second sound was essentially pure during "normal" conduction, but distinct splitting was present with the WPW complexes. It should also be noted that the first sound deflections were small and poorly defined with "normal" conduction, whereas they were of considerable amplitude with WPW beats. Moreover, these deflections began 0.05 second earlier in the WPW...
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Table 1
Data for Case 1. (E. S., 9-year-old girl)

<table>
<thead>
<tr>
<th></th>
<th>WPW</th>
<th>Normal</th>
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</thead>
<tbody>
<tr>
<td>Electrocardiogram</td>
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<tr>
<td>P-R, second</td>
<td>0.08</td>
<td>0.18</td>
</tr>
<tr>
<td>QRS</td>
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<td>0.08</td>
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<tr>
<td>P-S</td>
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<td>0.24</td>
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<tr>
<td>Phonocardiogram</td>
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<tr>
<td>P-S1, second</td>
<td>0.19</td>
<td>0.24</td>
</tr>
<tr>
<td>P-S2n</td>
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<tr>
<td>P-S2p</td>
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<td>Mechanical events</td>
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<td>P-CAa, second</td>
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<td>P-CA1</td>
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<td>P-RV</td>
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<tr>
<td>P-FAa</td>
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<tr>
<td>P-FA1</td>
<td>0.58</td>
<td>0.61</td>
</tr>
</tbody>
</table>

Abbreviations: P-R, interval from onset of P to onset of R. P-S, onset of P to end of S wave. P-S1, onset of P to main deflection of first sound. P-S2n, onset of P to pulmonic closure sound. P-S2p, onset of P to aortic closure sound. P-CAa, onset of P to carotid pulse upstroke. P-CA1, onset of P to carotid pulse incisure. P-RV, onset of P to right ventricular pressure rise. P-FAa, onset of P to intraventricular pressure rise. P-FA1, onset of P to infravesicular incisure.

The data (Table 1) indicate that the anomalous atrioventricular excitation is a WPW type with the following characteristics: the interval between P-R was 0.08 seconds, the QRS interval was 0.14 seconds, and the P-S interval was 0.22 seconds. The phonocardiogram showed that the P-S1 interval was 0.19 seconds, while the P-S2n and P-S2p intervals were 0.50 and 0.54 seconds, respectively. The mechanical events showed that the P-CAa interval was 0.30 seconds, the P-CA1 interval was 0.56 seconds, the P-RV interval was 0.16 seconds, the P-FAa interval was 0.35 seconds, and the P-FA1 interval was 0.58 seconds.

Figure 3
Case 1. The second beat is of the WPW type and pulmonic closure precedes the carotid incisure, indicating paradoxical splitting.

Table 2
Data for Case 2. (G. D., 47-year-old woman)

<table>
<thead>
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<th>WPW</th>
<th>Normal</th>
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</thead>
<tbody>
<tr>
<td>Electrocardiogram</td>
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<tr>
<td>P-R, second</td>
<td>0.13</td>
<td>0.18</td>
</tr>
<tr>
<td>QRS</td>
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<td>P-S</td>
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<tr>
<td>Phonocardiogram</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P-S1, second</td>
<td>0.26</td>
<td>0.26</td>
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<tr>
<td>P-S2n</td>
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</tr>
<tr>
<td>P-S2p</td>
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<tr>
<td>Mechanical events</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P-CAa, second</td>
<td>0.27</td>
<td>0.27</td>
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<tr>
<td>P-CA1</td>
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<tr>
<td>CAa—CA1, second</td>
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<td>0.31</td>
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Abbreviations: P-R, onset of P to onset of R. P-S, onset of P to end of S wave. P-S1, onset of P to main deflection of first sound. P-S2n, onset of P to aortic closure sound. P-S2p, onset of P to pulmonic closure sound. P-CAa, onset of P to carotid pulse upstroke. P-CA1, onset of P to carotid pulse incisure. CAa—CA1, carotid pulse upstroke to carotid incisure.

cycles. Pulmonic area sounds recorded along with the carotid artery pulse (fig. 3) demonstrated that the split was paradoxical, i.e., aortic closure followed pulmonic. The second component of the second sound, preceding the carotid incisure by 0.02 second, represented aortic closure, and the first component was inscribed at the time of pulmonic closure. In the curve recorded during electrocardiography, the right ventricular pressure rise began 0.04 second earlier in the anomalous excitation cycles (fig. 4). Finally, it is observed in the simultaneous electrocardiogram and carotid pulse curve that the P-CAa was 0.02 second earlier and P-CA1 was 0.04 second shorter during WPW cycles. A similar tendency was noted in the onset of infravesicular pressure (fig. 4). These observations are summarized in Table 1.

These events indicated that with WPW conduction, the early activated right ventricle contracted and ejected prematurely. These conclusions were supported by the early right ventricular pressure rise recorded during catheterization and by phonocardiograms and carotid pulses, which establish the fact of early pulmonic closure, the latter event preceding aortic closure during anomalous excitation. Since the onsets and incisures of the carotid pulse and infravesicular pressure were also earlier at this time, it seems likely that the left ventricle was at least partly activated in an anomalous manner from the right side.

Case 2
G. D. was a 47-year-old woman in whom the WPW syndrome was diagnosed during routine electrocardiography. She had been in good health but had experienced on occasion short flurries of rapid heart action. Physical examination was unremarkable except for a faint ejection murmur heard at the pulmonic area.

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Figure 4
Case 1. Cardiac catheterization strip. The right ventricular pressure rise occurs 0.01 second earlier with the WPW beat than with the normal beats. The time lines do not photograph. FA: femoral artery. RV: right ventricle.

Figure 5
Case 2. Twelve-lead electrocardiogram showing WPW conduction, type A.

The electrocardiogram shown in figure 5 indicated WPW conduction. The precordial R waves were tall from V2 to V6 (type A). But this type of conduction was unstable and normal beats could be induced during Valsalva straining or on deep inspiration. As WPW beats reappeared in figure 6, the second sound became broadly split. The position of the components with reference to the carotid incisura indicated that aortic closure continued to precede pulmonic closure. Moreover, the intervals from P to aortic closure and to carotid incisura were 0.03 to 0.04 second shorter during WPW conduction. Also the descending limb of the carotid pulse was steeper and the interval from upstroke to incisura was briefer. These observations, summarized in table 2, suggested that during WPW conduction, left ventricular systole was completed earlier. The events on the contralateral side remained unchanged.

Case 3
M. F. was a 29-year-old patient with a history of paroxysmal atrial tachycardia. She had an apical diastolic rumble and the clinical diagnosis of mitral stenosis and WPW syndrome. Figure 7, right, is a 4-lead electrocardiogram recorded during anomalous excitation. The P-R interval was 0.10 second and the QRS was 0.12 second. Delta deformities were present in leads I and CR4. The electrocardiogram on the left, recorded 2 months later, was essentially normal.

Phonocardiograms were recorded during both types of excitation (fig. 8). There was no essential difference between the normal (upper) record and the WPW (lower) record. The measured intervals are assembled in table 3. The deflection following the second sound in both records was an opening snap. This interpretation was further supported by the presence of a similar sound in the apical phonocardiogram, where it initiated a diastolic murmur.

In this case it would appear that anomalous excitation had no mechanical counterpart.

Phonocardiograms and pulses were available during WPW excitation alone in nine
patients. In six of these patients the intervals from onset of the P wave to the time of the first and second heart sounds, and to the upstroke and incisura of the carotid were not remarkable, and in five of them, the first sound was not split while the second sound was either pure or split by less than 0.04 second.

The three other cases were considered separately and their data was summarized in table 4. They were similar in that pulmonic closure was delayed by 0.06 to 0.08 second (figs. 9 and 10). Splitting of this degree was not normally observed in phonocardiograms recorded with the chest held in mid or full expiration. In addition, there was splitting of the first sound in each instance at the left sternal border. The first sound was not nor-

*Figure 6*

Case 2. As WPW beats reappear, the second sound splits widely due to earlier aortic closure. Note changes in the form of the carotid pulse, the incisura appearing early during WPW. The sound deflections have been retouched for clarity.

*Figure 7*

Case 3. The electrocardiogram on the right was recorded during WPW excitation, and the record on the left, made 2 months later, shows normal activation.
Figure 8

Case 3. Electrocardiogram, phonocardiogram, and carotid pulse during WPW conduction (lower record) and during normal conduction (upper record). There are no significant changes in the sounds or in the carotid events.

Table 3

<table>
<thead>
<tr>
<th>Data for Case 3. (M. F., 24-year-old woman)</th>
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<tbody>
<tr>
<td>Electrocardiogram</td>
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<tr>
<td>P-R, second</td>
</tr>
<tr>
<td>QRS</td>
</tr>
<tr>
<td>P-S</td>
</tr>
<tr>
<td>Phonocardiogram</td>
</tr>
<tr>
<td>P-S1, second</td>
</tr>
<tr>
<td>P-S2</td>
</tr>
<tr>
<td>Mechanical events</td>
</tr>
<tr>
<td>P-CA1, second</td>
</tr>
<tr>
<td>P-CA2, second</td>
</tr>
</tbody>
</table>

Abbreviations: P-R, onset of P to onset of R. P-S, onset of P to end of S wave. P-S1, onset of P to main deflection of first sound. P-S2, onset of P to main deflection of second sound. P-CA1, onset of P to carotid pulse upstroke. P-CA2, onset of P to carotid pulse incisura.

Closure in each sample suggests that the mitral and tricuspid sounds had become separated and that right ventricular contraction occurred late. In the one case of this group in which a 12-lead electrocardiogram was available, the precordial configuration was that of type A WPW (fig. 10), but the carotid pulse intervals did not suggest early ejection on the left side. In fact in this subject, and in W.B., the carotid upstrokes and incisuras were 0.02 to 0.03 second later than the mean of a control group, suggesting the possibility that activation was actually delayed on this side.

Discussion

The data presented here have been diverse. Study of the phonocardiogram and carotid pulse in seven patients with WPW furnished no evidence of ventricular asynchrony. Five patients with WPW electrocardiograms did exhibit anomalies of ventricular contraction. It is apparent from the evidence in two cases that during anomalous excitation, either ventricle may initiate or complete its contraction prematurely, and mechanical events on the opposite side may also be early. From three
other cases in this study it also appears that during anomalous excitation, contraction was delayed, especially on the contralateral side, but possibly on both sides.

No single formulation is likely to explain all the observations presented here. When contraction is completely unaffected, it would seem that the electrical prematurity is of limited consequence and that the normal order of ventricular activation has been maintained. This would be most likely to occur when there has been no impairment of transmission.
through the atrioventricular node and when the anomalous entry is at a point relatively distant from ready access to the specialized conduction system. Such a situation might exist if the pathway entered the ventricle anterobasally or posterobasally and subepicardially. In spite of the early insertion of a delta wave, propagation would be slow and the area of delta activated myocardium would be limited by the timely appearance in the normal depolarization wave. With most of the myocardium remaining in the control of the specialized tissue as it were, mechanical events would be unaltered. Case 3 appears to have behaved in this manner.

When mechanical evidence for prematurity is present, it may be supposed that the propagation of the anomalous impulse is favored by a long transmission time through the atrioventricular node, and the entrance of the anomalous impulse in proximity to specialized conduction tissue. This site could be anywhere near the atrioventricular node or in the septum. Such a situation seems to have existed in case 1 in which anomalous excitation caused early contraction in both ventricles. Figure 2, in which WPW and "normal" complexes alternate, is instructive for a number of reasons. It shows in the "normal" complexes that atrioventricular conduction was actually prolonged, since it is 0.18 second in a 9-year-old girl. It is curious that any sort of normalization should occur with a P-R interval of this duration, since one would expect all beats to remain of the WPW type. The morphology of the "normal" beats indicates that activation actually begins anomalously, i.e., one still sees a delta wave, but that in these beats conduction through the anomalous pathway seems to be depressed and impulses over it reach the ventricle only shortly before those from the specialized conduction system, which activates most of the myocardium and inscribes an almost normal QRS complex. Functionally then, in these beats the anomalous pathway is inconsequential, there are no mechanical abnormalities other than the soft first sound, which would be expected with a long P-R interval, and the situation is very similar to that outlined for case 3.

This situation contrasts sharply with the obviously deformed QRS complexes of the WPW beats. The mechanical abnormalities attending these beats are quite different from those described by Prinzmetal in his experimental study.7 Prinzmetal’s cine-film data suggested that during anomalous excitation, a small area in the ventricle precontracts. This contraction is too weak to open the semilunar valve, and also probably too weak to close the atrioventricular valve. Most of the ventricle then contracts normally and the total duration of systole is prolonged. In the WPW beats of case 1, however, the precontracting right ventricle exhibits a sharp early pressure rise as indicated in the cardiac catheterization curve. In left sternal border phonocardiograms the first heart sound appears early after a very short P-R interval and is therefore loud, in contrast to the faint sound inscribed after the long P-R interval.
of the "normal" beats. Pulmonic closure occurs before aortic, resulting in a paradoxical split of the second sound, and the total duration of right ventricular systole is not prolonged. Moreover, the data suggest that systole also begins and ends earlier on the left side as evidenced by the shorter P-S interval of the electrocardiogram in WPW beats, and by the shorter P to carotid upstroke and incisura intervals. In view of the broad, deformed QRS complex in these beats, the spread was probably transmural from right to left. By virtue of the very early appearance of the activation wave in the ventricle with WPW as compared to "normal" beats, the left ventricle is activated early even though this activation probably occurs in a manner similar to that of left bundle-branch block.

In case 2 both ventricles began their contraction on time in WPW beats when compared with normal beats. With WPW beats, the second sound became broadly split due to early aortic closure. Pulmonic closure time was unchanged. The form of the carotid pulse suggests that the normal beats have a longer systole duration than WPW beats and these considerations imply that left ventricular systole is more quickly terminated during the anomalous excitation. The reason for this is not clear. Since the WPW beats were induced by holding the breath after a full expiration, it is possible that the shortening of left ventricular systole was due to the effect of the respiratory maneuver on stroke volume. A full expiration, however, increases intrathoracic pressure and promotes pulmonary venous return. Therefore it usually acts to augment stroke volume in the left ventricle rather than to reduce it.

In the final examples activation was delayed on the right or contralateral side, although no prematurity could be found on the left, presumably the side of the anomalous focus. In Ferrer's experience delay was present on both sides.11 This situation could not be definitely established here. In two of the three cases in this category, however, the carotid upstrokes and incisuras were 0.02 to 0.03 second later than the mean of a control group in which the standard deviation was small, but this can only be regarded as suggestive. If it were the case, one would have to agree with Ferrer that both ventricles are activated in a transmural manner from a high basilar focus in the left ventricle. What has happened to the atrioventricular node and the normal conduction pathways under these circumstances? Their role seems to be obscure. Patient F. T., who belongs in this group, has a typical type A WPW electrocardiogram. He has had anomalous excitation for 14 years, and a normal electrocardiogram has never been recorded. Strenuous efforts were made to produce normal conduction, including hyperventilation, Valsalva maneuvers, 1.6 mg. of atropine intravenously with recordings in both supine and standing positions, 1.0 Gm. of procaine amide intravenously, and 5.0 Gm. of quinidine by mouth. None of these expedients produced normal complexes. It is a distinct possibility that in such a case, the atrioventricular node is nonfunctioning and that the anomalous pathway is the sole avenue of excitation of the ventricles.

Summary

The mechanical consequences of anomalous atrioventricular excitation were studied in 12 patients by means of phonocardiograms, carotid pulse tracings, and in one instance, by data from cardiac catheterization. Three of these patients were also observed during normal conditions so that they supplied their own control data.

In five instances mechanical anomalies were detectable as a consequence of the electrocardiographic abnormality. These anomalies were complex and consisted in one case of early onset and completion of ejection on both sides, and in another case of early completion on the left side. In three instances there was late activation on the contralateral side and possibly on the homolateral side as well.

The remaining cases exhibited no such abnormalities by the methods studied, and in one subject this is illustrated with records.
made during both anomalous and normal conduction.

The literature pertaining to this problem is critically reviewed, and an attempt is made to interpret both the previous and the present observations. The divergent nature of the observations suggests that the effect of anomalous excitation on the mechanics of the heart is the resultant of a number of variables, including the transmission time through the atrioventricular node and the site of entry of the anomalous focus.

It is possible that the atrioventricular node does not function in some cases in which Wolff-Parkinson-White conduction is always present and it is not possible to induce normal beats with drugs, exercise, or respiratory maneuvers.

Acknowledgment

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References


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HAROLD W. MARCH, ARTHUR SELZER and HERBERT N. HULTGREN

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