Anomalous Atrioventricular Excitation  
(Wolff-Parkinson-White Syndrome)

By Louis Wolff, M.D.

An opportunity to study the mechanism in pre-excitation was provided by 3 patients with unusual features: coexistence of right bundle-branch block and pre-excitation, occurrence of 2 types of anomalous ventricular complex, and simultaneous occurrence of pre-excitation and varying grades of atrioventricular block (first degree to complete). The conclusion was reached that a functioning structural atrioventricular neuromuscular bypass is present in these cases.

Although there is general agreement that premature depolarization of a fraction of ventricular muscle accounts for the abnormal electrocardiogram in the Wolff-Parkinson-White syndrome, there is considerable difference of opinion as to the manner in which it is brought about. The problem has been investigated in many ways: study of the electric phenomena associated with the heart beat; the effect on the cardiac mechanism of various pharmacologic agents and maneuvers, including exercise and carotid sinus stimulation; experimental procedures in both animals and man aimed at, or inadvertently producing, electrocardiograms resembling those occurring naturally in patients with the disorder; the anatomic demonstration of accessory atrioventricular (A-V) tracts; and the experimental creation of a short circuit between atria and ventricles capable of reproducing all the features seen in the pre-excitation syndrome. Although final proof is lacking, the weight of evidence favors the hypothesis of anomalous excitation taking place via a functioning accessory muscular bridge that bypasses the A-V node.

The A-V node delays passage of the impulse as it moves from the upper to the lower chambers; when the node is bypassed the A-V transmission interval is abbreviated. A different concept has been proposed, that the shortened transmission interval is the consequence of accelerated conduction in the tissues of the A-V node.

Evidence bearing on the problem will be presented in this paper consisting in vectorcardiographic observations, analysis of arrhythmias, and the demonstration of retrograde conduction from ventricles to atria via an accessory pathway.

Observations
Vectorcardiograms in 4 Patients with Wolff-Parkinson-White Syndrome

The vectorcardiograms reproduced in figures 1 to 4 were obtained from 4 patients with the Wolff-Parkinson-White syndrome. Patients exemplifying a wide range in age and cardiac status were chosen (table 1). The earliest forces, which correspond to the anomalous component or delta wave of the QRS complex, are remarkably similar in all the patients. Since these forces represent premature depolarization of ventricular myocardium, this similarity suggests an identical pathway and mechanism leading to pre-excitation.

The presence of infarction in the anterior and posterior walls of the left ventricle, and of the interventricular septum, apparently does not alter the anomalous mechanism in the cases studied (cases 2 and 4).

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![Diagram of Vectorcardiograms](image)

**Fig. 1** Top. Vectorcardiogram in case 1 (5 year old girl). In figures 1 to 4 arrows indicate direction of inscription of the QRS loop, and each interruption equals 0.0025 second. H = horizontal, S = sagittal, and F = frontal plane projection. The bottom of the figure is anterior (H) or inferior (S and F), and the top of the figure is posterior (H) or superior (S and F), in relation to the patient. The observer's left corresponds to right (H and F), or posterior (S), and the observer's right to left (H and F), or anterior (S) in relation to the patient.

**Fig. 2** Bottom. Vectorcardiograms in case 2 (55 year old man with anterior myocardial infarction). The infarct is concealed in the tracing with anomalous conduction (A), but is clearly evident when conduction is normal (B). Inferior infarct is simulated in the anomalous curve (A).

Similarly, right bundle-branch block in case 4 does not alter the anomalous mechanism, so that right bundle-branch is excluded as the anomalous pathway. The earliest forces in the right branch block tracing (figure 4A) represent ventricular depolarization via the left bundle, and those in the tracings with anomalous conduction (figure 4B) depolarization via the anomalous tract. Since the spatial orientation of these forces is strikingly different, the left bundle-branch may be eliminated as the anomalous pathway.

The claim has been made that disease in the A-V node is responsible for accelerated conduction in portions of the normal A-V connections. However, there is little probability of A-V nodal disease in at least 2 of the 4 cases here described. Since the initial forces in the 4 cases are similar, it is probable that the anomalous pathways are identical. Therefore, if the concept of accelerated conduction is valid, one would have to postulate acquired A-V nodal lesions of a kind which would affect identical portions of the A-V conducting mechanisms; the concept envisages specific destinations of impulses starting in different parts of the A-V node. Furthermore, it has been shown above that the anomalous mechanism utilizes neither the right nor the left bundle-branch; one or the other in all probability would be involved if portions of the normal...
A-V connections were used by the accelerated impulse. While it is possible on theoretical grounds that accelerated conduction might occur near an area of injury, or in areas suffering from some marginal deficiency, there is no proof of such injury and no proof that accelerated conduction actually occurs under the specified conditions. Moreover, such a situation, if it existed, would probably be unstable and temporary.

The diversity and specific nature of the cases studied make it appear unlikely that an anomalous center of impulse formation is responsible for the abnormal vectorcardiogram in all of them.

Finally, all that is required to reconcile the diversity of the cases and the striking similarity of the spatial orientation of the earliest forces of depolarization is the existence of an anatomic functioning accessory pathway located in approximately the same area in all 4 cases.

Arrhythmias

The electrocardiograms reproduced in figures 5 to 7 were obtained on a 53 year old man (case 5) with the Wolff-Parkinson-White syndrome. There was no clinical evidence of heart disease other than paroxysmal tachycardia that had been present for 20 years. Paroxysms occurred with great frequency and evoked annoying palpitation. He died suddenly and unexpectedly 2 years after the tracings were recorded.

A number of short paroxysms of tachycardia were recorded during an observation period of over 2 hours, some of which are reproduced here (figures 6 and 7). Normal ventricular complexes were present in several of the paroxysms, but never in the presence of a sinoatrial mechanism. Two varieties of abnormal ventricular complex were noted when the sinus mechanism prevailed, one of them commonly, the other rarely; the common type never occurred with atrial premature beats, or during paroxysmal rapid heart action.

A complete set of leads (fig. 5) demonstrates the presence of anomalous atrioventricular excitation. A sinoatrial rhythm and the rare type of ventricular complex are displayed in figure 6, top; there is a single atrial premature beat which is followed by a ventricular complex of the same morphologic type. The common variety of anomalous ventricular complex is displayed in the first 3 and last 3 beats in figure 6, middle. The middle 3 beats constitute a short paroxysm of tachycardia initiated by a premature P wave, and the ventricular complexes are of the rare type.

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age</th>
<th>Earliest forces</th>
<th>Terminal forces</th>
<th>Type (Rosenbaum11)</th>
<th>Clinical diagnosis</th>
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<tbody>
<tr>
<td>1</td>
<td>5</td>
<td>LPD</td>
<td>LPU</td>
<td>B</td>
<td>? Congenital heart disease, slight cardiomegaly</td>
</tr>
<tr>
<td>2</td>
<td>55</td>
<td>LPU (LPD)*</td>
<td>LPU (LPU)*</td>
<td>B</td>
<td>Anterior myocardial infarction (confirmed at autopsy)</td>
</tr>
<tr>
<td>3</td>
<td>58</td>
<td>LPU (RAU)*</td>
<td>LPU (LPU)*</td>
<td>B</td>
<td>Normal heart</td>
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<tr>
<td>4</td>
<td>78</td>
<td>LOU (RPU)†</td>
<td>RAU (RAU)†</td>
<td>A</td>
<td>Anterior, posterior, and septal myocardial infarction (confirmed at autopsy); right bundle-branch block</td>
</tr>
</tbody>
</table>

*Normal A-V conduction.
†Right bundle-branch block.
L = left, P = posterior, D = inferior, U = superior, O = no anterior or posterior displacement.
the P-R interval is abbreviated, and is even shorter than the anomalous P-R interval associated with the sinoatrial rhythm.

A longer paroxysm, again displaying the rare type of ventricular complex, is seen in figure 7, top, and, like the shorter one, is initiated by a premature atrial contraction. The QRS-T deflections present with the sinoatrial mechanism before and after the paroxysm are of the common variety. As in the shorter paroxysm (fig. 6, middle) the abbreviated P-R interval is shorter than the anomalous P-R interval during the dominance of sinoatrial rhythm. The cycle length initiating the paroxysm is the same in both; and the rate is 166 per minute in both.

A paroxysm with an identical rate, 166 per minute, but with normal ventricular complexes and normal P-R interval is shown in figure 7, bottom. The cycle length of the ventricular complex initiating the paroxysm is slightly longer than that of the corresponding beats in figures 6, middle and 7, top and longer than the cycle length in the remainder of the paroxysm. This is the result of the longer transmission interval through the A-V node compared to that through the anomalous pathway. The P-R interval is identical in all 3 paroxysms. Prolongation of the R-R interval does not occur in figures 6, middle, and 7, top, because the first QRS in each paroxysm represents a change from one anomalous pathway to the other with the onset of the paroxysm, rather than a shift from an anomalous pathway to the normal A-V connections. The first QRS-T complex of the paroxysm in figure 7, bottom, is different from both the normal and the anomalous beats, probably due to the sudden shortening of the cycle length and consequent aberrant intraventricular conduction. Both paroxysms in figure 7 are followed, after 2 sinus beats, by a ventricular premature contraction with a compensatory pause.

The ectopic atrial pacemaker responsible for the tachycardia in figures 6, middle, and 7, top and middle, is the same in all the paroxysms. In figures 6, middle, and 7, top the P-R interval is abbreviated and the QRS-T complex is anomalous, presumably because the ectopic impulse is conducted over a bypass, the one which is rarely used when the heart rate is slow, and the pacemaker is in the S-A node (fig. 6, top); in figure 7, bottom, the impulse reaches the ventricles via the normal atrioventricular connections, consequently the P-R interval and the QRS-T complex are of normal length.

Ventricular premature beats of identical contour occur in figures 6, bottom, and 7, top and bottom. The first interrupts a sinus mechanism with the rare type of ventricular complex, the last 2 a sinus mechanism with
the common type of ventricular complex. The coupling of the premature beats is fixed, but different from that which initiates the paroxysms of tachycardia, and the cycle length during the rapid heart action. The basic heart rate is variable, so that the fixed coupling suggests a re-entrant phenomenon. Since the course pursued by the anomalous impulse is different for each type of complex, it is unlikely that the re-entrant area lies within the anomalous pathway. The fixed coupling and morphology of the premature beats must mean that part of the sinus impulse traverses the normal A-V connections and somewhere along this pathway encounters the re-entrant area.

The cycle length of the premature beat in figure 6, top, differs from that of the ventricular premature beats. This beat is morphologically similar to the prevailing sinus complexes and there is no compensatory pause.

These features indicate that the mechanism is different from that responsible for the ventricular premature beats. Indeed, the occurrence of an abnormal P wave and abbreviated P-R interval establishes the diagnosis of an atrial premature beat followed by the same kind of atrioventricular excitation that exists during normal sinus rhythm.

These phenomena can be explained by the existence of 2 different bypasses which act as detours for atrioventricular conduction. These pathways are separate and distinct from the A-V node. One is the preferred pathway when the heart is under sinoatrial control, the other is the exclusive anomalous pathway when atrial premature beats or paroxysmal tachycardia occurs.

The fact that there is a preferred pathway when a sinus mechanism prevails suggests that the atrial end of this pathway is more acces-
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Fig. 7. Case 5. Leads B and C are bipolar orthogonal leads in sagittal and vertical axes, respectively. Top. Paroxysmal atrial tachycardia with anomalous ventricular complexes differing from the sinoatrial beats. One ventricular premature beat with compensatory pause. Leads B and C recorded simultaneously and mounted as continuous electrocardiogram. Bottom. Paroxysmal atrial tachycardia with normal ventricular complexes. One ventricular premature beat with compensatory pause. Leads B and C recorded simultaneously and mounted as continuous electrocardiogram.

Pronounced to the sinoatrial impulse than is the origin of the alternate pathway. The latter is the exclusive anomalous pathway for atrial premature beats and paroxysmal atrial tachycardia, suggesting that its origin alone is accessible to the impulse arising in the ectopic atrial focus. The evidence supports the view that a single ectopic atrial focus is responsible for the atrial premature beats and all the paroxysms of atrial tachycardia observed.

There is no evidence of disease of the A-V node, in that signs and symptoms of heart disease are completely absent, and the A-V conduction intervals are normal. The features of the ventricular premature beats indicate that conduction through the normal A-V connections occurs simultaneously with anomalous excitation. Part of the sinus impulse which is transmitted via the A-V node to the ventricles enters the re-entrant area and is responsible for the ventricular premature beats with fixed coupling.

These data cannot be explained by anomalous impulse formation or accelerated conduction through a portion or portions of the A-V conducting system.

Retrograde Conduction

A 22 year old Negro (case 6) was hospitalized after being hit on the head by the tailboard of a truck; he was rendered unconscious for 15 minutes. His only complaints were weakness and dull left parasternal pain. The physical examination disclosed an accentuated pulmonic second sound and a faint systolic murmur in the pulmonary area, but was otherwise not remarkable. There was no cardiac enlargement by x-ray, and the white cell count, erythrocyte sedimentation rate, and Kahn tests were normal.
The day after admission he complained of increased weakness and inability to sleep because of persistent precordial pain radiating into the left shoulder and left upper arm. Comfort was secured with codeine and an uneventful course ensued.

Electrocardiograms were obtained on August 29, August 30, September 3, and September 4, 1945.

The electrocardiogram in figure 8 reveals pre-excitation. Most of the beats are normal, but anomalous complexes occur singly, and in groups of 2 or 3. The "normal" P-R interval is constant at 0.19 second, and the anomalous P-R interval is 0.13 second; the P-J interval of the normal and anomalous complexes is 0.27 second. A sharp upward deflection follows the delta wave in the anomalous ventricular complexes in lead II.

The electrocardiogram in figure 9 discloses first degree A-V block, and normal ventricular complexes with a single exception in lead II; the rhythm is bigeminal. The P-R interval of the first beat in every pair is 0.18, and of the second beat 0.19 to 0.31 second. The P-J interval of the first beat of every pair and of the single anomalous complex in lead II, is 0.28 second. Inverted P waves (P') are superimposed on the S-T segments of the beats which initiate the long pauses, i.e., the second beat of each pair, and the third beat of the triad. The ventricular complexes which precede the P' deflections invariably are of the normal variety. There are 36 normal and 1 anomalous complex, and 18 inverted P waves. The RP' intervals are constant at 0.12 second.

The electrocardiogram in figure 10 displays first degree and high grade A-V block and intermittent pre-excitation; the beats are arranged in groups separated by long pauses. In each cluster of anomalous beats there are at least 2 types of abnormal QRS-T complex. An inverted P wave invariably deforms the S-T segment of the last ventricular complex of a group, except when the latter is anomalous.
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In this circumstance all the QRS-T complexes in the group are abnormal, and the P wave which follows the last QRS-T is blocked (Wenckebach phenomenon). The anomalous P-R interval is constant, but the "normal" P-R interval varies between 0.31 and 0.39 second; the P-P intervals, at times, display progressive lengthening. The first beat in each group is anomalous. The QRS-T complex preceding the inverted P wave is normal without exception, whether or not the group contains 2, 3, or more beats. The first beat of every group displays a sharp upstroke or downstroke which is missing in the subsequent anomalous beats. The RP' intervals are constant at 0.12 second. The entire tracing contains 12 normal and 37 anomalous beats, and 11 inverted P waves.

The electrocardiogram reproduced in figure 11 is normal; the P-R interval is constant at 0.19 second.

INTERPRETATION OF ELECTROCARDIOGRAMS

The sharply inverted P waves are characteristic of retrograde conduction, either via the A-V node, or an accessory muscular bridge connecting the lower and upper chambers. Retrograde conduction of the sinoatrial impulse by way of re-entry through the A-V node may occur when the combination of retarded antegrade conduction in one section of the node and unidirectional block in another exists; if the P-R interval is sufficiently prolonged, retrograde conduction through the area of unidirectional block will occur. The presence of retrograde P waves after normal P-R intervals (fig. 9) excludes this possibility in the case under discussion.

The other possibility, retrograde conduction through an accessory A-V pathway, is supported by the facts. Inverted P waves never follow anomalous complexes because the muscular bypass ef, just having transmitted the sinus impulse to the ventricles, and the activated myocardium at f (fig. 12) are refractory. The normal depolarization front and the anomalous depolarization wave meet and become extinguished, precluding access of the normal impulse to the accessory tract. That the association between a normal QRS-T and P' is not fortuitous is demonstrated in figure 9; the second QRS-T of each group of beats is followed by an inverted P with a single exception, that of the anomalous beat of the triad.
in lead II; the third beat of the triad, however, is normal and is followed by a retrograde P wave. The interdependence of inverted P waves and normal ventricular complexes is indicated by their respective incidence in figures 9 and 10; the lack of complete numerical correspondence between normal ventricular complexes and inverted P waves is due to the fact that retrograde conduction does not occur unless there is prolongation, no matter how slight, of the normal P-R interval (fig. 12). Inverted P waves never occur after the first QRS-T of a group of beats, either because the leading beat is anomalous, as it is, without exception, in figure 10, or because the ‘‘normal’’ P-R interval of the first beat of a group is not long enough, as in figure 9.

If one offshoot of the sinus impulse in the first beat of each pair in figure 9 is conducted through the A-V node, and another simultaneously penetrates the accessory A-V bridge but is not delivered to ventricular muscle, abnormal prolongation of the refractory period of the anomalous bundle must be the cause. This refractoriness prevents retrograde conduction through the accessory A-V connection when the normal depolarization front reaches the ventricular portion of the bypass f (fig. 12). However, minimal increase in the normal P-R interval (second beat of each pair in figure 9) allows complete recovery by the time the normal depolarization wave reaches f; retrograde conduction follows. The capricious and unpredictable appearance and disappearance of anomalous beats in pre-excitation may be explained by this characteristic of the accessory tract. Changes in vagal tone and in heart rate are the known factors that influence the refractory period and thereby affect conduction. The pattern of increasing P-P and P-R intervals in figure 10 is undoubtedly a manifestation of changing vagal tone. Data of a different sort, presented elsewhere,\(^8\) led to the conclusion that vagal control plays an important part in anomalous conduction.

Retrograde conduction via a muscular bypass is consistent with the brevity and constancy of the R-P' intervals, despite the wide
range of the preceding P-R intervals. It is probably more than coincidence that the anomalous P-R interval and the R-P' interval are approximately of the same order of magnitude; the anomalous P-R interval is a measure of conduction time over pathway acf, while the R-P' interval is a measure of conduction time over pathway dgfe (fig. 12); the anomalous tract, ef, with a transmission interval estimated to be 0.06 second, is common to both pathways.

The R-P' interval is remarkably constant, and is 0.12 second regardless of the length of the preceding P-R interval. It is similar, in this respect, to the remarkable constancy of the anomalous P-R interval under widely varying conditions. Undoubtedly a single explanation is applicable to both phenomena, involving, in part, a single pathway for the impulse which is responsible for the anomalous P-R interval and for the retrograde RP' interval; this is the tract ef. Since the QRS interval of the normal beats displays no variations, the transmission interval dgf (fig. 12) will be constant. There is no reason to suppose that variations in atrial transmission rates occur under the existing conditions; indeed, the duration of the P waves is constant throughout all the electrocardiograms. Therefore, if the tissues of the common pathway are endowed with an extremely brief relative refractory period, variations (first degree block) of the conduction interval will not occur though complete block will; this is in keeping with the facts.

The P waves are sharply inverted because the atrial end of the muscular bypass is far removed from the S-A node, lying either near the A-V node, or in the left atrium. Since the A-V node is asymmetrical in relation to the atrial mass the mean axis of atrial depolarization will be oriented to the left when retrograde conduction begins near the A-V node, and to the right when the bypass is connected to the left atrium. Consequently the P' deflection in lead I will be upright (or diphasic) in the former and inverted in the latter situation. In the case under discussion the P' waves in lead I are inverted, suggesting that the atrial end of the bypass is related to the left atrium.

Other mechanisms that have to be consid-
Fig. 12. Case 6. Normal A-V pathway \( a \)bed, and anomalous A-V pathway \( aef \). The time required for the sinoatrial impulse to reach the ventricular myocardium via \( abed \) is 0.18 second (fig. 9), and via \( aef \) is 0.13 second (fig. 8); the former is the normal P-R interval, and the latter is the anomalous P-R interval. The known facts in relation to conduction in atrial and ventricular muscle, A-V node, and Purkinje tissue permit the following transmission intervals to be postulated: \( ab = 0.06, bc = 0.11, cd = 0.01, ae = 0.07, cf = 0.06, fg = 0.04, abedf = 0.24 \), and \( abedge = 0.30 \) second. Interval \( ef \) may lie anywhere between 0.001 and 0.01 second, depending on the proportion of Purkinje fibers in this area; for our present purpose it matters little whether it is 0.01 or 0.001, so long as the same value is used in all the calculations. Interval \( df \) = 0.06 second, which is the interval between the beginning of QRS and the peak of R in normal complexes in lead III.

If the sinoatrial impulse uses both pathways simultaneously, and the anomalous offshoot is blocked at the ventricular end of the anomalous tract \( f \), conduction through the latter will not be evident. The normal offshoot of the sinoatrial impulse will reach the area of block via \( abedf \) in 0.24 second. It is not conducted beyond \( f \) when the normal P-R interval \( ad \) is 0.18 second, but reaches its destination in the atria when the P-R interval is 0.19 second (fig. 9). It is noteworthy that the retrograde conduction interval RP' is constant at 0.12 second, with a range of magnitudes of the preceding P-R interval from 0.19 to 0.39 second. When the preceding P-R interval is 0.18 second retrograde conduction does not occur. Since first degree A-V block of anomalous conduction is not observed under these unique conditions, it is postulated that the relative refractory period of the accessory bundle is very brief.

As possible explanations for the inverted P waves are the premature discharge of ectopic atrial or A-V nodal centers and direct mechanical or electric stimulation of the atria by ventricular contraction.

The morphology of the abnormal P waves, the absence of either fixed coupling or a common cycle length, and the exclusive occurrence of P' deflections after normal ventricular complexes militate against their origin in an ectopic atrial focus.

The constant R-P' intervals suggest a ‘lower’ A-V nodal mechanism with antegrade and retrograde conduction producing ventricular complexes and inverted P waves, respectively. However, the constancy of the R-P' intervals despite the pattern of orderly and progressive increase in the P-R intervals is against this possibility. Failure of the A-V node to escape during the long pauses is also inconsistent with this explanation.

Direct mechanical or electric stimulation of the atria by ventricular contraction has been suggested as a cause of the ventriculo-atrial sinus arrhythmia that occurs in A-V block and must be considered as an explanation of the inverted P waves in the electrocardiograms under discussion. Since the refractory period of atrial myocardium is not a factor, the apparent dependence of inverted P waves on prolongation of the P-R interval is inexplicable on the basis of direct stimulation. The latter, likewise, does not explain the occurrence of inverted P waves exclusively after normal ventricular complexes. Furthermore, it is not known that direct stimulation of the type mentioned is capable of producing sharply inverted P waves, or P waves of constant morphology, as observed here.

The most plausible explanation, then, for the inverted P waves is retrograde conduction through a muscular bypass circumventing the A-V node and functioning as a short circuit between the lower chambers and the left atrium.

**Discussion**

Theories dealing with the mechanism of pre-excitation may be divided into 2 fundamentally different groups: an anomaly of con-
duction, and an anomaly of impulse formation. In acute experiments designed to prove the concept of anomalous impulse formation electrocardiograms have been obtained which resemble clinical pre-excitation, but it is likely that ectopic impulse formation or other definitive mechanisms differing from pre-excitation are responsible for the observed phenomena. Experimental production of perpetual anomalous impulse formation has not been achieved. On the other hand, there is available considerable evidence of different kinds in support of the hypothesis of a muscular bypass of the A-V node, and new data have been presented in this paper.

Atrioventricular conduction in the Wolff-Parkinson-White syndrome displays properties that sharply differentiate it from conduction through the A-V node. The P-R interval is markedly abbreviated in the former, and, though variations in its length are not observed, complete blockade of the pathway occurs frequently and under many conditions. On the other hand fluctuations in transmission intervals over the normal A-V route are common and at times striking, but complete block is infrequent. Evidently the relative refractory period of the tissues of the accessory tract are remarkably brief in contrast to its marked prolongation, at times, in the A-V nodal tissues. In pre-excitation, quinidine and procaine amide either block A-V conduction completely or appear to have no effect on transmission intervals; they prolong conduction time without completely blocking passage of the impulse in the normal A-V bridge. Digitalis is capable of prolonging or completely blocking conduction in the A-V node, while it apparently favors anomalous atrioventricular conduction without changing the transmission interval. Vagal stimulation may prolong A-V conduction time or temporarily block the normal pathway, and it may dislocate the pacemaker from the S-A to the A-V node. In pre-excitation vagal stimulation does not change anomalous conduction time and has no other effect on anomalous conduction provided the cardiac pacemaker remain in the S-A node. It may cause the appearance of anomalous conduction if performed when the mechanism is normal, or, as in the presence of a normal mechanism, may dislocate the pacemaker from the S-A to the A-V node, at the same time converting anomalous to normal ventricular complexes.

These contrasts suggest that the tissues of the anomalous pathway and the A-V node are fundamentally different. These observations, together with others presented above, cannot be reconciled with the concept of accelerated conduction, which envisages conduction rates in the tissues of the A-V node greatly in excess of those with which we are familiar, and which treats the A-V node as the 'central nervous system' of the ventricles. These data provide no evidence that specific linkage of elements of the A-V node to predetermined fractions of ventricular myocardium is responsible for the anomalous complexes.

The hypothesis of a pathway that bypasses the A-V node is based on considerable experimental and clinical knowledge. The histologic demonstration of such pathways, their experimental creation, and a vast amount of electrocardiographic data support this concept. Certain tissues, for example atrial myocardium, conduct impulses at rates far in excess of the A-V node. Such tissues by creating a short circuit, make it possible for the impulse to escape the delaying action of the A-V node. The location of the accessory tract, whether imbedded in the A-V node, or far removed from it, is important only in respect to its effect on the morphology and duration of the anomalous complex; it so happens that the evidence indicates that the bypass is usually far removed from the A-V node. If the concept of accelerated conduction in the A-V node were correct, the tissues carrying the anomalous impulse would be expected to lie within or close to the node. The observations presented above reveal no such proximity.

If it is agreed that direct stimulation of one chamber by another does not occur, the electrocardiographic data here discussed can be interpreted to mean that a muscular bridge, probably the one which is responsible for the inverted P waves, is the same pathway
that conducts the anomalous sinus impulse to the ventricles and produces the delta wave. This is deduced under the stated conditions from the fact that the anomalous P-R interval is fixed, despite the presence of variable first degree and high grade A-V block of the normal A-V bridge.

The conclusion is warranted that the mechanism responsible for the Wolff-Parkinson-White syndrome is a functioning structural accessory A-V bridge that bypasses the A-V node, where normally the impulse is delayed. Early delivery of the impulse to the lower chambers initiates premature ventricular activation with consequent shortening of the P-R interval and lengthening of the QRS interval. Retrograde conduction through the same pathway occurs, and is a possible explanation for the atrial arrhythmias that occur in the syndrome. The noteworthy observation that the anomalous P-R interval and the retrograde R-P' interval are constant under a wide variety of conditions strongly suggests that part of these transmission intervals is a measure of conduction time through one and the same pathway.

Summary

Vectorcardiographic and electrocardiographic data in patients with the Wolff-Parkinson-White syndrome have been presented.

The significance of the spatial orientation of the earliest forces in the vectorcardiogram, an analysis of arrhythmias, and the demonstration of retrograde conduction have been applied to a study of the mechanism of anomalous atrioventricular excitation.

The data support the concept of one or more accessory functioning structural bypasses anatomically and functionally separate and distinct from the atrioventricular node.

It has been shown that the accessory tract is capable of retrograde conduction, thus providing a possible mechanism for atrial arrhythmias.

Summary in Interlingua

Es presentate datos vectorcardiographie e electrocardiographie ab patientes con le syndrome de Wolff-Parkinson-White.

Le significacion del orientation spatial del fortias initial in le vectocardiogramma, un analyse de arrhythmias, e le demonstration de conduction retrograde esseva applicate al studio del mecanismo de anormal excitation atrioventricular.

Le datos supporta le conception que il existe un o plure functionante shuntages structural accessorii que es anatomicamente e functionalemente separate e distincte ab le nodo atrioventricular.

Es monstrate que le via accessorii es capace de conduction retrograde, de maniera que illo representea un mecanismo possibile pro le arrhythmias atrial.

References


**John Wesley**  
**Physician and Apothecary**

"For more than twenty years I have numberless proof that regular physicians do exceedingly little good. I have, therefore, believed it my duty within the last four months to prescribe for between six and seven hundred of the poor in this city, medicine I know was proper." So began a letter written January 25, 1746, by one John Wesley, preacher, founder of the Methodist Church, scorned by the High Church of England.

John Wesley came to Oxford in June 1720, a week after his seventeenth birthday. It is not generally known that this fifteenth offspring of seventeen children spent great segments of his time in the study of medicine and natural philosophy at this university before he was ordained five years later. His friend and first biographer said, "Natural History was a field in which he walked at every opportunity to contemplate the structure of natural bodies and the instincts and habits of the animal creation." As a matter of fact, it was with difficulty that Wesley kept his devotion to physical science from trespassing upon his call to preach the gospel.

After his ordination and before he left for his mission in Georgia in October 1735 he made "anatomy and physic the diversion of his leisure hours."

In Georgia he continued to administer to the sick in body while carrying on his ministry to the spirit. He was interested in medicine as practiced by the Indians, and studied the native herbs from which they concocted their medical brews. He must have known Dr. Samuel Nunez, Spanish-speaking Portuguese Jew, first practitioner of medicine in the colony of Georgia, who had anchored at Tybee Island July 11, 1733, six months after Oglethorpe's arrival. For Wesley writes in his Journal about learning Spanish from his Jewish parishioners.

And he knew and exchanged medical lore with John Regnier, male nurse of the Moravians, and helped Regnier do the first autopsy in Georgia. The patient died because of a "Hematoma (blood clot) of the abdominal wall among other things."

After a confusing year and nine months Wesley left the New World on board the *Samuel* in December 1737 and returned to both his preaching and body healing in England—ALFRED A. WEINSTEIN. *John Wesley Physician and Apothecary*. The Georgia Review 10: 1, 1956.
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LOUIS WOLFF

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