Wolff-Parkinson-White Syndrome

Report of a Case Associated with Wandering Pacemaker, Atrial Tachycardia, Atrial Fibrillation, and Incomplete A-V Dissociation with Interference

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It is the purpose of this communication to present a case of pre-excitation (Wolff-Parkinson-White syndrome) associated with various arrhythmias, some hitherto not described, and to stress the many features of this unusual case that support the bypass concept of the origin of this syndrome.

The electrocardiographic pattern of short P-R interval and prolonged QRS with a normal P-S (Wolff-Parkinson-White syndrome, pre-excitation) has fascinated physicians since its description by Wolff, Parkinson, and White in 1930.1 Many theories have been advanced and numerous studies reported in an effort to explain this particular electrocardiographic pattern.2-4 Of the many concepts proposed, the 2 most widely accepted theories are the existence of an accessory pathway (bundle of Kent) and the presence of an irritable ventricular focus. It is not the purpose of this report to review either the concepts or the clinical significance of this syndrome, but to present a case that may have a bearing on the origin of W-P-W. This case has at one time or another manifested nearly all the hitherto reported disturbances of impulse formation and conduction complicating pre-excitation and, in addition, an arrhythmia that to our knowledge has previously not been observed with W-P-W.

Clinical History

The patient, a 58-year-old Negro woman was admitted to the Indianapolis General Hospital on April 27, 1956, with a history of progressive dyspnea first noticed in December 1955, followed by pedal edema, persistent cough, and extreme weakness. The patient apparently had been in good health and had required no medical attention prior to onset of present illness.

Examination on admission disclosed marked congestive failure, hypertension of 150/100-110, a regular tachycardia of 150, and marked cardiac enlargement. A mild hypochromic anemia, albuminuria, hematuria, a fixed specific gravity, and elevation of the blood urea nitrogen to 60 mg. per cent was also present.

The patient responded to treatment for congestive failure and the arrhythmias and was discharged on June 29, 1956. She was readmitted 3 times thereafter, however, because of generalized anasarea.

Description and Analysis of the Electrocardiograms

Figure 1A (April 27, 1956). This tracing was obtained before any medication was administered. The P-R interval is short (.08 second), the QRS prolonged (.12 second) with a distinct delta (Δ) wave on the upstroke, and the P-S interval measures .20 second. The S-T segment and T wave are opposite in direction to the QRS complex. A ventricular premature systole is present in lead I. The beginning of the R wave of this premature beat resembles very closely the delta wave of the other complexes. The findings here represent the Wolff-Parkinson-White syndrome with a ventricular premature systole.

The similarity of the first portion of the ascending limb of the ventricular premature beat to the delta wave suggests that the premature beat originates either near or in the bundle of Kent or in the irritable focus, depending on the concept of origin of W-P-W.

Figure 1B (September 2, 1956). This graph shows the normal pattern. P-R, QRS, and P-S measure .16, .08, and .24 second respectively. The T waves are inverted in leads I, aVL and V5.

Figure 2A. The 3 tracings (all lead I), each obtained on a different day, represent effect of

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Fig. 1. A. Control tracings obtained on admission. Wolff-Parkinson-White syndrome with a ventricular premature systole. B. Normal conduction. The T waves are inverted in leads I, aV1, and V5.

Fig. 2. A. Effect of vagal pressure on W-P-W. Note the change in rate and appearance of P and QRS waves. B. Lead I of a tracing with normal conduction showing appearance of idioventricular rhythm after vagal pressure.
vagal pressure on the W-P-W. In all 3 instances the heart rate slowed and the QRS contour changed. In the first and second strips, in addition to QRS alteration, 2 different P waves are present. The slightly taller, more pointed P wave is followed in each instance by a narrower and taller QRS (.14 second) complex with a P-S of .26 second. The shorter more rounded P wave is followed by a broader and lower QRS (.16 second) and P-S of .24 second. In the third strip only one type of P wave can be identified. Here the QRS shows gradual prolongation (.13 second to .16 second), the delta wave becomes more prominent, and P-S varies from .22-.26. The R-R intervals in the third tracing show a gradual change, while in the first 2 the variation of P wave is accompanied by a more abrupt change in R-R interval.

The change in contour of P wave and QRS complex can be easily explained by the well documented effects of vagal stimulation in the normal subject: (1) displacement of the pacemaker with change in appearance of the P wave, (2) delay of conduction at the AV node, (3) increase of rate of conduction through the AV node. The displacement of pacemaker toward the node and acceleration of conduction via the node explains the "paradoxical" response observed in the first 2 strips, namely, that the vagal stimulation resulted in narrower and more normally appearing complexes. In the third tracing the delay in conduction via the node gave rise to the expected increase in aberrancy of the QRS complex, by allowing more of the ventricle to become activated by an abnormal route.

Figure 2B (September 28, 1956). This figure shows lead I of a normal tracing. In the upper strip, either coincidentally with vagal pressure or as a result of the latter, an atrial premature systole appears and is followed by sinus arrest and a series of aberrant ventricular complexes. Similar QRS complexes (also the result of a vagal pressure) appear in the second strip. These are preceded by normal P waves with a slightly changing P-R interval. The third and fourth beats of the middle tracing, however, represent fusion (combination) with the sinus beat. The lower tracings shows a spontaneous atrial premature systole with escape.

The first strip shows sinus arrest, atrioventricular dissociation, and an atrial premature beat with an escape beat. In each instance the subsidiary pacemaker results in identical QRS complexes. These QRS complexes could originate in (1) ectopic ventricular focus or (2) AV node (with aberrant conduction-preferential pathway). The differential diagnosis between the 2 would be difficult, if not impossible, were it not for the appearance of the fusion beats (beats 2, 3 in strip 2). These suggest strongly that the focus is in the ventricle and that we are dealing with an idioventricular rhythm. Furthermore, nodal beats without aberrant conduction are presented in figure 4B. Changes described here are pertinent to the interpretation of figure 6.

Figure 3 (May 19, 1956). This tracing shows parallel variation of heart rate and of the appearance of P and QRS. With abrupt slowing of the rate the P wave becomes more upright (except in V1), the P-Δ longer, the QRS more normal (narrower, the delta wave forms a smaller portion of the ventricular complex) and the P-S shortens from .26 to .22 second.

When the P is associated with more normal QRS complexes, the P-S interval shortens (such a shortening of P-S was not observed in figure 2). The factors that resulted in slowing of heart rate in this instance not only displaced the pacemaker, perhaps toward the AV node, but also increased the conduction rate through the node. Such a displacement toward the node, and away from the bypass would explain the prolongation of the P-Δ interval. The relative prolongation of the P-Δ speaks very strongly against the irritable focus concept of W-P-W. There should be no reason for a delay in appearance of the delta wave if the latter resulted from mechanical stimulation of an irritable focus. The observation of similar changes during vagal stimulation (fig. 24) suggests that the "spontaneous" changes in this instance may also be due to vagal effect.

Figure 4A (May 12, 1956). Lead V1 shows atrial tachycardia with 1:1 response. The P-S cannot be determined. The QRS measures .14 to .16 second. The entire descending limb is thick and slurred.

Lead V1 (May 10, 1956) discloses an atrial
Fig. 3. This tracing shows parallel variation of heart rate and appearance of P and QRS. These changes occurred “spontaneously.”

Fig. 4. A. Atrial tachycardia in presence of W-P-W. Left upper strip shows 1:1 response, while the right reveals a 2:1 response. Lead V₅ represents 1:1 response punctuated by cycles of 2:1 response. B. Atrial tachycardia with 2:1 response and normal atrioventricular conduction, atrial standstill, and nodal rhythm. Note the broad and notched P waves. C. Atrial tachycardia with 2:1 block showing transition from W-P-W to normal conduction.
tachycardia with an atrial rate of 180 and ventricular responses at 90 per minute. The delta wave occupies one-half the descending limb of the complex. The remainder of the QRS is slender and narrow and its entire width measures .12 second.

Lead V4 (May 9, 1956) represents atrial tachycardia (rate 160) with a dominant 1:1 response punctuated by single cycles of 2:1 response. In the first QRS complex following a 2:1 response the ascending limb of the R is composed of an initially slurred (Δ wave) and a final narrow portion. The QRS complex measures .12 second and is of the RS type with a prominent S wave. Subsequent beats are of the RR’ type. The R wave is short with both limbs being slurred. The RR’ is narrow and the S wave is very shallow. The width of the QRS is .16 second.

That the arrhythmia is due to a rapid discharge from an ectopic focus, rather than a circus movement involving the bundle of Kent is suggested by (1) the P waves are not retrograde, (2) first beat of each cycle in lead V5 shows the characteristic fusion complex, (3) some of the P waves are not followed by QRS complexes (it would be impossible to explain the subsequent P-QRS if one were to assume a circus movement via bypass and AV junction). These tracings also show a decided difference between refractory period of the bundle of Kent or the ventricular musculature as compared with the normal conduction path. When the ventricular rate is slow (V1 with 2:1 response) and when the QRS follows a pause (first beat of each cycle in V5), the QRS is narrow and represents a fusion with conduction through the node. When, however, the rate speeds up there is marked slurring and prolongation of the complex, indicating that most if not all of the ventricle is activated via bundle of Kent or from the irritable focus. That fusion is no longer present is indicated by the change in the terminal portion of the QRS complexes in V4 with diminished depth of S wave. Furthermore, these beats are almost identical to those observed in figure 5 in which one can be quite sure that fusion does not exist.

Figure 4B (May 15, 1956). Atrial tachycardia with an atrial rate of 150 and 2:1 ventricular response. There are periods of atrial standstill with nodal rhythm. The P waves are peculiar in appearance, being broad (.16 second), notched, and upright in V1 and V2. They resemble to some extent the P waves observed in lead V1 in figure 3. The P-R measures .22 second.

This tracing was taken after 1.5 Gm. of quinidine was administered in an 8-hour period. Although the dose was not excessive it could account for the return to normal of the QRS, marked prolongation of the P waves, and finally atrial standstill with nodal escape.

Figure 4C (May 15, 1956). Atrial tachycardia with an atrial rate of 160 and 2:1 response. The first 4 ventricular complexes are of the W-P-W type with a small P wave, a P-R of .12 second, and P-S of .28. Subsequent beats are normal. The terminal portion of both the W-P-W and normal complexes have the same appearance and the P-S remains constant, indicating fusion. It is significant to note that the P-Δ and P-R are longer than in any other tracing. Such a delay of ventricular activation speaks strongly against the irritable focus concept of W-P-W and in favor of a bypass.

Figure 5 (May 15, 1956). The dominant rhythm in this tracing is atrial fibrillation, and the ventricle is activated entirely through an aberrant pathway (fig. 6). Exceptions are the normal beats that occur after an interval of .80 second. Such long intervals preceding the normal beats suggest that the refractory period of the normal conduction tissue is longer than that of the aberrant pathway. The few beats with a W-P-W complex occurring after an R-R of .80 second can be easily explained by assuming that the bypass has been invaded by fibrillatory waves without invoking a ventricular response (concealed conduction). As a result, aberrant conduction after the longer R-R (.80 or longer) tends to give an erroneous impression of the refractory period of the bypass. In 1 instance a normal beat follows a normal QRS after a period of only .72 second. This beat may represent an attempt at an active nodal rhythm (nodal tachycardia). The burst of remarkably regular aberrant rhythm with a rate of 160 beats per minute suggests a mechanism other than atrial fibrillation. It may be that it repre-
sents a tachycardia originating in or near the bypass. There are a few complexes that differ from both the dominant W-P-W and the normalized beats (e.g., beats 5 and 14 in fourth strip). These beats follow a prolonged R-R and represent aberrant conduction due to prolongation of the refractory period of the ventricles (Ashman phenomenon) superimposed on aberration secondary to W-P-W mechanism ("double aberrancy").

The lack of mechanical contraction of the atria and the presence of concealed conduction speak very strongly against the irritatable focus concept of W-P-W. The appearance of W-P-W complexes after periods varying from .80 second to over 1.0 second cannot be easily explained except by concealed conduction. Such a mechanism is not applicable to the irritatable focus concept. To accept the existence of an irritatable focus, one has to assume that its threshold of response to stimulation varied markedly, thus resulting in wide variation of the R-R interval.

Figure 7 (May 3, 1956). The arrhythmia complicating W-P-W represented in this tracing has hitherto not been described. This tracing was obtained after 3.0 mg. of digitoxin was administered in 3 days and reveals a double tachycardia with an incomplete atioventricular dissociation. At different times the ventricles are controlled by one or the other of the pacemakers. Occasionally impulses from both pacemakers invade the ventricle at the same time, giving rise to fusion (combination) beats. Two distinctly different (X, Y) and a number of intermediary (Z) QRS complexes are noted. The latter no doubt represent fusion (combination) between complexes X and Y.

The dominant rhythm is incomplete AV dissociation. The P-P intervals vary from .44 to .48 second and R-R interval from .40 to .44 second. The first of the Y complexes present in leads II, III, aV L, V 1, and V 4 appear earlier than the dominant QRS (X) would be expected (after an interval of only .32 second). It represents invasion (capture) of ventricle by a supraventricular impulse. It is obvious when
fig. 7. Incomplete atrioventricular dissociation with periods of activation of ventricles through the bypass. Note the idioventricular (X) complexes, Wolff-Parkinson-White (Y) complexes and the combination of the two (Z).

compared with other tracings that the initial portion of the Y complexes is a delta wave and that the QRS pattern is the W-P-W type. The initial portion of the fusion beats (Z) is the delta wave and the terminal portion represents excitation from the ectopic focus. The 2 possible sites of ectopic pacemaker responsible for the X type of QRS are in the ventricular musculature and the AV node with aberrant conduction. Because the QRS (X) complexes resemble the idioventricular complexes in figure 2B and because nodal beats without aberrant conduction were present in figure 4B, we consider this ventricular rather than nodal tachycardia. If such is the case, it is difficult to accept the irritable focus concept as the origin of the W-P-W. For the latter to be true, one would have to postulate the existence of 2 competing ventricular pacemakers without any depression of either, as shown by the ability of a second ectopic pacemaker to respond within .32 second. Such a situation is highly unlikely. On the other hand, these findings are easily explained by the bypass concept. The aberrant path has a very short refractory period allowing early activation of the ventricles by this route. That the aberrant path has a refractory period that is shorter than the normal conduction pathway is also shown in figures 4A and 5.

Summary

A case of Wolff-Parkinson-White syndrome (W-P-W) complicated by many different arrhythmias in a patient with severe heart disease is presented.

Several interesting features have a bearing on the etiology of W-P-W and some in our opinion strongly support the bypass concept: (1) incomplete AV dissociation with interference giving rise to W-P-W complexes, idioventricular complexes, and the combination of the two; (2) “double aberrancy”; (3) concealed conduction; (4) difference in refractory period of the 2 pathways resulting in changing appearance of QRS; (5) changing P-Δ interval; (6) variation in contour of P and QRS secondary to vagal stimulation or occurring “spontaneously” parallel with a change in heart rate; (7) findings that suggest that impulse can originate in the bypass without a preceding P wave and thus without evidence of mechanical stimulation; and (8) presence of W-P-W during atrial fibrillation where mechanical contraction of the atria is absent.
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SUMMARIO IN INTERLINGUA

Es presentate un caso de syndrome Wolff-Parkinson-White in un patiente con sever morbo cardiac, complicate per numerose differente arrhythmias.

Plure interessante aspectos del caso es de interesse pro le etiologia del syndrome e in nostre opinion supporta fortemente le concepto del circumconduction. Iste aspectos es: (1) Incomplete dissociation atrio-ventricular con interferentia, resultante in complexos de Wolff-Parkinson-White, complexos idioventricular, e combinationes del duo; (2) "duple aberrantia"; (3) conduction celate; (4) differentias del periodo refractori del 2 vias, resultante in alterationes de QRS; (5) alteration del intervallo P-A; (6) variation de contorno in P e QRS, secundari a stimulation vagal occurrente "spontaneamente" parallel a alterationes del frequentia cardiac; (7) constatationes que suggere que impulsos pote originar se in le circumconduction sin precedente undas P e assi sin evidente stimulation mecanic; e (8) presentia de complexos de Wolff-Parkinson-White durante le fibrillation atrial quando contraction mecanic del atrio es absente.

REFERENCES

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