Wolff-Parkinson-White Syndrome

Conversion of Type A to Type B
Electrocardiographic Changes

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SUMMARY
This paper reports a patient with Wolff-Parkinson-White syndrome who presented with a tachycardia showing an unusual QRS morphology closely resembling that of a ventricular tachycardia. On reversal to a normal rhythm the electrocardiogram showed changes of a type A preexcitation, with subsequent conversion to a type B pattern. This phenomenon was observed on two separate occasions. Such conversion would suggest the presence of two distinct sites for bypass location resulting in preexcitation, thus lending support to the theory of aberrant atrioventricular conduction via a bundle of Kent or neuromuscular tissue connecting atrium to ventricle. The configuration of the QRS complexes during the tachycardia would appear to be due to a circus movement with antegrade anomalous atrioventricular conduction and retrograde atrial stimulation via the bundle of His, although in this case, due to the presence of two distinct anomalous atrioventricular conduction pathways, both antegrade and retrograde conduction via these two pathways alone cannot be excluded.

Additional Indexing Words:
QRS complex Preexcitation Tachycardia Atrioventricular conduction

The electrocardiographic changes of a short P-R interval with slurring of the QRS complex due to a delta wave, occurring in healthy adults prone to attacks of supraventricular tachycardia, was described by Wolff, Parkinson, and White in 1930. In 1945, Rosenbaum, Hecht, Wilson, and Johnson classified the electrocardiographic changes seen in this syndrome into two types, depending on the polarity of the delta waves in the right precordial leads. In type A the delta wave is upright in all the precordial leads, with small or absent S waves, while in type B the delta wave is negative, with prominent S waves in the right precordial leads. Although these classifications appeared somewhat arbitrary, they corresponded to the vectorcardiographic groups described by Bleifer, Kahn, Grishman, and Donoso, where the delta vector or the delta plus QRS sE loop in type A is anteriorly oriented at +30 to +120 degrees in the horizontal plane, while in type B these vectors are oriented between +30 and -60 degrees in the horizontal plane.

The mechanisms underlying the occurrence of these electrocardiographic changes have been studied, and there is evidence to show that the type A pattern is due to preexcitation of the left ventricle, while type B is due to preexcitation of the right ventricle, the excitation being mediated through neuromuscular tissue.

A patient with preexcitation presenting with a tachycardia showing an unusual QRS morphology is being reported. On reversal to a normal rhythm, a type A electrocardiographic pattern was observed which subsequently converted to a type B pattern, a phenomenon which does not appear to have been reported.
Case Report

P. J., female, aged 24 years, was admitted to the hospital having had continuous palpitations for 9 days and a history of similar attacks over several years. The pulse and heart rate were 180 per minute, and no clinical abnormality could be detected in the cardiovascular system.

The initial electrocardiogram showed a tracing resembling a ventricular tachycardia with large, broad QRS complexes at 180 per minute. On reversal to a normal rhythm after intravenous lidocaine the electrocardiogram showed a type A pattern of preexcitation with positive QRS complexes and delta waves in all the precordial unipolar leads and negative deflections in leads III and aV_F.

The electrocardiogram done on the next day showed changes of a type B pattern with prominent negative QRS complexes and delta waves in the right unipolar chest leads and in lead V_1, and positive QRS complexes and delta waves in the left precordial leads; leads III and aV_F now showed positive complexes. This type B pattern persisted until the patient was discharged on the ninth day.

This sequence of events was once again observed when she was readmitted with another attack of paroxysmal tachycardia (figs. 1 and 2).

Discussion

The unusual features in this case are, first, the conversion of a type A electrocardiographic pattern to a type B change and, second, the occurrence of a tachycardia with an unusual morphology which has been referred to as a pseudoventricular tachycardia. These features were constantly observed on two different occasions.

The changing electrocardiographic patterns seen here are significant with regard to the mechanism of bypass location occurring in this syndrome and to the QRS morphology observed during the tachycardia. Although preexcitation was thought to occur in two distinct

Figure 1

Electrocardiogram taken during tachycardia showing the large, broad ventricular complexes, the tracing closely resembling that of a ventricular tachycardia. Lead II during the termination of the arrhythmia is also shown.
sites in the right ventricle in both the type A and type B patterns, it is now considered that the right ventricle is the site for preexcitation in cases with the type B change, while the left ventricle is probably the site for preexcitation in those cases showing a type A change. This latter contention is supported by studies reporting (1) the association of bundle-branch block in patients with the type A and type B preexcitation changes; (2) anatomic descriptions of neuromuscular atrioventricular connections and the bundle of Kent in patients with type B preexcitation; (3) the epicardial excitation of the ventricles; and (4) the observation that preexcitation associated with Ebstein’s syndrome is of type B, while in the rare cases of an Ebstein anomaly of the left side in corrected transposition the preexcitation is of type A configuration.

The occurrence of both type A and type B preexcitation in a single patient does not appear to have been recorded in earlier reports, and this phenomenon would seem pertinent to the mechanism of preexcitation. Such conversion of type A to type B in a single patient would suggest that preexcitation is possible in two distinctly different sites in the same ventricle or, more probably, in either ventricle, which necessarily lends support to the theory that preexcitation occurs via an accessory bundle or through neuromuscular tissue connecting the atria and ventricles. It is evidence at variance with the hypothesis that aberrant conduction in the atrioventricular node itself may be the cause for preexcitation.

The configuration of the ventricular complexes during the tachycardia may also be related to the presence of this dual pathway.

Figure 2

Electrocardiogram taken immediately after termination of the tachycardia shows a type A pattern (time, 4 PM), while that recorded the following day (9:30 AM) shows the type B change.
for atrioventricular preexcitation in this patient. Durrer, Schoo, Schuilenburg, and Wellens have discussed the evidence for the existence of antegrade and retrograde impulse conduction in the accessory bundle and the atrioventricular node as being responsible for the mechanism causing the tachycardias in patients with this syndrome, this being supported by clinical arguments in favor of retrograde conduction via the accessory pathway during supraventricular tachycardia.

In their paper, Durrer et al. state that...

"...attacks of supraventricular tachycardia may be initiated by an atrial or ventricular premature beat, if differences in the state of refractoriness of both bundles [in this case, the bundle of Kent and the atrioventricular nodal connection] are present. The atrial premature beat will have to be blocked in the Kent bundle but conducted to the ventricles through the normal atrioventricular conduction system (fig. 1C). If a ventricular premature beat could initiate an attack of supraventricular tachycardia, conduction in the Kent bundle from ventricles to atria has to be present, and the atrioventricular conduction system must be refractory temporarily, with the refractoriness disappearing when the retrograde atrial impulse reaches the atrioventricular node. No such attack will occur, if retrograde Kent conduction is blocked.

For a ventricular type of tachycardia—if possible at all in the WPW syndrome (fig. 1D)—the postulated pathway of excitation during the initiating atrial or ventricular beat, as outlined above, has to be reversed. In this instance an atrial beat has to be blocked in or near the atrioventricular node, but conducted to the ventricles through the Kent bundle, with retrograde activation of the atrioventricular conduction system, and atrial activation by way of the atrioventricular node. For a ventricular premature complex to initiate a circus movement of this type, an essential condition is the presence of retrograde block in the Kent bundle during this beat."

These workers also state that

"The only specific mechanism, inducing ventricular tachycardia unique for the WPW syndrome that we can conjecture at this moment, is a circus movement in the reverse direction compared with the one present in supraventricular tachycardia. Basically this is a supraventricular type of tachycardia with exclusive antegrade anomalous A-V conduction and retrograde His conduction. The diagnostic criteria then would be regular broad ventricular complexes of about the same rate as the rate during supraventricular tachycardia, with pre-excitation configuration and 1:1 atrioventricular nodal P waves. A survey of the literature gives only one case probably fulfilling these criteria."

It would appear, then, that the broad ventricular complexes observed in this patient during tachycardia were due to a circus movement of the type described above, but with the hypothetic possibility that antegrade and retrograde conductions could also have occurred via the two distinct atrioventricular connections (irrespective of the atrioventricular node itself) in this patient, as evidenced by the existence of both type A and type B pre-excitation.

References

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