

## Wide QRS Complex Tachycardia in a Patient with Wolff-Parkinson-White Syndrome and Cardiomyopathy: What is the Mechanism?

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### Case Presentation

A 32-year old woman with severe mitral regurgitation, congestive heart failure, and Wolff-Parkinson-White syndrome developed frequent, nonsustained episodes of a wide QRS complex tachycardia several days postpartum. Figure 1 shows an ECG with telemetry lead I and modified chest lead (MCL) recorded while the patient complained of palpitations. The same pattern of initiation and termination of the wide QRS complex tachycardia was seen repeatedly. An example of ventricular preexcitation during sinus rhythm is shown in an ECG with identical leads in Figure 2. What is the mechanism of the wide QRS complex tachycardia?

### Commentary

The ECG in Figure 1 shows a wide QRS complex tachycardia at a cycle length of 240 msec with an apparent 1:1 AV relationship. The differential diagnosis of a wide QRS complex tachycardia with a 1:1 AV relationship includes ventricular tachycardia, antidromic reentrant tachycardia, and paroxysmal supraventricular tachycardia with aberrant conduction.

Inspection of the first three QRS complexes in Figure

1 shows sinus rhythm at a cycle length of 640 msec followed by a slightly premature atrial depolarization with a different P wave morphology and shorter PR interval. The first three QRS complexes are not preexcited. It is not possible to discern whether the wide QRS complex depolarization at the onset of the tachycardia is preceded by a premature atrial depolarization. Therefore, inspection of the initiation of the tachycardia is not useful in differentiating ventricular tachycardia from antidromic reentrant tachycardia or supraventricular tachycardia with aberrant AV conduction.

The open arrow following the first wide QRS complex in Figure 3 shows an atrial depolarization that is seen with a fixed AV relationship during the tachycardia. At the termination of the tachycardia, the closed arrow shows an atrial depolarization with a longer ventriculoatrial interval that is followed by a narrow QRS complex. Termination of ventricular tachycardia should be independent of events arising in the atrium or AV node. Therefore, ventricular tachycardia is exceedingly unlikely to terminate simultaneously with a change in the AV relationship unless the tachycardia cycle length changes as well. Termination of supraventricular tachycardia, however, frequently occurs with a simultaneous change in the AV relationship. Additionally, there is no reason for termination of ventricular tachycardia to be linked with a narrow QRS complex unless a supraventricular depolarization captures the ventricle prematurely, which is not seen in this case. Conversely, termination of a supraventricular tachycardia with aberrant AV conduc-

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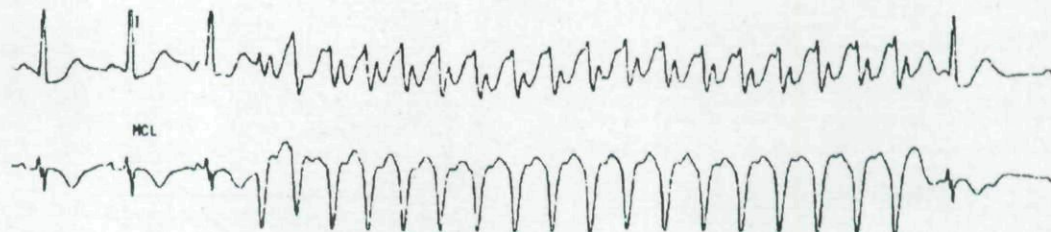


Figure 1.

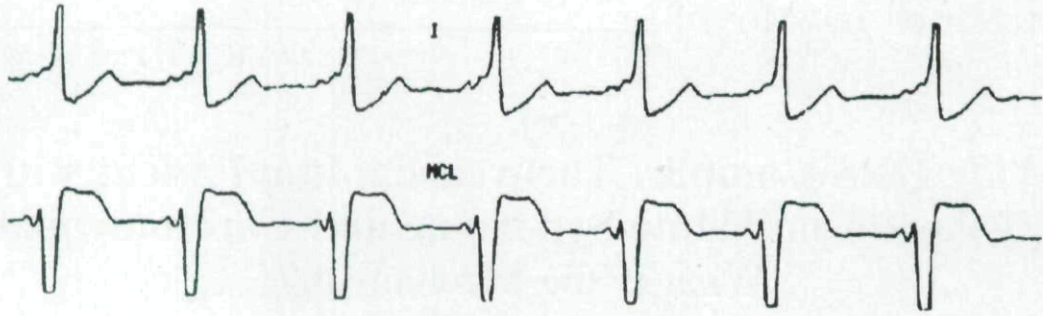


Figure 2.

tion may be preceded by a longer cycle length and subsequent loss of bundle branch block, as is seen in this case.

The remaining differential diagnosis of the supraventricular tachycardia mechanism includes antidromic or orthodromic reentrant tachycardia, AV nodal reentrant tachycardia, and atrial tachycardia. Antidromic reentrant tachycardia is unlikely given the morphology difference seen in lead I between the QRS complexes in the tachycardia (Fig. 1) and preexcited sinus rhythm (Fig. 2). Further evidence against antidromic reentrant tachycardia lies in the observation that the RP interval is shorter than the PR interval during the tachycardia. Atrial tachycardia is less likely than orthodromic reentrant tachycardia or AV nodal reentrant tachycardia because the AV relationship appears to be critically linked to the termination of the tachycardia. Between orthodromic reentrant

tachycardia and atypical AV nodal reentrant tachycardia as the underlying supraventricular tachycardia mechanism, the observed intermittent preexcitation during sinus rhythm and the RP-PR relationship during tachycardia weigh heavily in favor of orthodromic reentrant tachycardia.

An electrophysiology study reproduced the wide QRS complex tachycardia and its characteristic termination with a narrow QRS complex. The patient was found to have a single, right posterolateral accessory pathway and dual AV nodal pathways. The mechanism of the wide QRS complex tachycardia was orthodromic reentrant tachycardia with aberrant conduction that terminated with retrograde block in the accessory pathway and a single (retrograde slow-antegrade fast) AV nodal reentrant beat with loss of aberrant conduction.

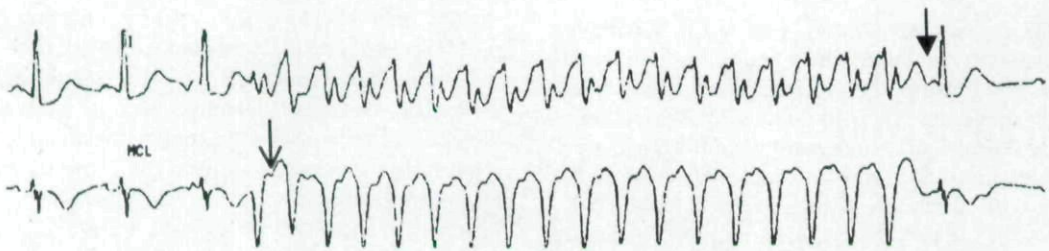


Figure 3.

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