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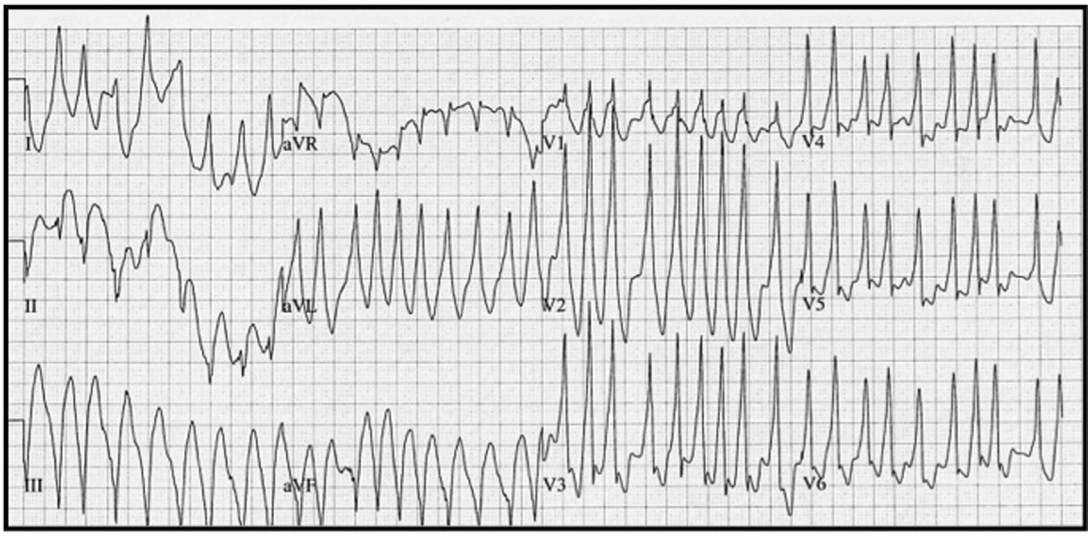


**Figure 1.** ECG monitor demonstrating a rapid, irregular, wide QRS complex tachycardia. There is significant beat-to-beat variation in the width and amplitudes of the QRS complexes. The axis is leftward and constant. Significant motion artifact is noted in leads I and II.

[Ann Emerg Med. 2016;68:92.]

A 20-year-old man without significant medical history presented to the emergency department with palpitations associated with weakness and dizziness during the past several hours. He denied chest pain, dyspnea, loss of consciousness, or other significant complaints. His only medication was clarithromycin for recent bronchitis; he denied illicit substance use. His blood pressure was 112/81 mm Hg, and pulse rate on the monitor was approximately 220 beats/min, with a wide complex rhythm. A 12-lead ECG was obtained (Figure 1).

*For the diagnosis and teaching points, see page 105.*
*To view the entire collection of ECG of the Month, visit www.annemergmed.com*

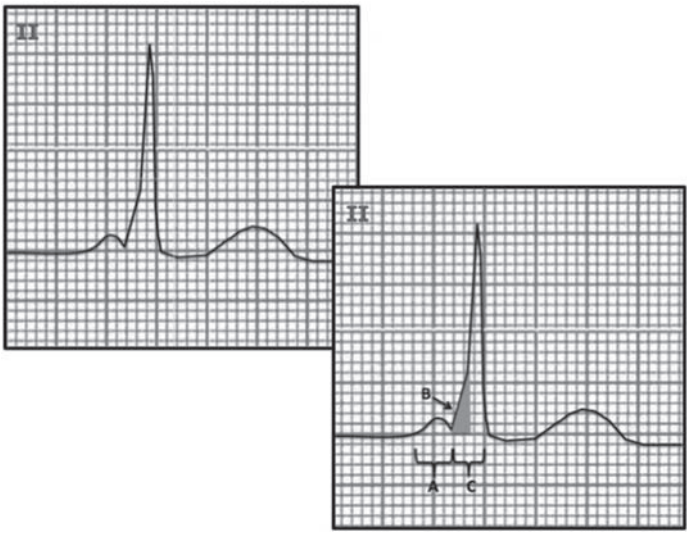


**Figure 2.** The classic triad of electrocardiographic findings in Wolff-Parkinson-White's syndrome during sinus rhythm, including the shortened PR interval (A), Δ wave (B), and minimally widened QRS complex (C).

ECG OF THE MONTH

*(continued from p. 92)*

## ECG ASSESSMENT

The ECG revealed an extremely rapid ventricular rate of 220 beats/min, with a QRS complex duration of approximately 0.20 seconds. Closer inspection revealed that the QRS complex morphology varied in width and amplitude from one beat to the next, and there was variation of the R-to-R intervals.

## CLINICAL COURSE

The rhythm was initially mistaken for torsades de pointes, considering the rapid rate, widened QRS complex, and variation in QRS complex morphologies. The patient was treated with intravenous magnesium for presumed torsades de pointes. Minutes later, he developed hypotension with confusion. He was then electrically cardioverted, with immediate conversion to sinus rhythm and normalization of his clinical condition.

A review of the postconversion 12-lead ECG (in sinus rhythm) revealed the classic electrocardiographic triad of Wolff-Parkinson-White's syndrome, including shortened PR interval, Δ wave, and minimally widened QRS complex (Figure 2). With this knowledge, rereview of the initial ECG resulted in recognition of Wolff-Parkinson-White's syndrome–related atrial fibrillation.

## DISCUSSION

The initial step in the electrocardiographic assessment of a rapid, wide QRS complex tachycardia is assessment of regularity. At more rapid rates, irregularity can be difficult to determine without specific comparison of R-R intervals. Irregularity strongly suggests atrial fibrillation. Causes of wide QRS complexes with atrial fibrillation can be due to an underlying bundle branch block, a rate-related bundle branch block, or ventricular pre-excitation, such as encountered in Wolff-Parkinson-White's syndrome.

Ventricular pre-excitation is defined as a "premature activation of the ventricular myocardium by an impulse that travels by an anomalous path and avoids physiologic delay in the atrioventricular junction."[1] The Wolff-Parkinson-White's syndrome pattern includes the ECG findings of shortened PR interval, a Δ wave, and widened QRS complex[1] (Figure 2). The syndrome is present when patients demonstrate this triad of ECG findings and symptomatic arrhythmia (palpitations, syncope, or actual dysrhythmia). The 4 arrhythmias encountered in Wolff-Parkinson-White's syndrome include orthodromic atrioventricular reentrant (or reciprocating) tachycardia with narrow QRS complex (65%), antidromic atrioventricular

reentrant (or reciprocating) tachycardia with wide QRS complex (10%), atrial fibrillation (25%), and malignant ventricular arrhythmias (rare).[2]

This patient demonstrated Wolff-Parkinson-White's syndrome–related atrial fibrillation with its classic ECG presentation, including a rapid, irregularly irregular rhythm, significantly widened QRS complex, and beat-to-beat variation in the morphology of the QRS complex. Variation of the QRS complex width and morphology results from differences in the contribution of impulses arriving through the accessory pathway and the atrioventricular node. The widest QRS complexes can result from impulses that initiate ventricular depolarization through the accessory pathway; narrow QRS complexes result from impulses that initiate ventricular depolarization through the atrioventricular node. QRS complexes of intermediate duration can result from impulses that initiate ventricular depolarization through both the accessory pathway and atrioventricular node. In a wide QRS complex tachycardia, such occasional narrow complexes can be confused with capture beats and intermediate complexes confused with fusion beats, resulting in the incorrect perception that the findings are diagnostic of ventricular tachycardia, another pitfall in the accurate diagnosis of atrial fibrillation with Wolff-Parkinson-White's syndrome.

Unstable patients with Wolff-Parkinson-White's syndrome–related atrial fibrillation are best managed with synchronized cardioversion, using sedation if the clinical situation permits. Hemodynamically stable patients can be managed with intravenous procainamide (17 mg/kg infused during approximately 45 to 60 minutes) or with synchronized cardioversion and sedation. Medications with significant atrioventricular node-blocking effect should be avoided in all such instances. These agents (amiodarone, calcium channel and $\beta$-adrenergic blocking agents, adenosine, and digoxin) can promote enhanced conduction through the accessory pathway, resulting in an increased ventricular rate and cardiovascular collapse. Patients with this arrhythmia should be subsequently evaluated with electrophysiologic study for possible ablation of the accessory pathway.[3]

The distinction of torsades de pointes from Wolff-Parkinson-White's syndrome–related atrial fibrillation can be difficult. Both rhythms present with tachycardia, wide QRS complexes, and irregularity. In Figure E1*A* (available online at www.annemergmed.com), an example of torsades de pointes is displayed; note the changing polarity, amplitude, and morphology of the QRS complexes. In Figure E1*B* (available online at www.annemergmed.com), an example of Wolff-Parkinson-White's syndrome–related atrial fibrillation is presented. Note the variation in the QRS complex morphologies, with consistent, unchanging polarity; the amplitude of the QRS complexes is changing yet minimally so.

Patients who present with torsades de pointes have many clinical similarities to those with Wolff-Parkinson-White's syndrome–related atrial fibrillation, including the potentially younger age of the patient, absence of significant preexisting cardiac disease, and the ECG findings noted above. The emergency physician incorrectly identified the rhythm as torsades de pointes, considering the QT-interval-prolonging effect of clarithromycin.

If the patient is unstable with either rhythm, then synchronized cardioversion is the therapy of choice. The stable patient with torsades de pointes is best managed with magnesium sulfate and correction of any inciting issues (electrolyte abnormality, medication toxicities, etc); the stable patient with Wolff-Parkinson-White's syndrome atrial fibrillation is best managed with procainamide.

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