A 71-year-old man with a history of previous myocardial infarction was referred to our hospital for recurrent symptoms of palpitation and syncope. During an episode of palpitations, a 12-lead ECG (Figure 1) was obtained in the emergency room. The ECG shows a wide-QRS complex tachycardia at a rate of 167 bpm. The P waves are positive in lead II (↑) before each QRS complex, and the PR interval is 80 milliseconds. The QRS duration is prolonged with apparent slurring of the QRS upstroke, suggesting the possible presence of a delta wave. Are these features consistent with a diagnosis of atrial tachycardia with pre-excitation? However, the possibility of ventricular tachycardia (VT) also should be considered in our patient because of the old myocardial infarction. Therefore, the ECG diagnosis is challenging. What are the diagnosis and best management strategy for this patient?

Please turn the page to read the diagnosis.
A wide complex tachycardia may represent either VT (80%) or a supraventricular rhythm with aberrant conduction (20%). The relationship between the P wave and QRS complex is a key consideration in the differential diagnosis of wide QRS complex tachycardia. When doubt exists in an emergency situation, it is safest to assume that a wide complex tachycardia is VT, particularly in patients with known cardiovascular disease such as prior myocardial infarction. In our case, cardioversion was administered immediately in view of hemodynamic deterioration (hypotension). The ECG obtained after cardioversion, demonstrated in Figure 2, shows sinus rhythm. The wide QRS complexes have 2 different morphologies. First, the conducted QRS complexes have a right bundle-branch block morphology, with Q waves in the inferior leads consistent with an old inferior wall myocardial infarction. In addition, the early QRS complex (the 2nd, 4th, 8th, and 10th QRS complexes in long strip II) is wider than the sinus complexes. No P waves are seen before any of these QRS complexes. Hence, these are premature ventricular complexes.

Comparative analysis of the ECG in sinus rhythm and in wide QRS complex tachycardia may help in the differential diagnosis. In our case, pre-excitation is not observed during sinus rhythm after cardioversion. Although the QRS complex is wide in sinus rhythm as a result of a right bundle-branch block, it is different from the left bundle-branch block morphology seen in tachycardia (Figure 1), suggesting the initial diagnosis of VT in Figure 1. How are we to analyze the P wave in lead II in Figure 1? There are 2 plausible explanations for the appearance of P wave in lead II in Figure 1: true P wave (namely, atrial tachycardia coexisting with VT) or pseudo-P wave. The key to distinguishing a true P wave from a pseudo-P wave is accurately measuring the QRS complex duration, synchronously recording multileads, and confirming the starting point of the QRS complex. Figure 3 (leads II and III) shows the original ECG amplified on the computer to determine the distinct measuring point. The earliest starting point of the QRS complex is in lead III (vertical line), and the vertex of the P wave in lead II is exactly at the beginning of the QRS complex. It is now evident that the descending limb of the P wave was actually the initial portion of the q wave of the QRS complex and that the ascending limb of the P wave was actually the upstroke of the negative T wave. Therefore, what appeared to be a P wave in lead II was actually a pseudo–P wave.

Our patient underwent electrophysiological study and catheter ablation. The wide QRS complex morphology induced during the procedure was identical to the QRS morphology of the tachycardia seen in Figure 1. Intracardiac electrogram revealed atrioventricular dissociation (Figure 4), further confirming that the rhythm in Figure 1 was not VT. However, further investigation revealed that the P wave seen in lead II was actually a pseudo–P wave due to the presence of a left bundle-branch block.

**Figure 2.** The ECG after cardioversion shows right bundle-branch block with an old inferior wall myocardial infarction and premature ventricular complexes.

**Figure 3.** Synchronous recording of leads II and III intercepted in Figure 1.

The earliest starting point of QRS complex is in lead III (vertical line), and the vertex of the P wave in lead II is exactly at the beginning of the QRS complex.
was indeed VT. Radiofrequency catheter ablation of the VT was performed, which rendered the clinical tachycardia noninducible.

In summary, the relationship between the P wave and QRS complex is a key point for the differential diagnosis of wide QRS complex tachycardia. If the P waves in tachycardia are not identical to those seen in sinus rhythm, synchronously recording multileads and confirming the starting and terminal point of the QRS complex can help distinguish a true P wave from a pseudo-P wave.

**DISCLOSURES**

None.

**REFERENCES**


**FOOTNOTES**

*Circulation* is available at http://circ.ahajournals.org.
Wide QRS Complex Tachycardia: What Is the Diagnosis?
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Circulation. 2017;135:1870-1872
doi: 10.1161/CIRCULATIONAHA.117.027647
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2017 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/135/19/1870

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