Amal Mattu, MD; David Zira, MD; Jeffrey A. Tabas, MD; William J. Brady, MD

Electric Countershock. Tachycardia, Ventricular. Arrhythmias, Cardiac.

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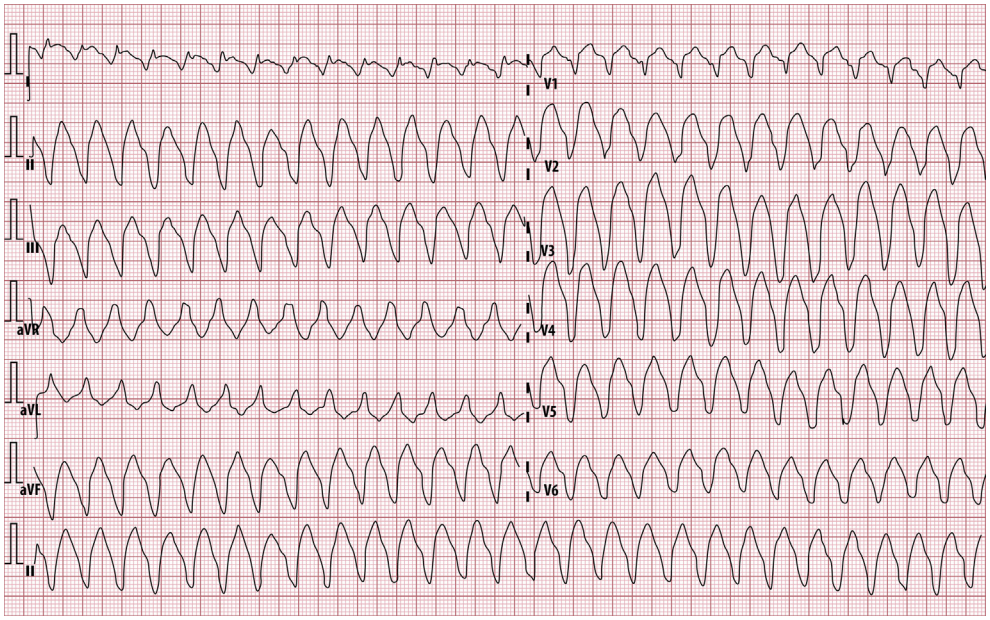

Figure 1. Initial ECG with wide-complex tachycardia at a rate of roughly 165 beats/min. The QRS complex is most evident in lead I in which there is an initial slurred Q wave and relatively narrower subsequent R wave. This provides a clue to identify the QRS complex and subsequent T wave in the synchronously traced leads II, III, aVR, aVL, and aVF. Note that there is a negative concordance of the QRS complexes in all leads except aVR and aVL.

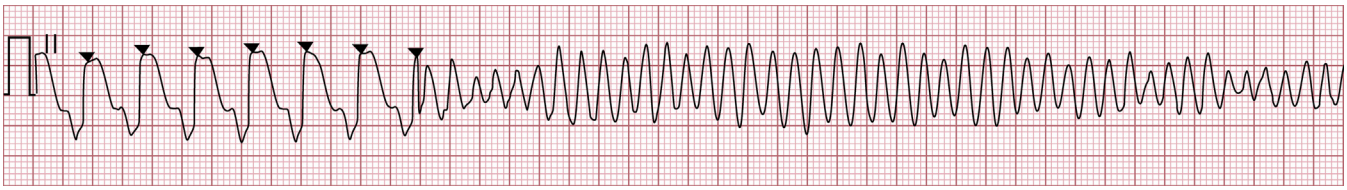

Figure 2. Monitor ECG rhythm strip with initial wide-complex tachycardia (left of the strip) and degeneration after electrical shock to polymorphic ventricular tachycardia (right of the strip). Note the arrowheads, indicating a synchronized setting for electrical cardioversion.

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A 50-year-old man with palpitations and shortness of breath presented to the emergency department. He was immediately placed on a cardiac monitor and noted to have a regular wide–QRS complex tachycardia with a rate of 160 beats/min, a blood pressure of 88/55 mm Hg, and a respiratory rate of 28 breaths/min. The examination was notable for diaphoresis, lethargy, and bibasilar rales. A 12-lead electrocardiogram (ECG, Figure 1) revealed a regular wide-complex tachycardia.

Due to the patient's hemodynamic instability, a decision was made to immediately cardiovert the patient. He was given 20-mg etomidate intravenously followed by synchronized cardioversion using a biphasic defibrillator at 70-J energy. Immediately following the cardioversion, he lost pulses, and the monitor showed polymorphic ventricular tachycardia (Figure 2).

For the diagnosis and teaching points, see page 114.
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ECG OF THE MONTH

(continued from p. 113)

CLINICAL QUESTION

Why did the patient develop polymorphic ventricular tachycardia?

ECG INTERPRETATION

There is a wide-complex tachycardia at a rate of roughly 165 beats/min. There is negative concordance across the precordium (i.e. the QRS complexes are primarily negative in leads V1 through V6) and there are no P waves that can be clearly identified. No further interpretation was attempted given the inaccuracy of algorithms in the emergency setting to distinguish supraventricular tachycardia from ventricular tachycardia.

CLINICAL COURSE

The patient was successfully defibrillated back to sinus rhythm and was admitted to the hospital for further evaluation and treatment.

DISCUSSION

The patient presented with a hemodynamically unstable (but not pulseless) wide–QRS complex tachycardia (Figure 1). Immediate synchronized electrical cardioversion is the recommended therapy in this setting.[1] With proper synchronization, the monitor should identify the QRS complexes and thus determine the appropriate portion of the QRS-T cycle for energy delivery. Delivery of energy on the T wave during cardioversion can result in electrical destabilization and malignant ventricular dysrhythmia. Here, the monitor inaccurately identified the T waves as QRS complexes (Figure 3). As a result, the monitor delivered the shock on the T wave rather than on the QRS complex,

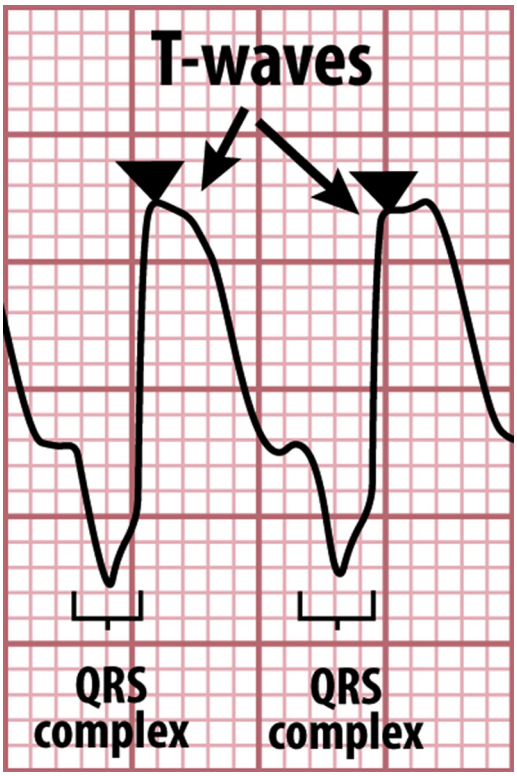

Figure 3. Enlarged image of QRS-T complexes. The QRS complex is distinguished from the T wave by its sharper peak or nadir (nadir in this case), whereas the T wave has a more rounded peak and nadir. The location of the arrowheads demonstrates that the monitor has incorrectly identified the large T waves as QRS complexes.

producing an electrical R-on-T phenomenon, inducing polymorphic ventricular tachycardia. This mechanism is similar to the mechanism of R-on-T-initiated ventricular arrhythmia due to premature ventricular contraction or due to delivery of a ventricular pacing stimulus on the T wave due to failure to sense. In either instance, electrical destabilization can occur.[2,3]

Defibrillation is the preferred approach for treating an arrhythmia resulting in pulselessness to avoid any delays that may result in cardioversion due to tracking of the QRS complex. Cardioversion is preferred in the unstable patient with pulses to avoid malignant ventricular events that can result from defibrillation that falls on the T wave. Rarely, such a malignant ventricular event can result from cardioversion when the ECG demonstrates large T waves that are misinterpreted as QRS complexes, such as in wide–QRS complex tachycardias or in the presence of hyperkalemia.[4] Therefore, it is critical for health care providers to ensure that the monitor is accurately identifying the QRS complex before delivering synchronized cardioversion. In the first ECG, the QRS complex is most evident in lead I in which there is an initial slurred Q wave and relatively narrower subsequent R wave. This provides a clue to identify the QRS complex and subsequent T wave in the synchronously traced leads II, III, aVR, aVL, and aVF. When the monitor is inaccurate in identifying the QRS complex, it is often helpful to change the sensing lead to a different one in which the QRS complexes and T waves are more easily distinguishable. Additionally, the provider can increase the amplitude of the measured waves, thus increasing the size of the QRS complexes and potentially may result in the correct identification of the ECG complexes.

TEACHING POINTS

1. While synchronized cardioversion decreases the risk of an R-on-T phenomenon compared with unsynchronized defibrillation, it may still occur, resulting in pulseless ventricular tachycardia or fibrillation.
2. Situations in which the monitor may track and therefore deliver a shock upon the T wave instead of the QRS complex include some wide-complex tachycardias and severe hyperkalemia.
3. Changing the sensing lead or increasing the amplitude can avoid this problem when these conditions are recognized.

Author affiliations: From the Department of Emergency Medicine, University of Maryland School of Medicine, Baltimore, MD (Mattu); Department of Emergency Medicine, Wolski Hospital, Warsaw, Poland (Zira); Department of Emergency Medicine, University of California—San Francisco School of Medicine, San Francisco, CA (Tabas); Department of EmergencyMedicine, University of Virginia School of Medicine, Charlottesville, VA (Brady).

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