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Ventricular Tachycardias

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While atrial tachycardias are associated with narrow QRS complexes (<120 milliseconds) and use the normal cardiac conduction system for transmission, ventricular tachycardias (VTs) are characterized by wide QRS complexes and do not use the normal cardiac conduction system. Because the signal does not follow the most efficient pathway, conduction takes longer, resulting in a wide QRS complex. Identifying wide- versus narrow-complex tachycardia is one important factor in attempting to differentiate between ventricular and atrial tachycardia. The most common type of wide-complex tachycardia is ventricular tachycardia (VT), followed by supraventricular tachycardia (SVT), which manifests with aberrancy and pre-excitation as in Wolff-Parkinson-White (WPW) syndrome. SVT with aberrancy originates in the atria but is conducted aberrantly and not through the regular conduction system, causing a widened QRS. It is virtually indistinguishable from VT on an ECG and is treated initially just like VT.

Ventricular tachycardia is defined as three or more consecutive ventricular beats (QRS >120 milliseconds) with a heart rate of 100 beats per minute or more. It may originate anywhere in the heart below the AV node. Common etiologies of VT include structural heart disease, ischemia, and electrolyte abnormalities.

**TYPES OF VT**

*Non-sustained VT* lasts less than 30 seconds and terminates without therapeutic intervention. *Sustained VT* lasts longer than 30 seconds, causes significant hemodynamic symptoms (hypotension, syncope), or requires therapeutic intervention to terminate (medications, cardioversion). VT may further be categorized as *monomorphic* (originating from one ventricular focus) (Figure 1) or *polymorphic* (originating from more than one ventricular focus) (Figure 2). The two are differentiated on ECG by the presence of more than one morphology, or appearance, of the QRS complexes. The most common type of polymorphic VT is *torsades de pointes*, meaning twisting of the pointes, which frequently occurs as a result of electrolyte abnormalities or congenital long QT syndrome.

**SYMPTOMS**

Some patients with ventricular tachycardia have no symptoms, while others experience palpitations, syncope, and sudden death. Symptoms depend on how well the tachycardia is perfusing the vital organs, including the brain.

**TREATMENT**

http://www.jaapa.com/ventricular-tachycardias/article/211903/
Treatment of VT consists of rate-lowering medications, such as beta-blockers, or cardioversion via electricity or antiarrhythmic medications. Treatment of torsades de pointes starts with IV calcium infusion because this disorder has a strong association with electrolyte abnormalities, specifically hypocalcemia.

**ECG CHALLENGE**

A 57-year-old male with a history of myocardial infarction underwent coronary artery bypass surgery 1 year ago and was lost to follow-up. Recently, after complaining to his wife of sweats, palpitations, and chest pain, he was brought to the hospital by emergency medical services. The man was responsive but in significant distress and hypotensive. *Figure 3* shows the ECG obtained in the emergency department (ED).

Step-by-step assessment reveals the following:

1. Is the heartbeat regular? Yes, the QRS complexes march out.

2. What is the heart rate? Find a QRS complex on or near a dark line.
   - **Method A:** Counting the large boxes, we see that there are almost 2 large boxes before the next QRS complex. Two boxes would put the rate at about 150 beats per minute, or we could estimate it at 170 beats per minute.
   - **Method B:** There are about 18 QRS complexes in 6 seconds (30 large boxes), which gives an estimated heart rate of 180 beats per minute (18x10).
   - **Method C:** There are two large boxes between the QRS complexes, which gives us 300/2 = 150 beats per minute.

3. There are no discernible P waves on this ECG.

4. PR interval cannot be determined.

5. The QRS complexes span more than 3 small boxes, which is wider than normal.

6. The ST segments are upsloping across most leads. This is a common feature of VTs and is not specific but could indicate ischemia. In more rapid VT (ie, greater than 200 beats per minute), the ST segment is often not visible.

7. The T waves go in a direction opposite to that of their corresponding R wave (ie, T waves go up when R waves go down and vice versa), suggesting T-wave inversion. This, too, is a nonspecific finding.

8. There are no U waves.

This ECG represents a wide-complex tachycardia that meets the criteria for ventricular tachycardia because there are three or more consecutive ventricular beats with a heart rate greater than 100 beats per minute. Because all the QRS complexes in a given lead resemble each other in appearance, this is a monomorphic VT.
The patient was successfully cardioverted in the ED and initially started on antiarrhythmic medications. Later, he received an automatic implantable cardiac defibrillator (AICD). The etiology for his arrhythmia was thought to be myocardial ischemia.

REFERENCES


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