# Ventricular Tachycardia in Coronary Artery Disease

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**KEYWORDS**

• Ventricular tachycardia • Electrocardiograph • Myocardial infarction • Reentry • Mapping
• Catheter ablation

**KEY POINTS**

- The mechanism of ventricular tachycardia (VT) in the setting of previous myocardial infarction is reentrant excitation within the infarct scar.
- The electrocardiograph (ECG) during VT shows a wide QRS tachycardia. The diagnosis is established by excluding aberrant supraventricular and preexcited tachycardias.
- The QRS complex in postinfarct VT is produced when the reentrant excitation wavefront exits the scar border and activates normal ventricular myocardium.
- The ECG is a vital tool in the initial assessment of both the underlying substrate as well as the exit site of scar-related VT.
- The surface ECG is necessary in pace mapping and entrainment mapping during localization and ablation of postinfarct VT.

## PATHOPHYSIOLOGY OF SCAR-RELATED VENTRICULAR TACHYCARDIA

Multiple human mapping studies have confirmed that postinfarct monomorphic ventricular tachycardia (VT) is caused by scar-related reentry.[1–4] The anatomic substrate for VT consists of surviving, poorly coupled myocyte bundles within the dense healed infarct scar. Often, these scars are large and confluent with associated dyskinesis or frank aneurysm formation. Unidirectional conduction block and slow conduction through these bundles facilitate the development of macroreentrant VT circuits.[5] The activation wavefront through these regions during VT is constrained by both dense scar and functional barriers to form the protected diastolic isthmus, during which no electrical activity is recorded on the surface electrocardiograph (ECG). In sinus rhythm, such bundles can be defined by scar heterogeneity on magnetic resonance imaging, electroanatomic voltage mapping, or electrogram analysis.[6–8]

The QRS complex on the surface ECG is recorded when the wavefront exits the dense scar and activates the remaining ventricular myocardium. The vector, duration, and sequence of ventricular depolarization determine the VT morphology on the surface ECG, and these are in turn determined by the size, location, and electrophysiologic characteristics of the infarct substrate.[9] Additional factors such as antiarrhythmic drugs, ECG electrode positioning, surgical scars, and geometric variations in the normal cardiothoracic anatomic relationship can affect the VT morphology.[10] Polymorphic VT is seen predominantly in the acutely ischemic heart and is often caused by Purkinje cell irritability related to diastolic calcium overload, causing triggered activity.

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## GENERAL PRINCIPLES SURROUNDING THE ECG CHARACTERISTICS OF VT

- VT presents on the surface ECG as a wide complex tachycardia (WCT) with ventriculoatrial (VA) dissociation or QRS morphology features of a ventricular origin (see later discussion). If the sinus rhythm ECG shows a wide QRS complex, a narrower QRS during tachycardia is diagnostic of VT.
- Left ventricular (LV) free wall sites of origin give rise to right bundle branch block (RBBB) configurations (net positive QRS complex in V1).
- Left bundle branch block (LBBB) configurations (negative QRS complex in V1) are seen with septal and right ventricular (RV) sites of origin.
- Septal sites of VT origin give rise to narrower QRS complexes as a result of simultaneous rather than sequential LV and RV activation.
- The QRS axis varies most closely with the craniocaudal direction of net ventricular activation (eg, left bundle left superior axis VT exiting from basal septal side of an inferior infarct scar).
- Positive QRS concordance is seen with basal VT origins activating the ventricles toward the anteroapical region, in the direction of the precordial ECG electrodes. This process may also be seen with preexcited tachycardia with antegrade conduction over a bypass tract that inserts into the basal annular region of the ventricle.
- Negative QRS concordance is seen with apical VT sites of origin, in which the net activation vector proceeds away from the precordial leads.

## ECG CHARACTERISTICS OF VT IN CORONARY ARTERY DISEASE

An initial analysis of the sinus rhythm ECG may help in ECG interpretation during VT.

- The absence of pathologic Q waves during sinus rhythm is unusual in patients with postinfarct VT because of the presence of large, often aneurismal, scars. However, on many occasions, Q waves are better appreciated during VT than during sinus rhythm.
- Anteroseptal Q waves or a poor precordial R wave progression suggest previous anterior or septal infarction. Similarly, inferior or lateral Q waves suggest an inferolateral infarct scar.
- Baseline bundle branch blocks in patients with coronary artery disease (CAD) are usually RBBB. The presence of LBBB is unusual after infarct and should raise suspicion of a nonischemic dilated cardiomyopathy (NICM). Such patients may have coexisting CAD, but their VT substrate and electrophysiologic characteristics are typical for NICM.[11]
- Atrial, RV, or biventricular pacing may also be seen, given that many of these patients have implantable cardioverter-defibrillators (ICDs).

In the setting of previous infarction, the RV is rarely involved in VT circuits. Thus the following rules apply:

- RBBB configuration VTs exit the infarct scar on the LV free wall.[9]
- LBBB configuration implies a VT exit on or adjacent to the septum.[9]

Further interpretation of VT QRS morphology is assisted by knowledge of the infarct location. Because of the typically smaller scar mass, ECG localization of VT exit site is more accurate with previous inferior infarction.

### *Previous Inferior Infarction*

- Most VTs in these patients have a basal exit and consequent preserved precordial R waves. However, more extensive infarction can result in apical exits.
- RBBB morphology VT typically exits from the basal lateral aspect of the inferior scar, usually with right superior axis. More superior exits along the lateral wall lead to an increasingly more inferior frontal plane axis (**Fig. 1**).
- LBBB morphology VT (especially with a left superior axis) in this context characteristically exits on the inferobasal septum (see **Fig. 1**).
- Some VTs having these configurations in patients with inferior infarct have the mitral annulus as 1 boundary of their diastolic corridor (so-called mitral isthmus VTs) and may exit this slow zone on either the septal or the lateral aspect of the annulus (see **Fig. 1**).[12]

### *Previous Anterior Infarction*

- LBBB morphology VTs with left superior axis usually exit from the apical septum (**Fig. 2**). However, large anteroseptal infarcts may result in negative precordial concordance with these VTs, with additional Q waves in I and aVL.
- RBBB morphology VTs with right or left superior axis are the most difficult to localize and can arise from multiple regions around the apex. More posterolateral exits distinguish themselves with a greater R wave in aVR than aVL.
- LBBB VT with right inferior axis generally exits from the superior midseptal aspect of the