# Unique ECG During Sinus Rhythm in a Patient With a Postmyocardial Infarction–Sustained Ventricular Tachycardia

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## ECG CHALLENGE

The ECGs in Figure 1A and 1B are from a 72-year-old patient with a history of chronic recurrent sustained ventricular tachycardia (VT) and an inferior nonrevascularized ST-elevation myocardial infarction 10 years before his current admission in our hospital after a new episode of VT (Figure 1A). Because of hypotension and poor peripheral perfusion, the patient underwent a synchronized DC shock cardioversion. His left ventricle was remodeled with a current left ventricular (LV) ejection fraction of 30% and an inferior wall LV aneurysm. The ECG after cardioversion (Figure 1B) shows sinus bradycardia (the patient was chronically on amiodarone and betablockers), abnormal Q waves in leads II, III, and aVF, prolonged QT interval with prominent U waves (amiodarone), and an increased QRS voltage in lead V5 (35 mm).

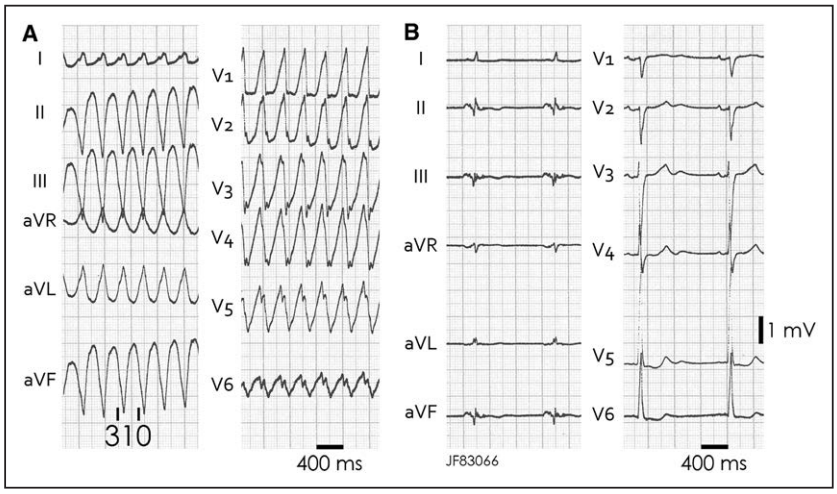


**Figure 1. 12-lead ECG during VT and sinus rhythm.**
**A,** ECG of the broad QRS VT documented at admission. **B,** ECG in sinus rhythm after electric cardioversion. The high-pass filter was set at 0.05 Hz and the low-pass at 200 Hz. The morphology of the VT suggests an exit point at the left ventricular basal inferoposterior region.

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Is there anything unusual in the ECG during sinus rhythm (Panel B)? We offer you 5 possibilities:

1. The voltage in the extremity leads is in the low range, in contrast to the chest leads, which suggests a large pericardial effusion.
2. Late potentials are mainly recorded in the inferior leads.
3. The P wave merges with the Q wave in the inferior leads, suggesting the presence of an accessory pathway.
4. It is not sinus bradycardia but 2:1 2nd-degree A-V block.
5. None of the above.

Please turn the page to read the diagnosis.





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## RESPONSE TO ECG CHALLENGE

The correct answer is that late potentials are mainly recorded in the inferior leads. Figure 2 shows the ECG during sinus rhythm recorded at 100 mm/s paper speed with a sensitivity of 20 mm/mV. We appreciate:

1. Late potentials (arrows) recorded after the end of the QRS complex, visible in the inferior leads but also present with this sensitivity in aVR and aVL
2. Fragmentation of the terminal forces of the QRS in the inferior leads, in V1 and V2

We asked the same question to an audience of ≈300 cardiologists during an interactive voting session on ECG interpretation. The replies were:

1. 11%: The voltage in the extremity leads is in the low range, in contrast to the chest leads, which suggests a large pericardial effusion.
2. 20%: Late potentials are mainly recorded in the inferior leads.
3. 21%: The P wave merges with the Q wave in the inferior leads, suggesting the presence of an accessory pathway.
4. 37%: It is not sinus bradycardia but 2:1 2nd-degree A-V block.
5. 11%: None of the above.

Postmyocardial infarction late systolic potentials usually have small amplitudes and are seldom registered on the conventional surface 12-lead ECG. Our case is unique because late systolic LV potentials within the scar during sinus rhythm were of sufficient amplitude as to be visible on the surface ECG.

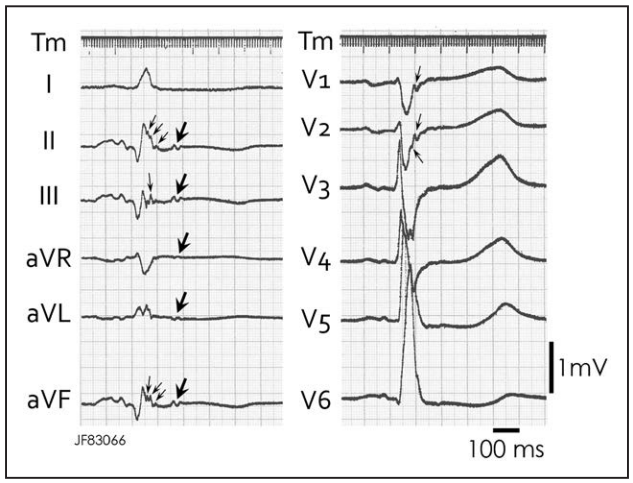


**Figure 2. 12-lead ECG during sinus rhythm at 100 mm/s paper speed and with a sensitivity of 20 mm/mV.** The high-pass filter was set at 0.05 Hz and the low-pass at 200 Hz. The thick arrows point to the late ventricular potentials recorded in several leads. Also note the fragmentation of the terminal forces of the QRS in leads II, III, aVF, V1, and V2 (small arrows).

A fragmented QRS complex is a marker of myocardial scarring and impaired intraventricular impulse propagation. It is controversial if a fragmented QRS may indicate an increased risk for cardiac arrhythmic events in patients with ischemic or nonischemic systolic LV dysfunction.[1] The extreme manifestation of scarring and delayed ventricular activation is the recording of late systolic potentials during the ST segment. This finding can be recognized in the surface ECGs of patients with arrhythmogenic right ventricular cardiomyopathy as a post-QRS deflection (epsilon wave) registered after the J point. In arrhythmogenic right ventricular cardiomyopathy, the areas of ventricular myocardium generating late systolic activation are relatively close to the chest electrodes recording V1, V2, and V3. In these patients, epsilon waves can also be recorded in right precordial leads.[2]

In patients with postmyocardial infarction scars, particularly in those developing VT, we can frequently register discrete post-QRS systolic potentials during sinus rhythm during endocardial or epicardial catheter electrode mapping.[3] These post-QRS systolic potentials in patients with ischemic scars are only exceptionally reflected on the surface ECG. Our case is unique because the surface ECG depicted late systolic ventricular potentials within the ST segment (Figures 1B and 2). This late systolic activity on the ECG correlated with endocardial post-QRS delayed LV activation within the inferior infarction scar, as recorded during catheter-electrode mapping (Figure 3). We are unaware of any similar finding previously reported in patients with an ischemic scar. We have mentioned the filtering settings of our ECG recordings because a lower low-pass value (eg, 40 Hz) could have eliminated the tiny deflections observed during the ST segment in our case.

## DISCLOSURES

None.

## AFFILIATIONS

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## FOOTNOTES

*Circulation* is available at http://circ.ahajournals.org.

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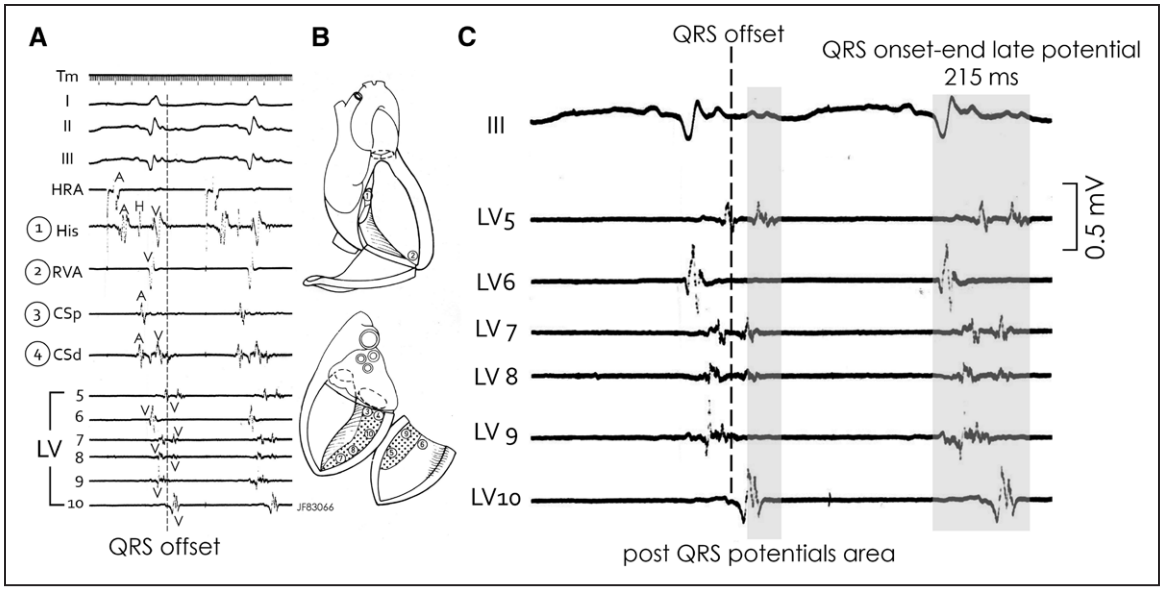


**Figure 3. Catheter-electrode endocardial ventricular mapping.**
**A,** Endocardial ventricular mapping during right atrial pacing; vertical line indicates the end of the QRS complex. **B,** Numbers indicate the points at which the various intracardiac recordings have been obtained. **C,** enlarged view displaying lead III of the surface ECG and 6 bipolar LV recordings at various sites, as indicated in **B**. Note that we were able to register endocardial post-QRS potentials within the area of late potentials on the surface ECG. Also note the presence of systolic electric activity within a window of 215 ms, from the onset of the QRS to the offset of the late ventricular potentials. CSd indicates coronary sinus distal; CSp, coronary sinus proximal; HRA, high right atrium; LV, left ventricle; and RVA, right ventricular apex.

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