R-wave double counting secondary to antiarrhythmics and ablation leads to unnecessary shock

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Introduction
A 61-year-old man with recurrent ventricular tachycardia (VT) and dual-chamber implantable cardiac defibrillator (ICD) was evaluated following a shock and syncope. ICD interrogation revealed monomorphic VT misclassified as ventricular fibrillation (VF) secondary to R-wave double counting. Increasing postventricular sense blanking period and discontinuing class I antiarrhythmic medication prevented R-wave double counting.

Case report
A 61-year-old man with a Medtronic (Medtronic 6947 Sprint Quattro secure lead and Medtronic Evera XT DR) dual-chamber ICD presented with syncope and an ICD shock. He was working at home when he had palpitations and subsequently received a shock followed by syncope, which resulted in dental trauma. Physical examination was largely negative other than the facial trauma.

He had a 15-year history of nonischemic cardiomyopathy and heart failure with reduced ejection fraction (ejection fraction 22%) NYHA class 3, ACC/AHA stage C. He was on guideline-directed medical therapy and amiodarone for frequent episodes of nonsustained VT. A VT ablation of the left ventricular inferoseptum was performed at an outside facility following an episode of sustained VT 4 weeks prior, and amiodarone was switched to quinidine.

Prior to interrogation of the device, the considerations were as follows: (1) appropriate shock secondary to ventricular arrhythmia for programmed therapy within the appropriate zone; (2) inappropriate shock owing to atrial arrhythmia or nonphysiologic signal; and (3) unnecessary therapy owing to misclassification.

Cardiac device interrogation revealed slow VT with a cycle length of 470 ms; it was misclassified in the VF zone owing to R-wave double counting, leading to an unnecessary shock. The total duration of the recorded intracardiac electrogram during VT episode before quinidine and the ablation was less than 100 ms (Figure 1). However, during the current episode, while the patient was on quinidine the electrogram duration increased to 120–130 ms, exceeding the postventricular blanking period (Figure 2). The device setting at baseline was monitor-only zone commencing at 450 ms, VT treatment zone at cycle length of 410 ms, and VF zone detection at 290 ms. The ICD was reprogrammed to increase the ventricular blanking period postventricular sensing from 120 to 130 ms. During overnight monitoring, multiple slower VT episodes in the monitor zone were documented, and therefore the monitor zone was changed to VT 1 zone with antitachycardia pacing (ATP). The cycle length of detection was changed to 500 ms.

KEY TEACHING POINTS
- R-wave double counting can be identified by a classic short-cycle railroad-track pattern usually slightly greater than the postventricular blanking period and frequently causes misclassification of ventricular tachycardia as ventricular fibrillation.
- During tachyarrhythmia, class I antiarrhythmics with native disease can cause use dependent conduction delay, leading to R-wave double counting.
- Implantable cardiac defibrillator programming changes like vector change, true bipolar setting, reducing sensitivity, increasing postventricular blanking period, and delay sensitivity decay can be applied to circumvent R-wave double counting, although caution is needed to prevent undersensing of ventricular tachyarrhythmia.

KEYWORDS Antiarrhythmics; Double counting; R wave; Unnecessary shocks; Ventricular blanking

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Owing to recent clinical changes, abnormal electrogram noted on the septum during prior ablation, and multiple VTs, positron emission tomography was performed to rule out cardiac sarcoidosis. The patient continued to have recurrent VT with appropriate ATP therapy for slow VT. Acceleration of the cycle length to the next zone led to a recurrent shock. He subsequently underwent combined epicardial and endocardial ablation and class I antiarrhythmic was discontinued to prevent use dependency–related conduction slowing during VT.

**Discussion**

Inappropriate detection of tachyarrhythmia by an ICD can be mainly attributed to overlapping nonphysiological or physiological nonarrhythmic signals that are detected as arrhythmia. Nonphysiological signals are due to either electromagnetic interference or lead integrity issues. Physiological signals may be intracardiac (usually T waves) or extracardiac myopotentials.

Ventricular oversensing of physiologic intracardiac signals resulting in 2 detected ventricular electrograms for each cardiac cycle, leading to inappropriate detection and therapy of VT or VF, have been reported. While T-wave oversensing is not infrequent, R-wave double counting is extremely rare. R-wave double counting occurs if the duration of the sensed electrogram exceeds the short ventricular blanking (postventricular blanking) period in ICDs. The delayed component of the R wave soon after the ventricular blanking period corresponds to an interval in the tachyarrhythmia zone. The duration of the device-detected interval

![Figure 1](image1.png)

**Figure 1** Intracardiac electrogram during sinus rhythm and ventricular tachycardia before any ablation and class I antiarrhythmic initiation. The initial far-field electrogram is sensed and the second near-field component is within the blanking interval during the ventricular tachycardia.

![Figure 2](image2.png)

**Figure 2** Double counting of the R wave. After inferoseptal ablation and initiation of quinidine leading to misclassification of ventricular tachycardia into ventricular fibrillation zone owing to temporal separation of far-field/near-field electrograms owing to increased conduction delay.
that begins with the second event approximates the difference between the spontaneous ventricular cycle length and the ICD’s blanking period. This produces a characteristic railroad-track pattern on a plot of stored ventricular intervals. Routinely, the device delivers ATP while charging. However, this programmable parameter is limited to ventricular arrhythmia of at least 200 ms. Hence, whenever R-wave double counting occurs within 200 ms, ATP therapies are not delivered during charging.

R-wave double counting often results from local ventricular conduction delay within the recording sphere of the sensing electrode. As most leads are placed in the right ventricular (RV) apical septum, septal conduction delays may lead to double counting. In supraventricular conducted rhythm, ICDs with short ventricular blanking periods ≤120 ms (Sorin, Biotronik, Medtronic) that are connected to integrated bipolar leads have higher probability of double counting. R-wave double counting may be precipitated by reversible conduction block caused by hyperkalemia or sodium channel–blocking antiarrhythmic drugs. Ventricular oversensing is more likely to occur in integrated than dedicated bipolar leads, although in our patient it was a true bipolar sensing (RV tip to RV ring).

In this patient, there can be multiple causes of R-wave oversensing. This patient was on quinidine, which is a class I antiarrhythmic that primarily works by blocking the fast inward sodium current. The characteristic of use dependency, where the antiarrhythmic drug binds the sodium channel with greater affinity at higher heart rates, further delays the conduction. Owing to the long elimination half-life of amiodarone, its residual effect along with quinidine may have a cumulative effect on the sodium channel, further prolonging QRS duration. The native disease process affecting the interventricular septum may be a contributing factor as well. Additional slowing of conduction secondary to ablated septal myocardium perhaps played a role.

The primary intervention to reduce R-wave double counting is to increase the ventricular blanking period. Prolongation of the ventricular blanking period from the nominal value or delaying the sensitivity decay (only possible in the St. Jude ICD, currently Abbott) corrects ventricular double counting in the majority of the cases and must be proposed as the first step. A common concern with this strategy is true VF undersensing when the blanking period is overextended. Similarly, decreasing the programmed ventricular sensitivity may resolve the problem in a certain number of cases, but this option requires that reliable sensing of VF will occur by means of defibrillation threshold testing at the reduced level of sensitivity. In Medtronic ICDs, switching from true bipolar sensing to integrated bipolar sensing vector change may also be attempted. In general, integrated bipolar programming may increase double counting owing to increased duration of the electrogram from the larger anode. However, if there is a favorable amplitude alteration of the delayed signal, this may be useful. When the blanking period cannot be prolonged adequately, a lead revision or addition of a separate pacing/sensing lead in a DF1 system (not feasible with DF4 system) may be proposed. Accordingly, in this patient the ICD was reprogrammed, and the blanking period was increased, which decreased his inappropriate detection and improved his quality of life. However, he continued to have recurrent VT despite reinitiation of amiodarone and a second ablation procedure performed at our institution. To the best of our knowledge, this is the first case report that illustrates the effects of native ventricular septal involvement, conduction slowing owing to antiarrhythmic medication effects, and possible role of prior ablation of the septum leading to R-wave double counting. After the second ablation procedure, the patient had a quiescent period of 5 weeks, after which the VT recurred and he underwent cardiac transplantation both for worsening heart failure and for recurrent VT. The cardiac autopsy of the native heart showed chronic lymphocytic myocarditis of the septum and secondary amyloidosis (Figure 3).

Conclusions
This case illustrates the effects of native septal involvement and conduction slowing owing to class IA medication leading to R-wave double counting. Recognition of this on cardiac device interrogation is critical in instituting necessary programming changes and clinical planning to improve clinical outcomes.

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