Ventricular tachycardia slower than the rate cut-off of a subcutaneous cardiac defibrillator sensed and successfully treated as a result of oversensing

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Introduction
The subcutaneous implantable cardioverter-defibrillator system (S-ICD) has been an important alternative to transvenous defibrillators for certain clinical situations, such as when there is a venous access problem or high risk of bacterial endocarditis. This device reliably detects ventricular tachyarrhythmias and effectively delivers shocks. However, oversensing is more common with the S-ICD compared with transvenous systems. We present a case of a patient in whom a change in the rhythm from sinus rhythm to slow ventricular tachycardia (VT) resulted in reduced R-wave sensing and T- and P-wave oversensing.

Case presentation
A 47-year-old man with acquired immunodeficiency syndrome, diabetes, and nonischemic cardiomyopathy underwent implantation of a primary-prevention S-ICD (Cameron Health/Boston Scientific, San Clemente, CA) using standard technique. His left ventricular ejection fraction was 10%, and his NYHA functional class was II. Previously, a transvenous ICD system had been explanted because of lead-related endocarditis.

Before S-ICD implant, the patient passed electrocardiographic (ECG) prescreening in all S-ICD sensing vectors. Surface 12-lead ECG at screening showed sinus rhythm with QRS amplitude of 0.3–1.5 mV in limb leads and 0.3–2.2 mV in precordial leads (Figure 1A). The S-ICD’s automated vector-selection algorithm chose the secondary vector for sensing. The corresponding screening ECG lead had a 2.5-mV QRS amplitude in either supine (Figure 1B) or standing (Figure 1C) positions. The conditional and shock zones were programmed to 190 beats per minute (bpm) and 220 bpm, respectively.

Eight months later, the patient presented in slow VT at approximately 110 bpm (Figure 2) after receiving 3 ICD shocks. The VT had a right bundle branch block–like morphology with absolute base-peak QRS amplitudes of 0.8–3 mV in limb leads and 0.5–1.5 mV in precordial leads, generally similar to QRS amplitudes in sinus rhythm (Figure 2). In contrast, ICD interrogation (Figure 2) showed that subcutaneous electrograms (EGMs) had variable and markedly lower amplitudes in VT (0.4–0.8 mV) than in sinus rhythm (2–2.5 mV). The S-ICD oversensed P and T waves during VT and thus incorrectly calculated the ventricular rate to be in the shock zone, resulting in shocks for VT slower than the Conditional Zone rate threshold.

Chest radiographs showed no change in generator or lead position compared with postoperative radiographs (Figure 3). However, in comparison with the recommended placement of the S-ICD generator on the fascia over the latissimus dorsi muscle, the posteroanterior radiograph shows that the generator is positioned in the overlying subcutaneous tissue.

In sinus rhythm, S-ICD sensing was accurate in all 3 vectors; the sensing vector was changed empirically from secondary to primary. During supine bicycle exercise, the patient achieved a maximum sinus rate of 130 bpm without oversensing or change in QRS morphology compared with baseline. In 6 months of follow-up, the patient received no further ICD shocks.

Discussion
Accurate sensing of subcutaneous EGMs in VF without oversensing in organized rhythms is challenging; and the S-ICD includes procedures and technology to achieve this goal.

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similarities between the cutaneous ECG and subcutaneous EGM signals. The goal is to identify patients in whom the baseline QRS duration is too long for the S-ICD’s blanking period or the ratio of QRS amplitude to T-wave amplitude is insufficient to permit rejection of T waves by the S-ICD’s dynamic sensitivity.

However, ECG prescreening cannot identify changes in the QRS-T complex caused by intraventricular conduction delays that develop after implant or slow, well-tolerated, and self-terminating VT. Wilson et al reported a case in which sensing during sinus rhythm was reliable, but R-wave double counting occurred during VT slower than the programmed detection rate, resulting in shock delivery. Subsequently, the patient developed right bundle branch block during sinus rhythm. This change in the QRS-T complex resulted in T-wave oversensing in all S-ICD vectors, requiring explant of the S-ICD and replacement with a transvenous ICD. In our case, the root cause of the oversensing during slow VT was low-amplitude R waves that prevented dynamically adjusting sensitivity from rejecting T and P waves.

To the best of our knowledge, this is the first report of amplitude discrepancies between surface ECG leads and S-ICD EGMs caused by a change in cardiac rhythm. S-ICD EGMs had adequate amplitude in sinus rhythm but very low amplitude in slow VT, while all surface ECG leads had adequate absolute R-wave amplitudes during both sinus rhythm and VT. Standard 12-lead ECG leads may not correlate precisely with S-ICD sensing vectors; however, prescreening ECG leads and S-ICD EGMs in sinus rhythm had a close amplitude correlation for corresponding vectors.

We do not know if this same discrepancy would have occurred if the pulse generator had been implanted in the recommended tissue plane rather than in subcutaneous tissue. However, the difference in QRS-T vectors between EGMs recorded at the 2 positions would likely be small; and a more superficial generator should more closely replicate the surface ECG recording than a deeper one. In addition, sinus-rhythm EGMs had adequate amplitude both at baseline and on the stored EGM that recorded VT. The former

Figure 1 A: Twelve-lead electrocardiogram during sinus rhythm. Screening surface electrograms in lead II (secondary vector) during sinus rhythm at prescreening in B: supine and C: standing positions.
observation excludes the possibility that the location of the generator accounts for the observed, rhythm-related discrepancy between ECG and EGM amplitudes, and the latter observation excludes postural changes as its cause.

In summary, this case illustrates a previously reported limitation of S-ICD ECG prescreening: It can only reject QRS-T complexes prone to oversensing in the rhythm at the time of screening. To the best of our knowledge, we provide the first

**Figure 2** Twelve-lead electrocardiogram of the ventricular tachycardia (VT) episode. A: The absolute amplitude of the QRS in lead II is 2.3 mV. B: The subcutaneous implantable cardioverter-defibrillator’s stored subcutaneous electrogram of the VT episode in which T- and P-wave oversensing resulted in delivering inappropriate shock. The shock terminates the VT after a few beats and sinus rhythm with similar rate to the VT rate initiates. Note R-wave amplitude is low in VT (0.4–0.8 mV) and much larger in sinus rhythm after the shock (beginning at 61 sec, 2.5–3 mV).
evidence that changes in cardiac rhythm can result in diminutive subcutaneous R waves on the secondary S-ICD sensing vector without low-amplitude R waves on any standard surface ECG lead. Further investigation is required to determine the conditions that permit rhythm-dependent discrepancies between the amplitude of surface ECG R waves and S-ICD EGMs, including generator placement in the subcutaneous space.

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