Pitfalls of pacemaker detection of ventricular high-rate events

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Introduction
Modern pacemakers have enriched diagnostic features that can provide detailed information about arrhythmic events and, hence, impact clinical management. In contrast to ambulatory electrocardiographic monitors and event recorders, these pacemaker capabilities provide continuous as opposed to intermittent monitoring, and do not require symptom-based patient activation to capture sporadic arrhythmias. As such, the information stored about the presence or absence of arrhythmic episodes and their frequency, duration, time of onset, and recorded rates can be critical in establishing a diagnosis and monitoring therapy. However, diagnostic pacemaker features have their caveats and pitfalls. Knowledge of these important limitations can help inform pacemaker troubleshooting.

Case report
A 66-year-old woman was transferred to our center after a resuscitated cardiac arrest. The presenting rhythm was not documented. Her evaluation revealed severe triple-vessel nonrevascularizable coronary artery disease, with a left ventricular ejection fraction of 30%. A secondary-prevention implantable cardioverter-defibrillator (ICD) was strongly recommended but was declined by the patient despite a clear understanding of the high risk for sudden death and the confirmed capacity to make reasoned decisions. During her hospitalization, recurrent bouts of polymorphic ventricular tachycardia (VT) were documented, along with sinus bradycardia and paroxysmal atrial fibrillation with postconversion pauses that limited beta-blocker therapy. The patient agreed to dual-chamber pacemaker implantation (Epyra DR, Biotronik, Berlin, Germany). The evening after the procedure, she experienced a nocturnal 20-second run of polymorphic VT (cycle length 200–240 ms) recorded by telemetry (Figure 1). Pacemaker interrogation revealed normally functioning leads with R-wave sensing at 18.9 mV. The ventricular high-rate (VHR) detection feature was set to record events triggered by 8 beats at 180 beats per minute (bpm), yet no such episode was logged. Why did the pacemaker fail to detect a VHR?

Discussion
The differential diagnosis entertained for pacemaker failure to detect this electrocardiographically documented prolonged VHR event included artefact simulating VT, true undersensing of polymorphic VT, and functional undersensing. Artefact, such as from body movement or intermittent skin-electrode contact, was essentially ruled out by the clinical context, multiple prior bouts of nonsustained VT, and absence of normal or paced QRS complexes during the episode.1 Undersensing of a low-amplitude polymorphic

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Figure 1 Polymorphic ventricular tachycardia (VT) recorded by telemetry. Shown is a telemetry lead II recording of polymorphic VT post pacemaker implantation. The patient was in slow paroxysmal atrial fibrillation prior to the onset of VT, with occasional ventricular paced beats. The tachycardia lasted 20 seconds, had a cycle length that predominantly ranged between 200 and 240 ms, and terminated spontaneously.

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(Heart Rhythm Case Reports 2018;4:163–165)
VT was considered but deemed unlikely. Contrary to the fixed sensitivity of a traditional pacemaker, the sensing algorithm of Epyra devices is comparable to an ICD, with a threshold start and rapid decay to a high sensitivity value that is nominally set to 2 mV (Figure 2). These settings are less sensitive than standard ICDs (eg, nominally set to 0.3–0.6 mV), rendering the potential for undersensing of VHR events more likely with pacemakers than defibrillators. However, it is doubtful, albeit not beyond the realm of possibility, that no sequence of 8 consecutive beats during a prolonged episode of relatively organized polymorphic VT exceeded 2 mV given the intrinsic R wave value of 18.9 mV.

Functional undersensing was therefore thought to represent a more plausible hypothesis. One possible means by which a device may functionally undersense a VHR event is by noise behavior algorithms. Upon detection of exceedingly rapid signals, noise algorithms trigger noise periods during which no event can be recorded. However, for the recent Biotronik pacemakers (ie, Evia and subsequent models), the noise window is triggered when events are faster than 51 ms. In preceding models, the noise window was activated by cycle lengths shorter than 125 ms. The cycle length of the polymorphic VT in our patient (ie, 200–240 ms) was remote enough from the threshold value to exclude triggering of the noise algorithm as a credible explanation for functional undersensing.

Finally, in further exploring the possibility of functional undersensing, definitions for VHR event and ventricular refractory period were reviewed. Typically, events that fall within a refractory period are used for binning but not for timing purposes. This allows arrhythmias such as atrial fibrillation to not be tracked, yet to qualify as refractory (ie, “Ar”) events that can trigger mode switch algorithms. In Biotronik pacemakers, the ventricular refractory period is nominally programmed at 250 ms. However, as illustrated in Figure 3, contrary to Medtronic, Boston Scientific, and Sorin pacemakers, events that fall within this refractory period are not used to increase the VHR counter. As a result, VTs faster

**Figure 2** Dynamic sensitivity adjustment. After each paced or sensed ventricular beat, automatic sensitivity control (ASC) initiates a detection hold-off period (121 ms for sensed beats; 200 ms for paced beats). For sensed ventricular beats, the subsequent sensing threshold is first set to 50% of the measured peak amplitude within the first 80 ms of this interval. For paced ventricular beats, the subsequent sensing threshold is set to the nominal minimal value (ie, 2 mV for Epyra devices). After a step duration of 125 ms, the threshold is set to 25% of the peak amplitude but never below the minimum threshold of 2.0 mV. This functionality allows signals with varying and small amplitudes to be detected with a 1:4 signal-to-noise ratio.
than 250 ms (ie, 240 bpm) will not be recorded because sensed beats will fall within the ventricular refractory period. This explanation for functional undersensing was, therefore, retained. Upon recognition of this likely mechanism, the patient was contacted on several occasions to decrease the ventricular refractory period (ie, minimal programmable value of 200 ms) but failed to respect appointments. She previously attended her 1-month follow-up visit, at which time no VHR episode was recorded on a maximum tolerated dose of carvedilol (ie, 25 mg twice a day).

Importantly, this functional undersensing limitation is applicable to the gamut of Biotronik pacemaker models but not defibrillators. For Biotronik defibrillators, the ventricular refractory period is set to 200 ms and is nonprogrammable. Moreover, events that fall within the ventricular refractory period increase the counter. Although St. Jude pacemakers also ignore refractory events in considering VHR episodes, they employ a dynamic ventricular refractory period that decreases in duration as the heart rate increases. If a fixed sensitivity is programmed, the shortest ventricular refractory period attains 175 ms (ie, 342 bpm). If the sensitivity is programmed to “auto,” the shortest ventricular refractory period reaches 125 ms (ie, 480 bpm). Functional undersensing of VT owing to this mechanism is, therefore, unlikely with St. Jude pacemakers.

Little has been published about VHR events beyond their prognostic value in identifying patients at higher risk for tachy-brady syndrome and mortality in the setting of heart failure. Nevertheless, clinicians often rely on a search for VHR events in the diagnostic evaluation of symptomatic pacemaker recipients with palpitations, dizziness, or syncope in order to exclude tachyarrhythmias. In so doing, the caveats discussed herein should be considered, including the possibility of not detecting events slower than the VHR cutoff value, true undersensing, and functional undersensing if ventricular rates exceed 240 bpm in patients with Biotronik pacemakers.

Acknowledgments
The authors thank Ms José Girard from Biotronik, Inc. for her expert technical assistance.

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