A Pseudo-Pseudofusion Beat Preceding Onset of Ventricular Tachycardia in a Patient with an Implantable Cardioverter Defibrillator

HUSSAM ABUISSA, MD, FACC, FHRS
The Cardiac Center of Creighton University Medical Center, Omaha, NE

ABSTRACT: Implantable cardioverter defibrillators (ICDs) are commonly used for primary and secondary prevention in patients with cardiomyopathy. Although newer devices have advanced features to ensure better performance and safety profiles, unintended consequences are inevitable. This case highlights an adverse effect of prolonging the postventricular atrial refractory period following a premature ventricular contraction: precipitation of ventricular tachycardia. We discuss the phenomenon of functional undersensing and its potential impact on device function, as well as possible programming changes that might help eliminate it.

KEYWORDS: blanking, pseudo-pseudofusion beat, post-ventricular atrial refractory period, ventricular tachycardia.

Case Presentation
This is a 74-year-old man with ischemic cardiomyopathy (left ventricular ejection fraction, 0.25) status postcoronary artery bypass graft surgery and percutaneous coronary intervention, New York Heart Association class III heart failure, and paroxysmal atrial fibrillation. He underwent dual chamber implantable cardioverter defibrillator (ICD) implantation in 1999 for inducible ventricular tachycardia with generator exchanges in 2003 and 2009. His most recent generator was a Boston Scientific E110 Teligen (Natick, MA). Programmed parameters were as follows: mode: DDDR; lower rate: 60 beats per min (bpm); upper rate: 115 bpm; paced and sensed atrio-ventricular (AV) delay: 180-300 ms; search AV delay: 350 ms; postventricular atrial refractory period (PVARP): 250 ms; ventricular refractory period (VRP): 250 ms; PVARP after premature ventricular contraction (PVC): 400 ms; ventricular blank after atrial pace: 65 ms. The ventricular tachycardia (VT) zones were set as follows: VT-1: 170 bpm, no therapies; VT: 200 bpm, antitachycardia pacing (ATP)+shock; ventricular fibrillation (VF): 220 bpm, shock only.

He was on amiodarone (200 mg daily) for atrial fibrillation. He presented to the local emergency department with complaints of worsening dyspnea on exertion and orthopnea. A 12-lead electrocardiogram revealed that he was in VT at a rate of 144 bpm, below the VT-1 detection rate on the ICD. He was given a 150-mg intravenous bolus of amiodarone, which failed to control his arrhythmia, and he was subsequently shocked externally with 150 Joules of biphasic energy that resulted in conversion into normal sinus rhythm followed by atrial pacing and ventricular sensing.

Device interrogation revealed normal lead and device parameters with no VT detections. We subsequently changed the VT zones as follows: VT-1: 140 bpm, no therapies; VT: 170 bpm, ATP+shock; ventricular fibrillation (VF): 220 bpm, shock only.

He continued to do well but had another episode of slow VT while hospitalized. A telemetry rhythm strip was obtained at the onset of VT (Figure 1).

Why didn’t the atrial pacing spike following the PVC capture and was rather followed by a native ventricular beat? Was this beat sensed by the device? And, if so, why...
was it followed by a ventricular-paced beat after an interval shorter than the lower rate programmed on the device?

**Interpretation**

The failure to respond to a physiologically appropriate signal occurring during the alert period of the timing cycle is termed undersensing.\(^1\) It can reflect a true pacing system malfunction, a functional limitation of the system due to unique algorithms in a given device, or a response to the programmed settings of the device. Functional undersensing typically occurs when an intrinsic event falls within a blanking or refractory period and is not sensed as a function of the pacemaker programming.\(^1\) A frequent source of confusion is the appearance of atrial undersensing caused by the occurrence of a P wave during the PVARP.\(^2\) A careful review of the rhythm strip (Figure 2) revealed a PVC following the last atrial-paced/intrinsic ventricular beat. This resulted in prolongation of the PVARP after the PVC (programmed to 400 ms on the device) so that the next P wave fell in the refractory period and was, therefore, not tracked. The device then delivered an atrial pacing stimulus, which did not capture the atrium because it was still refractory from the previous beat. However, the patient happened to have an intrinsic ventricular beat (which was technically a pseudo-pseudofusion beat) after the atrial pacing spike, which was also “ignored” by the device because it fell in the ventricular blanking period and was followed by a ventricular paced beat at a short coupling interval, resulting in ventricular reentry and initiation of VT.

Device interrogation was performed (Figure 3) and confirmed our initial suspicion raised by the telemetry rhythm strip. As the device telemetry shows, there was evidence of atrial pacing at the sensor-determined rate followed by ventricular sensing for the first two beats. These were followed by a PVC, which resulted in extension of the refractory period (as indicated by the VVP mark) and made the next A (which was most likely a sinus beat rather than a retrograde A) fall in the refractory period. Atrial pacing at the sensor-determined rate then resumed after the atrial escape interval following the PVC had lapsed. However, just after the atrial pacing spike, an intrinsic ventricular event (conducted sinus beat) was sensed. Because it occurred in the 65-ms ventricular blanking period following atrial pacing (as indicated by [VS]), it was considered “noise,” and the device then delivered a ventricular pacing stimulus after the search AV delay had lapsed. Therefore, this paced ventricular stimulus occurred after a short coupling interval and precipitated VT. It must be noted that only the first 20 ms of the 65-ms blanking period are considered the “hard” blanking window, and the remaining 40 ms actually represent the “noise” window, which explains the notation of the event on the marker channel.

Because no therapies were programmed in the monitoring zone, we attempted to terminate the tachycardia by ATP through the device. However, multiple attempts using both burst and ramp therapies failed to do so, and the patient was eventually shocked externally with 200 Joules of biphasic energy.

We subsequently re-interrogated the device. There was no evidence of retrograde conduction with ventricular pacing at different rates, which confirmed our suspicion that the undersensed A in our patient was in fact a sinus P wave followed by intrinsic ventricular conduction. Therefore, we turned off the “PVARP after PVC” feature on the device. We also decreased the detection rate in the VT-1 zone to 130 bpm and added ATP therapy in that zone. The patient had no recurrent episodes of sustained VT during the rest of his hospital stay.

**Commentary**

Current implantable devices have become very sophisticated and are loaded with a variety of algorithms designed to improve their overall performance. Extending the PVARP after a PVC, a nominal setting on this particular device, is a feature that makes the retrograde P wave fall in the atrial refractory period and, as such, prevents it from being tracked and precipitating pacemaker-mediated tachycardia. Our case highlights the potential adverse effects of such an algorithm, especially in patients with no evidence of retrograde conduction. It also underscores the importance of thoroughly evaluating patients with apparent device malfunction shown on 12-lead electrocardiogram or telemetry. Although a careful review of the telemetry rhythm strips was helpful in ruling out device malfunction and discerning the mechanism of tachycardia initiation in our patient, adding a monitor zone was of paramount importance in confirming our initial suspicion with device telemetry.

Another potential undesirable effect of extending the PVARP after a PVC is repetitive non-re-entrant AV
Figure 2: A careful review of the telemetry rhythm strip revealed that the patient exhibited atrial pacing at the sensor-determined rate (—) for the first two beats with intrinsic ventricular conduction. These were followed by a PVC (arrowhead) that resulted in extension of the refractory period to the programmed value of 400 ms (short double arrow) and made the next A (black arrow) fall in the refractory period. Atrial pacing at the sensor-determined rate (gray arrow) then resumed after the atrial escape interval (long double arrow) following the PVC had lapsed. However, just after the atrial pacing spike, an intrinsic ventricular event (star) was sensed. Because this occurred in the 65-ms ventricular blanking period following atrial pacing (small rectangle), it was considered “noise,” and the device then delivered a ventricular pacing stimulus after the search AV delay of 350 ms had lapsed (large rectangle). This stimulus occurred after a short coupling interval and precipitated VT.

Figure 3: Device interrogation tracing at tachycardia onset. Atrial electrograms, ventricular near-field electrograms, ventricular far-field (shock) electrograms, and the marker channel are shown. AP-Sr: atrial pace-sensor rate; VS-Hy: ventricular sense-in hysteresis offset; PVC: premature ventricular contraction; PVP: PVARP after PVC; (AS): atrial sense during total atrial refractory period; [VS]: ventricular sensing during blanking; VP-Sr: ventricular pace-sensor rate; VT-1: ventricular tachycardia 1 zone sense.
synchrony that also results from not “sensing” the retrograde P wave and causes atrial pacing with no capture and subsequent ventricular pacing.3 This is essentially what occurred in our patient except for the fact that it was a sinus beat that was undersensed and resulted in intrinsic ventricular conduction, thereby setting the medium for ventricular re-entry and tachycardia after ventricular pacing.

Physicians and device technicians should be encouraged to tailor the device settings to their patients’ needs and be aware of the potential unintended effects of the available algorithms.

References: