DEVICE THERAPY

COMPLEX CASE STUDY

Post Implantable Cardioverter-Defibrillator Syncope: What is the Mechanism?

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ABSTRACT. This manuscript is a case report of defibrillator shock-induced atrial crosstalk inhibition of ventricular pacing. The patient involved had severe ischemic cardiomyopathy and was presented with ventricular arrhythmia storm. Shortly after defibrillation during this multishock episode, he would experience dizziness followed by syncope. Initially the mechanism was not known, but during his inpatient stay it was determined to be bradycardia-mediated syncope secondary to residual shock energy, resulting in atrial crosstalk and pacing inhibition in the ventricular channel. We detail the pathophysiology behind this phenomenon and discuss the possible programming changes to eliminate it.

KEYWORDS. implantable cardioverter-defibrillator, ventricular tachycardia, atrioventricular, smart blanking.

Case presentation

A 52-year-old man with non-ischemic cardiomyopathy and complete heart block underwent implant of a dual-chamber biventricular implantable cardioverter-defibrillator (ICD). He presented to the hospital with decompensated heart failure and electrical storm. Despite optimal medical therapy and dual antiarrhythmic drug therapy with amiodarone and mexilitene, he continued to have incessant ventricular tachycardia (VT) requiring ICD shocks. The baseline device settings are shown in Figure 1. He underwent several VT ablations, but he continued to experience frequent VT. He eventually received a left ventricular assist device as a bridge to heart transplant.

On one such occasion, telemetry of the patient showed sustained VT (Figure 2), which required multiple rounds of antitachycardia pacing and an ICD shock. Post ICD shock he was found to have loss of ventricular pacing resulting in a ventricular escape beat at 25 bpm and syncope (Figure 3). Approximately 3.5 min later, resumption of ventricular pacing was noted. Interrogation of his Cognis N118 ICD (Boston Scientific, St. Paul, MN) showed lead impedances, pacing thresholds, and sensing were all stable and unchanged from prior interrogations. VT detections and ICD therapies were all appropriate. He had an integrated bipolar Boston Scientific model 0185 ICD lead. His LV lead had previously been turned off due to suspected proarrhythmic effects. Why was ventricular pacing inhibited?

Comment

Upon further review of the patient’s ICD parameters and recordings, the cause for loss of ventricular capture was better understood. During “normal” dual-chamber pacing, the patient’s atrioventricular (AV) blanking periods were programmed to “Smart Blanking” settings. “Smart Blanking” is a feature used to allow a wider sensing window for arrhythmias (minimizing undersensing) while maintaining prevention of crosstalk (oversensing). “Smart Blanking” combines a shorter refractory period of 37.5 ms followed by paced events and 15 ms following sensed events (compared with a usual range of 45–125 ms) along with an adjustable sensitivity threshold during the blanking period as displayed in Figure 4. The design of this combination is to minimize the risk of blanking in chamber arrhythmic
events while maintaining the ability to ignore low-amplitude cross-chamber artifact.

However, during the post-shock period the device automatically extends the “RV-Blank after A-Pace” time out to 85 ms to prevent crosstalk. After the programmed post-shock pacing duration expires (a nominal value of 30 s) and “normal” dual-chamber pacing resumes, “Smart Blanking” shortens the “RV-Blank after A-Pace” from 85 ms back to 37.5 ms.

In this patient’s case, crosstalk between the atrial and ventricular leads was ongoing well after the post-shock pacing duration of 30 s. Initially post shock, the crosstalk was occurring approximately every 45 ms, falling within the 85 ms post-shock blanking period (Figure 2). However, once “post-shock” pacing transitioned back to “normal” pacing after 30 s, the crosstalk then fell outside the normal blanking period of 37.5 ms, resulting in inhibition of ventricular pacing (Figure 3).

Initially unresolved questions included: 1) Why did the patient have crosstalk now when he has not had it before? 2) Why only post ICD shock? The patient had been in the hospital with recurrent VT requiring multiple ICD therapies. Post defibrillation, residual energy is contained within the defibrillator lead with the amount of retained energy proportional to that delivered by the device. The movement of this energy into the myocardium can increase the likelihood of AV crosstalk that will eventually resolve with complete dissipation of the energy in the defibrillator lead.

To prevent this from occurring again, there were a number of reprogramming approaches that could be taken. One would be to program “RV-Blank after A-Pace” to be fixed at its maximum of 85 ms at all times. This way the crosstalk would fall within the blanking period and prevent any inappropriate inhibition of pacing. However, this could result in underdetection of slow VT, since ventricular complexes could fall within the blanking period and would not be counted towards VT detection.

Another option is to reprogram to VVI pacing. This would eliminate cross-chamber blanking altogether, and avoid post-shock inhibition of pacing. However, in this patient with advanced heart failure, loss of AV synchrony may have resulted in worsening heart failure symptoms.

The final option is to extend the patient’s post-shock pacing duration beyond the nominal setting of 30 s. By extending the post-shock pacing time, the blanking period remains at 85 ms during the period where...
Figure 2: (a) Normal dual-chamber pacing without crosstalk is seen followed by initiation of ventricular tachycardia. (b) Ventricular tachycardia is terminated with a 31-J shock after which post-shock pacing begins. During post-shock pacing, crosstalk exists but falls within blanking as indicated by the dash on the ventricular channel in the enlargement.

Figure 3: Telemetry shows the transition from post-shock dual-chamber pacing to normal pacing at 30 s where crosstalk still exists resulting in inhibition of ventricular pacing. At 3 min 36 s post shock, atrioventricular crosstalk ends and ventricular pacing resumes.
Crosstalk may occur. This allows the residual post-shock energy to dissipate during post-shock pacing and eliminates the inhibitory pacing effect of AV crosstalk. “Smart Blanking” could then continue to be used during normal pacing for optimum detection of slow VT and prevent delay in VT detection.

Case follow-up
Following this adjustment, the patient did not experience any further episodes of post-shock crosstalk-mediated inhibition of ventricular pacing and was successfully transitioned to LVAD support.