CASE REPORT

Two Cases of Ventricular Proarrhythmia from Automatic Threshold Measurement Features in a CRTD and an ICD

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The present case report presents two cases in which automatic threshold measurement features in an implantable cardiac defibrillator and in a CRTD act proarrhythmic by inducing polymorphic ventricular arrhythmia. By programming these features off the problem was solved. The mechanisms seem to be different from previously reported proarrhythmic interactions from such device features.

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ventricular tachycardia/fibrillation; biventricular pacing/defibrillation

CASE 1

A 61-year-old male patient was admitted from the pacing clinic after suffering several syncopal episodes. Device interrogation revealed several episodes of ventricular fibrillation (VF). His cardiovascular record was significant for the following: He had a mechanical mitral valve and a mechanical aortic valve (rheumatic valve disease). Severe left ventricular dysfunction (ejection fraction 25%, left ventricular diameter 9 cm, left atrial diameter 8 cm). No coronary artery disease. He had a cardiac resynchronization and defibrillator device (CRTD) system (Medtronic Consulta CRTD, single zone VF from 200 bpm, VF detection 18/24). He previously had atrioventricular (AV) nodal ablation for permanent atrial fibrillation (afib) with rapid ventricular response. He was diagnosed with prostate cancer with pelvic metastases. Despite the above, his cardiovascular status was quite stable and his functional capacity stable estimated NYHA class IIIA.

A new coronary angiography was unremarkable. He was in a well-compensated state at admittance. His electrolyte status was normal. He had no escape rhythm after the previous AV nodal ablation.

During hospital stay, he suffered three episodes of VF (one representative episode shown in Fig. 1, all episodes were similar). Interrogation of the CRTD device revealed that all VF episodes were initiated during capture management evaluation of threshold in either atrium (during which biventricular pacing in the Medtronic Consulta CRTD device is temporarily suspended and only right ventricular (RV) pacing performed¹) or right ventricle. Several nonsustained polymorphic ventricular tachycardia (PMVT) episodes in the device memory were reviewed and were initiated similarly. During right ventricular pacing (Vp) late coupled ventricular extrasystoles (VES) was observed. These were not sensed by the CRTD (Figs. 1 and 2) due to late occurrence coincident with the next scheduled paced beat, which was delivered after surface ECG initiation of the VES leading to varying degrees of fusion beats (Figs. 1 and 2). This elicited episodes of VF (Figs. 1 and 2). The nonsensed ventricular complexes were only evident because the patient suffered episodes during hospital admittance, when his heart rhythm was monitored (Fig. 2). As seen in Figures 1 and 2 these complexes were not apparent from the interrogation of the episodes from the device

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Figure 1. Rhythm strip from representative episode. Beginning and end of episode is shown. From above: bipolar atrial channel, bipolar ventricular channel, marker channel. Vp = right ventricular pacing; Bv = biventricular pacing. Ap = Atrial pacing. Ar = refractory atrial event. The sequence of events signifies atrial capture management (see text).

(marker channel). Furthermore, on one occasion VF was induced during manual RV threshold determination. The events on the marker channel in Figure 1 indicate that atrial capture management is ongoing before the VF. The atrial pacing stimulus occurring earlier than expected is the atrial test stimulus that does not capture as evidenced by the atrial refractory event (Ar) following and occurring "on time" as judged from the previous atrial sensed events. This characterizes the atrial capture management by the method of "resetting of the atrial chamber" as opposed to the method dependent on av conduction.¹ Furthermore, the Vp events during capture management indicate that during atrial capture management left ventricular (LV) stimulation is inactive. Capture management was programmed off. During follow-up on home monitoring during the next 9 months, no further episodes appeared.

CASE 2

A 61-year-old male was admitted after syncope due to PMVT spontaneously terminated with shock

aborted. Six years earlier, he received a single chamber ICD after cardiac arrest due to VF. Workup at the index episode revealed normal coronary arteries, normal left ventricular function with only slight dilatation of the left ventricle (6 cm) and slight mitral valve prolapse without insufficiency. Cardiac magnetic resonance imaging (MRI) was normal. No late potentials on signal averaged ECG. During the following 6 years, he suffered no episodes (without any medical treatment) until recently, where several sustained episodes of monomorphic ventricular tachycardia (MVT) were detected and therapy with antitachycardia pacing (ATP) and ICD shock were delivered by the device. Due to initiation of betablocker therapy, he developed symptomatic sinus bradycardia and he was upgraded to a dual chamber ICD (St. Jude Fortify Assura DR, programmed in DDDR 60/140, base rate/max sensor and tracking rate, AV delay 200/180 paced/sensed, VIP on with 100 ms, Tachy zones: VT1 from 176, 5 ATP programmed, FVT from 200, 2 ATP before shock programmed, VF from 240, detection 25 intervals in all three zones). A new workup was unchanged.

Figure 2. The corresponding telemetric monitoring during same episode as in Figure 1. Inset shows morphology of right ventricular paced event. Ventricular extrasystoli initiated coincident with initiation of capture management (and only RV pacing).

A representative episode as follows from Figure 3 shows a likely device related algorithm implicated in inducing the two similarly initiated episodes. Totally four similarly initiated episodes were observed. During rate responsive pacing, VES with a late coupling interval to the previous complex occurred simultaneously with the atrial pacing stimulus. Due to the cross-chamber blanking period on the ventricular channel, the VES was unsensed and the Vp stimulus after the completion of the AV interval falls in the relative refractory period of the repolarization phase from the VES. Since Autocapture was programmed active the noncapture of the Vp stimulus elicits a backup stimulus with high output (5 V, 0.5 ms according to the Autocapture feature). The combination of a slightly later stimulus and higher output overcomes the relative refractory period and during the vulnerable phase initiates a propagated response, which initiates an episode of nonsustained polymorphic VT. The longest of the episodes lead to syncope.

It is well described that unsensed events during the atrial pacing-induced cross-chamber ventricular blanking period can carry the risk of Vp during the vulnerable phase.^{2,3} However, in the present case this would probably not have initiated VT, if the autocapture feature had not been programmed active. It is the autocapture related high output backup stimulus that is most probably responsible for the proarrhythmic effect in the present case by overcoming the higher threshold during the relative refractory period of the unsensed event.

DISCUSSION

The two cases demonstrate proarrhythmic effects of two different automatic threshold testing features. In the first case, the right Vp during automatic threshold measurement in both atrial and right ventricle induces VES and initiates VF at several occasions to a degree approaching the definition of electrical storm. The VES occur

Figure 3. Episode from interrogation of representative episode leading to syncope. Vp: ventricular pacing, Ap = atrial pacing, VPP = V pace with backup stimulus due to loss of capture (LOC) from the first delivered impulse, SIR = sensor indicated rate.

sufficiently late, so that they are not sensed before the next scheduled pacing stimulus. Apparently, the biventricular pacing in this patient acts antiarrhythmic in contrast to right Vp.

Furthermore, the episodes were concentrated when the patient was in sinus rhythm. Only during this time, the atrial capture management algorithm would be active and thus increasing the probability of the phenomenon. At a first glance, it would have been easy to incorrectly assume that the episodes were related to right ventricular capture management only and turn off only this, while leaving the atrial counterpart active and not recognizing that it was the latter that was responsible for the episode. This case demonstrates an example of a device-programming feature that could have had fatal consequences for the patient, especially so if he had a CRT and not CRTD.

In the second case, the episodes are induced short time after the patient was upgraded to a dual chamber ICD. He previously had a single chamber ICD (VVI 40). At that time, he had equally

many VES with the same morphology and coupling interval. He had MVT episodes at that time. When upgraded because of symptomatic sinus bradycardia from the necessary medical treatment, he was paced in the atrium most of the time. During sensor-indicated rate increase coincident with increased activity-related VES frequency, the VES several times coincided with the atrial pacing stimulus eliciting a very short cross-chamber blanking period in the ventricular channel making the VES unsensed. It is well known to carry the risk that the Vp stimulus, after completion of the paced AV interval, falls in the vulnerable phase of the VES. However, this is actually not the sole mechanism here since the stimulus does not capture (LOC in the marker channel). Instead, it is the autocapture feature with beat to beat capture assurance that leads to the propagated response since the LOC leads to a slightly later and high output impulse of sufficient strength to overcome the relative refractoriness and elicit a propagated response that initiates the nonsustained PMVT. The autocapture was programmed off. The ICD

was programmed in atrial inhibited sensing and pacing (AAI) modus. No further episodes were observed for the following months. These two cases emphasize that automatic threshold features should be programmed very judiciously depending on clinical characteristics of the individual patient.

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