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Initiation of Ventricular Tachycardia by Interruption of Pacemaker-Mediated Tachycardia in a Patient with a Dual-Chamber Implantable Cardioverter Defibrillator

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A 74-year-old man with a dual-chamber implantable cardioverter defibrillator implanted 3 years before experienced multiple ventricular tachycardias (VTs). All episodes were initiated by pacemaker-mediated tachycardia (PMT) that was either stopped by atrial undersensing or the tachycardia termination algorithm of the device. After the termination of PMT, two rapid ventricular paced beats, the first initiated by artificial triggering and the second due to retrograde conduction of the first one, initiated VT that was successfully terminated by antitachycardia pacing or a direct current shock of the device. All episodes revealed this pattern of initiation with a short-long-short ventricular sequence inducing VT. (PACE 2009; 32: 1227–1230)

Introduction

Pacemaker-mediated tachycardia (PMT) can be encountered in patients with dual-chamber pacemakers or dual-chamber implantable cardioverter defibrillators (ICD) in the presence of retrograde ventriculo-atrial (V-A) conduction.1,2 PMT can be stopped successfully by a tachycardia termination algorithm that prolongs the post ventricular atrial refractory period (PVARP) for one beat.3 We report on a patient with a dual-chamber ICD that after the termination of PMT had one paced beat with retrograde conduction that triggered a second ventricular stimulated beat. The sequence of short (by PMT), long (by the tachycardia termination algorithm), and short sequence (by the ventricular triggered beat) induced through retrograde conduction repeatedly initiated sustained ventricular tachycardia (VT) in this patient.

Case Report

A 74-year-old patient with a previous myocardial infarction, who received a Guidant Vitality DR 1871 ICD (Boston Scientific, St. Paul, MN, USA) because of spontaneous monomorphic VT was hospitalized because of arrhythmic storm. The atrial lead impedance had increased to a value >3,000Ω and the atrial electrogram showed spurious signals causing undersensing in the atrial channel evoking rapid ventricular stimulation (Fig. 1).

Records of the onset of the VT have an identical pattern for all the episodes (Fig. 2). It started with ventricular pacing at the maximum tracking rate due to PMT. The PMT is interrupted by the tachycardia termination algorithm of the device that prolongs the PVARP for one cycle after 16 beats at the maximum tracking rate (Fig. 2., episodes 1 and 4) or in less than 16 beats at maximum tracking by undersensing of a retrograde P-wave (Fig. 2., episodes 2 and 3).

After the termination of PMT, the pacemaker starts atrioventricular sequential stimulation at the sensor-indicated rate because atrial noncapture ventricular pacing is followed by retrograde conduction, which evokes one ventricular paced beat at the maximum tracking rate (Fig. 2, episode 1). A similar pattern after the termination of PMT can be seen by artificial triggering in the atrial channel (Fig. 2., episodes 2–4). These two ventricular paced beats with a short coupling interval (463 ms) were preceded by a relative long ventricular interval (1,085 ms) and a short ventricular paced interval due to PMT-initiated VT. This pattern of initiation with a short-long-short sequence was seen in all VT episodes. In all recordings, far-field ventricular activity is sensed in the atrial channel, which, however, played no role in the mechanism of tachycardia initiation or termination.

To avoid recurrence of the problem, the system was programmed to the single-chamber mode at a low ventricular stand-by rate, which prevented further arrhythmias related to the previous described mechanism. Implantation of a new atrial lead will be performed at the elective replacement time of the device, which is anticipated within 1.5 years.

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Figure 1. Programmer strip recording showing the atrial electrogram, ventricular electrogram, shock lead electrogram, and annotations. The atrial electrogram shows spurious signals that trigger ventricular stimulation. The atrial lead impedance had increased above 3,000 Ω. Annotations: AP-Sr, VP-Sr = Atrial/ventricular pacing at the sensor driven rate; AS = atrial sensing; (AS) = atrial sensing in atrial refractory period; [AS] = atrial sensing in atrial blanking period; VP-MT = ventricular pacing at maximum tracking rate.

Atrial lead impedance > 3000 Ω

Figure 2. Four episodes showing the onset of ventricular tachycardia with an almost identical pattern. The PMT is terminated by the tachycardia termination algorithm in episodes 1 and 4, and by atrial undersensing in episodes 2 and 3. After the termination of PMT, the first ventricular paced beat gives retrograde conduction due to atrial noncapture (episode 1) or artificial atrial triggering (episodes 2–4).
Discussion

Pacemaker-related tachycardia has already been reported in the early days of cardiac pacing.4,5 PMT awareness started shortly after the introduction of devices with the capability of sensing and pacing in the atrium and pacing the ventricle synchronous with these atrial events. Due to the properties of the device, retrograde V-A conduction could be sensed by the atrial amplifier, giving rise to ventricular pacing again with V-A conduction, thus initiating pacemaker-mediated or “endless-loop” tachycardia.1,2

After application of a programmable PVARP, the initiation of PMT could be controlled by programming a PVARP long enough to ignore the retrograde P-wave. As an additional protection against sustained PMT, a tachycardia termination algorithm was incorporated in these devices.3 The termination algorithm detects pacing at the maximum tracking rate following atrial sense events, and after 16 consecutive ventricular paced beats, PVARP is extended for one beat, which ignores retrograde atrial activation thus breaking PMT.

There might be several causes for the initiation of PMT (e.g., atrial noncapture, artificial atrial triggering), but they all have in common that ventricular pacing is not preceded by atrial depolarization or the interval between atrial depolarization and ventricular pacing is long enough to regain atrial excitability by ventricular stimulation.

In our patient, it was clear that all episodes of PMT were initiated by artificial atrial triggering due to spurious signals caused by a conductor break in the atrial lead (Fig. 1). Figure 2 demonstrates the termination of the PMT by the tachycardia termination algorithm that prolongs PVARP at the 16th beat of the PMT. This is shown by the annotations at the last beat of the PMT in which the retrograde P wave is within the prolonged refractory period indicated by the annotation (AS), in spite of the identical coupling interval of 463 ms.

The “short-long-short” ventricular sequence is a well-known phenomenon responsible for the initiation of polymorphic ventricular arrhythmias in long QT syndromes.6–8 Ventricular arrhythmias evoked by a short-long-sort sequence are triggered either by early after depolarization or dispersion of repolarization.6 In our patient, RR-intervals during VT were stable and, we presume a monomorphic VT is most likely.9,10

The intervals and morphology of the QRS complexes initiating VT (Fig. 3, right panel) are identical to the first two QRS complexes of the PMT (Fig. 3, left panel). In both situations, the first ventricular stimulus is initiated by artificial

![Guidant Vitality DR 1871](image_url)

**Figure 3.** Surface electrocardiogram leads aVR, II, and atrial lead signal. The onset of PMT (left panel) and monomorphic VT (right panel). This illustrates that the first two beats of the PMT (arrows) and the first two beats initiating VT (arrows) are identical, with the same coupling interval of 463 ms (encircled) between both beats. For further explanation see text.
atrial triggering through spurious signals, leading to retrograde atrial activation. This atrial activation again triggers a ventricular stimulus, in both circumstances with an identical coupling interval of 463 ms, determined by the upper tracking rate of the device. In the right panel (Fig. 3), it is followed by a ventricular premature beat initiating monomorphic VT, whereas in the left panel (Fig. 3) PMT is continued. The difference between both recordings is the “long” interval preceding VT and PMT with, respectively, 950 ms and 675 ms and the rhythm preceding this “long” interval, which is a regular fast rhythm (PMT) in the right panel (Fig. 3) and irregular in the left panel.

Long-short-long sequences are a well-known trigger for monomorphic VTs in ICD patients. In the PREVENT study, this long-short-long mechanism as the cause of monomorphic VTs and the beneficial effect on reduction of VTs by application of a rate smoothing algorithm has been demonstrated.

The initiation of PMT could be further prevented by programming the device to a VVI pacing mode at a low stand-by rate. As this was clinically well tolerated by the patient, it was decided to avoid premature generator replacement or closely timed surgical procedures and to postpone replacement of the malfunctioning atrial lead to the time of generator replacement.

Conclusions

This case report demonstrates that PMT and the algorithm to terminate this device arrhythmia create a short-long-short ventricular interval sequence, which repeatedly initiated VT. The initiation could be prevented by programming the pacing system to a single-chamber pacing mode, which was a feasible option in our patient.

References