Pacemaker-mediated tachycardia with narrow QRS complexes

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Key Clinical Message
Inverted connection of the atrial and ventricular leads is an unusual circumstance during the implantation of a dual-chamber pacemaker. Yet, PMT may present in the absence of complex mechanisms. The detection and termination algorithms used by the device proved to be efficient for the adequate diagnosis and treatment.

Keywords
Antidromic, orthodromic tachycardia, pacemaker.

Introduction
Pacemaker-mediated tachycardia (PMT) is a common complication in patients with dual-chamber devices (DDD-VDD). This type of tachycardia is originated by an artificial reentrant circuit which uses the atrioventricular (AV) node and the His-Purkinje system as a retrograde pathway and an anterograde pathway that requires the presence of a ventricular stimulus after a sensed retrograde P wave (antidromic tachycardia) [1, 2]. Exceptionally, spontaneous QRS complexes can be seen in PMT (orthodromic tachycardia).

We describe a case of an orthodromic PMT with anterograde conduction through the AV node and the His-Purkinje system, and retrograde conduction by the pacemaker leads due to an inverted connection during the generator replacement.

Case Report
A 58-year-old woman underwent dual-chamber pacemaker (DDDR) implantation in 2008 due to symptomatic sinus node disease. The patient did not present device-related complications or symptoms of tachycardia during follow-up and reached the elective replacement indication (ERI) in December 2016. The pacemaker was replaced by a Boston Scientific model Advantio K083 device programmed in DDD mode with the following settings: output pulse of 3.0 V (pulse amplitude) and 0.4 msec (pulse width), pacing rate of 60–100 bpm, AV interval of 250 msec, ventricular refractory period of 250 msec, and atrial blanking period of 45 msec (sensed) and 125 msec (paced). After the new generator was connected, the monitor displayed paced ventricular beats at a stimulation rate of 60 bpm followed by spontaneous P waves, probably due to retrograde conduction (Fig. 1A). Then, bursts of tachycardia with narrow QRS complexes were initiated by a stimulated P wave at 250 msec after an effective ventricular stimulus (Fig. 1B). These episodes were recorded in the device memory as PMT, and the algorithm of PMT termination was initiated after the detection of 16 consecutive ventricular pace (VP) and atrial sense (AS) cycles occurring at the programmed maximum tracking rate (Fig. 2) [1].
After checking the presence of spontaneous ventricular activity, the pacemaker was programmed in the different pacing modes that are summarized in Figure 3. The DDI mode put in evidence that the first stimulus activated the ventricle and a second ineffective stimulus occurred after an interval of 250 msec (Fig. 3A). The AAI mode demonstrated that atrial pacing stimulated the right ventricle, and in the VVI mode, the ventricular stimulus activated the atrium and was followed by a narrow QRS complex after a prolonged AV interval of 320 msec (Fig. 3B and C, respectively). All these data suggested that the connections of the atrial and ventricular leads were inverted, and thus, they stimulated and sensed inverse chambers.

After both leads were properly reconnected, the PMT immediately stopped. The patient remains without symptoms so far. Subsequent follow-up with 24-h Holter monitoring and device interrogation did not show new episodes of PMT.

Discussion

Pacemaker-mediated tachycardia is a common complication observed with dual-chamber pacing. Usually, PMT is initiated by a ventricular premature beat or a paced ventricular beat that is not preceded by a P wave within the programmed AV interval (lack of atrial capture or sensing) and is followed by a retrograde P wave that is sensed by the atrial lead. Once the programmed AV interval ends, the pacemaker generates a stimulus in the ventricular lead. The arrhythmia is sustained as long as retrograde conduction persists (antidromic PMT) [2, 3].

Exceptionally, PMT may adopt an opposed direction. In this case, there is anterograde activation through the AV node, bundle of His and branches with narrow QRS complexes (orthodromic PMT).

This type of tachycardia was observed in the nineties in some models of pacemakers (Vitatron Quintech DDD 931, Telectronics Quadra 9221, Biotronik Diplos 06, Physios and TuR Reficard duo). These devices had the option of programming synchronous atrial stimulation following a ventricular premature beat to prevent the propagation of the retrograde stimulus. If the anterograde conduction was preserved, this atrial activation could propagate and activate the ventricles. The pacemaker would recognize this spontaneous ventricular activation as a ventricular premature beat (because it did not detect

![Figure 1. ECG monitoring after connecting the new pacemaker generator. (A) Pacemaker rhythm with paced ventricular beats, followed by P waves which seem to be retrograde. (B) After the third paced ventricular beat, an atrial captured beat (asterisk) initiates a narrow QRS complex tachycardia at a heart rate of 110 bpm that is perpetuated by atrial stimulus.](image)
a spontaneous or paced P wave) and would then emit a new synchronous atrial stimulus which perpetuates the reentry mechanism [4, 5].

In 2004, Berruezo et al. reported a case of orthodromic PMT in a patient with cardiac resynchronization therapy (Medtronic InSync III model 8042). This device incorporated a ventricular sense response algorithm to ensure a high percentage of biventricular pacing in patient with atrial fibrillation, providing biventricular stimulation each time the device detects spontaneous ventricular activity.
The PMT was due to dislodgment of the left ventricular lead to the coronary sinus, near the AV groove, and during sinus rhythm the stimulation of the left ventricle resulted in simultaneous capture of the left atrium and left ventricle. After left atrium activation, the electrical impulse propagated through the AV node and activated the right ventricle. This spontaneous right ventricular activation was sensed by the right ventricular electrode that triggered biventricular pacing and left atrial capture, perpetuating the tachycardia [6].

In our patient, the PMT was caused by an inverted connection of the atrial and ventricular leads during generator replacement which produced stimulation of the right ventricle by the atrial lead. The retrograde P wave was wrongly recognized by the device as a spontaneous ventricular activation. In the absence of a P wave due to retrograde block of the ventricular–atrial conduction, the device emitted a ventricular stimulus which activated the right atrium. Then, the electrical impulse propagated through the normal AV pathway and activated the right ventricle. The device interpreted it as spontaneous atrial activation and emitted a new ventricular stimulus that triggered an orthodromic reentrant tachycardia.

Compared with antidromic PMT, orthodromic PMT has distinctive features as the ventricular stimulus captures the atrium and triggers the tachycardia. It is important to consider that variations of the AV interval could not have prevented the development of PMT. Conversely, the tachycardia could only be suppressed if the spontaneous ventricular event cannot by detected by the atrial lead due to prolongation of the postventricular atrial refractory period (PVARP) >350 msec.

This case has been useful to analyze an unusual mechanism of PMT and to understand the algorithm used by the device during this arrhythmic event. Despite the fact that the connections of the atrial and ventricular leads were inverted, the device was capable of making the adequate diagnosis and treatment of this type of event.

**Conflict of Interest**

None declared.

**Authorship**

HSY: conceived, designed, and drafted the manuscript. EA, PF, and DE: analyzed and interpreted the data. RT, HG, JP, and RA: critically reviewed the manuscript.

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An unusual variant due to orthodromic reentry

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