One problem solved by the occurrence of another?

Recurrent tachycardia during pacemaker interrogation

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Case presentation

A 76-year-old man was seen in the pacemaker clinic for routine follow-up. He had undergone implantation of a dual-chamber pacemaker (SJM Verity ADx XL DR 5356) for bifascicular block and high-degree atroventricular (AV) block on Holter-ECG after suffering syncope. After the implant, no more syncope occurred and the patient was feeling well. During the follow-up visit, recurrent runs of a tachycardia were observed (fig. 1), from which the patient was completely asymptomatic.

The following five questions need to be answered:

1. What is the tachycardia mechanism?
2. How does the tachycardia start?
3. Why does the AV delay prolong during tachycardia?
4. What is the mechanism of tachycardia termination?
5. What needs to be done to fix the problem?

What is the tachycardia mechanism?

The tracing in figure 1 shows a tachycardia with a CL of 500 ms and a 1:1 AV conduction. The tachycardia starts
with a prematurely sensed atrial event (third atrial event on the tracing, marked * in figure 2) and the tachycardia is tracked to the ventricle with an AV delay of 240 ms. During tachycardia, there is double ventricular farfield oversensing in the atrium. The first additional signal on the atrial channel, marked with a blue up-arrow in figure 2, falls into the postventricular atrial blanking period (PVAB) and represents a farfield signal from the ventricular paced beat. The second oversensed signal (marked with a red down-arrow in figure 2) is probably oversensing of the end of the QRS complex; T-wave oversensing is unlikely because it occurred even before the T-wave. This atrial signal falls into the postventricular atrial refractory period (PVARP).

The differential diagnosis of the tachycardia at this point is an atrial tachycardia conducted to the ventricles by tracking of the pacemaker, or an endless-loop reciprocating pacemaker-mediated tachycardia (PMT) due to retrograde conduction of ventricular paced beats that are sensed in the atrium after the PVARP. The end of the tachycardia contains the solution to this: after seven beats, the tachycardia terminates with an atrial beat that is no longer tracked to the ventricle (marked § in figure 2). This makes atrial tachycardia highly unlikely, as one would have to assume simultaneous termination of an atrial burst and loss of ventricular tracking. The mechanism therefore is an endless-loop reciprocating pacemaker-mediated tachycardia.

How does the tachycardia start?
In this patient with higher degree AV-Block, there usually is concealed antegrade conduction into the AV-node preventing retrograde conduction of following ventricular paced beats back to the atrium. With the second atrial event in the tracing, however, there must have been block above the node or in the node, which then allowed retrograde conduction after the second ventricular paced (VP) event and initiation of the PMT (marked * in figure 2). Alternatively, this third atrial sensed (AS) event might also be a premature atrial beat, which blocks in the AV node and allows the next VP event to conduct back to the atrium and initiate the PMT. Given that the VA time is the same for all the tachycardia beats, this possibility appears less likely. Other precipitating factors capable of initiating a PMT could be ventricular extrasystoles, loss of atrial capture, atrial oversensing followed by atrial underpacing, atrial extrasystoles, or intermittent loss of atrial sensing.
Why is the atrioventricular delay prolonged during tachycardia?
The sensed and paced AV delays are programmed to 150 ms and 170 ms and are seen on the first and second beat on the tracing. With the third beat (first tachycardia beat), the AV delay is prolonged to 240 ms, which remains fixed throughout the tachycardia. The prolongation is due to upper-rate behaviour. Maximum tracking rate is 120/min, equalling a minimal ventricular interval of 500 ms. The interval between the two AS events initiating tachycardia, however, is 410 ms. Accordingly, the next VP can only be delivered after 500 ms, which results in a prolongation of the sensed AV delay (150 ms) by 90 ms to 240 ms.

What is the mechanism of tachycardia termination?
All contemporary dual-chamber pacemakers have algorithms to prevent endless-loop PMT. In the case of this patient’s SJM pacemaker, the PMT algorithm is active and would have intervened after 10 beats of PMT. However, the tachycardia already stopped after seven beats. On a closer look, the mode-switch was seen to be activated owing to inappropriate ventricular Far-Field oversensing in the atrium. With the inappropriate activation of the mode-switch and the associated change from the DDD-R into the DDI-R mode, the PMT terminated after seven beats even before the PMT algorithm was activated. The annotation of AMS (automode switch), however, only appears after the next VP event in older SJM devices.

What needs to be done to fix the problem?
The intrinsic VA time during V pacing was measured at 260 ms. After extending the PVARP from 250 ms to 275 ms, the PMT no longer occurred. Instead, the retrogradely conducted beats fell appropriately into the PVARP (fig. 3).

Summary
In summary, we report a pacemaker case, in which a common problem was resolved by the coincidental occurrence of a second common problem: a pacemaker-mediated tachycardia was terminated by inappropriate mode switch as a result of atrial oversensing. Even though the patient was asymptomatic in this case, the exact knowledge of the mechanisms leading to the occurrence of the two frequent problems as well as the appropriate steps to resolve them are important for the management of pacemaker patients.

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