INTRODUCTION

A 41-year-old woman with a dual-chamber pacemaker underwent radiofrequency ablation of the slow pathway for treatment of atrioventricular nodal reentrant tachycardia. After ablation, a pacemaker-mediated tachycardia exhibiting changes in cycle length and AV interval was induced. The mechanisms responsible for these variations are discussed.

Pacemaker-mediated tachycardia is a well-known complication of dual-chamber pacing systems. These arrhythmias may be related to tracking of fast atrial rhythms, myopotentials, lead noise, and electromagnetic interference. However, the archetypal pacemaker-mediated tachycardia is endless-loop tachycardia that may happen in patients with intact ventriculoatrial (VA) conduction through the atrioventricular (AV) node or an accessory pathway. This arrhythmia is a macro-reentrant circuit in which the antegrade limb is formed by the pacemaker and the retrograde limb by VA conduction.

It is well recognized that right ventricular pacing promotes long-term adverse effects on left ventricular function. Therefore, to minimize right ventricular pacing, advanced algorithms promoting intrinsic AV conduction have been developed. Atrioventricular search hysteresis prolongs AV delay to allow intrinsic AV conduction. If no spontaneous AV conduction is found, AV delay returns to a programmed basic value. These changes in the AV delay, sometimes leading to beats exhibiting AV block, may result in complex rhythm strips. Further, sudden changes in AV conduction may promote retrograde unidirectional block in patients with dual AV nodal physiology.

We describe a patient with dual AV nodal physiology in whom a pacemaker-mediated tachycardia exhibiting changes in cycle length and AV interval was induced.

CASE REPORT

A 41-year-old woman with documented narrow QRS complex tachycardia was referred for electrophysiology study and radiofrequency catheter ablation. She had a
dual-chamber pacemaker (Evia-DR-T, Biotronik, SE & Co KG) implanted 4 years prior to this admission to treat recurrent vasovagal syncope. In non-pacemaker-dependent patients undergoing ablation, we routinely program the device to VVI mode at 30 bpm before the procedure. But in this case, the device was inadvertently programmed to DDD with heart rate of 30 bpm, dynamic AV delay of 200/100 (ms), and postventricular atrial refractory period (PVARP) of 175 ms. Intrinsic Rhythm Support (IRS-plus) with up to 400 ms AV hysteresis and pacemaker-mediated tachycardia protection were on. During electrophysiology study, typical, slow-fast atrioventricular nodal reentrant tachycardia (AVNRT) was reproducibly induced by atrial pacing and slow pathway ablation was performed. After ablation, arrhythmia induction with the use of isoproterenol was attempted using programmed atrial stimulation from the proximal coronary sinus. The ablation catheter was positioned at the high right atrium (HRA) to record local potentials. Following a 600-ms atrial drive, a single 360 ms extrastimulus initiated a pacemaker-mediated tachycardia of 580 ms cycle length (Figure 1). During tachycardia, a change in cycle length from 580 to 540 ms preceded by an abrupt shortening of the AV interval (measured from the HRA electrogram to the ventricular pacing spike) from 400 to 250 ms was noted (*). What are the mechanisms associated with this change in tachycardia cycle length?

3 | DISCUSSION

Pacemaker-mediated tachycardia is a complication of dual-chamber tracking modes that can occur in patients with intact VA conduction.1 In this arrhythmia, a retrograde P wave is sensed outside the PVARP initiating an AV interval that results in ventricular pacing. This cycle then reoccurs perpetuating the tachycardia. This arrhythmia may be triggered by ectopic beats (atrial and ventricular), loss of atrial capture or sensing, and long AV delays.

In our case, pacemaker-mediated tachycardia induction was dependent on a critical AV delay. Although AH and HV intervals were not recorded at this time, tachycardia was

**FIGURE 1** Induction of pacemaker-mediated tachycardia with a single 360 ms atrial extrastimulus. Tachycardia cycle length changes from 580 to 540 ms preceded by sudden shortening of the atrioventricular interval from 400 to 250 ms (*). On the right side of the tracing, there are three stimulation artifacts (cycle length 600 ms) that do not capture the atrium. From top to bottom: ECG leads I, II, III, V1, and V6; high right atrium (HRA); coronary sinus (CS) proximal (CSp), mid (4, 3, and 2), distal (CSd). Paper speed 50 mm/s
initiated by gradual prolongation of PR interval until a critical conduction delay was reached (Figure 2). At a cycle length (S1-S1) of 600 ms, atrial extrastimulation with a coupling interval (S1-S2) of 380 ms results in ventricular activation through the normal conducting system with pseudofusion (pacing spike does not capture). On the mid panel (370 ms), fusion activation (*) is evident on the ECG. On the right panel (360 ms), full ventricular capture with a long AV delay occurs, enabling concentric retrograde atrial activation and initiation of tachycardia. Abbreviations are as in Figure 1. Paper speed 100 mm/s

As shown in Figure 3, in the beginning of tachycardia, cycle length was 580 ms, whereas AV and VA intervals, measured from HRA to ventricular pacing spike, were 400 ms and 180 ms, respectively. After six atrial cycles, however, sudden shortening of the AV interval from 400 to 250 ms is noted. In the next cycles, further decrease to 160 and 150 ms occurs. This is a feature of the IRS-plus algorithm (Biotronik) to promote intrinsic AV conduction and limit right ventricular pacing. The repetitive hysteresis function extends the AV interval over six atrial cycles. If spontaneous AV conduction is not detected, the AV delay returns to a basic value. However, shortening of the AV interval to 250 ms (cycle #7) instead of the programmed value of 160 ms (cycle #8) may be related to activation of the pacemaker-mediated tachycardia protection algorithm. It is well recognized that the IRS-plus algorithm may cause pacemaker-mediated tachycardia with several changes in cycle length.

The pacemaker-induced AV interval optimization, per se, does not explain the change in tachycardia cycle length from 580 to 540 ms Although AV interval was shortened in 250 ms, cycle length decreased by 40 ms
only. This observation can be explained by an increase in conduction time along the retrograde limb of the circuit. Sudden shortening of AV interval advances ventricular activation by 150 ms (cycle #7) producing block in the retrograde fast pathway with a jump to the slow pathway, prolonging VA conduction time from 180 to 390 ms (cycle #8) with a shift of the earliest retrograde atrial activation from the coronary sinus ostium (CSp) to the fourth bipolar pair (CS4). After termination of the tachycardia by overdrive atrial pacing, the ablation catheter was moved to the right ventricle. Retrograde dual pathways could be demonstrated by decremental ventricular pacing (Figure 4), but atypical, fast-slow, AVNRT was not induced. In addition, induction of retrograde Wenckebach phenomenon by rapid ventricular pacing makes an accessory pathway unlikely. Corroborating our findings, up to 23% of patients with slow-fast AVNRT exhibit retrograde jumps associated with shifts of atrial activation from the right to the left side of the septum. Nevertheless, since a His bundle electrogram was not recorded we cannot ascertain that retrograde conduction was over the fast pathway. In view of the somewhat long VA interval to the HRA (180 ms), it is conceivable that retrograde conduction occurred via separate slow pathways (slow-slow AVNRT).\(^5\)

Pacemaker-mediated tachycardia must be differentiated from tracking atrial tachycardia. Induction was dependent on a critical AV delay.\(^1\) Retrograde atrial activation sequence during tachycardia and VVI pacing at 120 bpm was similar. Finally, tachycardia was no longer inducible by reprogramming the pacemaker mode from DDD to VVI. Despite dual AV nodal physiology, programmed atrial and ventricular stimulation could not induce any tachycardia. At 1-year follow-up, the patient remains free of tachycardia. This case highlights the importance of understanding device algorithms to allow appropriate management of arrhythmias induced by pacing, particularly when other types of tachycardia are involved.
ACKNOWLEDGMENT
We are grateful to Mr Caio Vinha, from Biotronik Brazil, for his expert technical assistance.

CONFLICT OF INTEREST
None declared.

AUTHOR CONTRIBUTIONS
GF and LCP: wrote the manuscript and care of the patient. FS, CC, and MF: critically reviewed the text and care of the patient. All the authors read and approved the final manuscript.

ETHICAL APPROVAL
Approval for this report was obtained from the ethics committee of Hospital Israelita Albert Einstein. A copy of the approval is available for review by the Editor-in-Chief of this journal.

CONSENT FOR PUBLICATION
Written informed consent was obtained from the patient for publication of this case report and its accompanying image. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

DATA AVAILABILITY STATEMENT
This case report is not supported by any grants. All data generated or analyzed during this study are included in this paper.

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**How to cite this article:** Fenelon G, Paul LC, Scuotto F, Cirenza C, Franken M. Change in cycle length during pacemaker-mediated tachycardia: What is the mechanism?. *Clin Case Rep*. 2021;9:e04271. https://doi.org/10.1002/ccr3.4271