ELECTROCARDIOGRAM UNKNOWNS

Long and longer retrograde conduction. What is the solution?

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Introduction

Cardiac implantable electronic devices (CIED), such as pacemakers and implantable cardioverter-defibrillators, are implanted to prevent and treat rhythm disorders. However, CIED may also lead to the development of arrhythmias. We describe a case with a CIED-related arrhythmia where close examination of the electrocardiograms will reveal the diagnosis, etiology, and solution for this arrhythmia.

Case report

A 70-year-old patient visited the outpatient clinic because of fatigue and palpitations. These symptoms started after the implantation of a cardiac resynchronization therapy internal cardioverter-defibrillator (CRT-D) with a plugged left ventricular port (Boston Scientific Resonate CRT-D G242) for primary prevention because of a hypertrophic obstructive cardiomyopathy. He had a pre-existent left bundle branch block with a QRS duration of 178 ms, but a preserved left ventricular function with an ejection fraction of 60%. A "plugged" CRT-D was chosen in this patient over a standard dual-chamber ICD because national health care policies limit the reimbursement of a new device when an upgrade would be necessary within a few years. This choice makes it possible to upgrade the device to CRT with the implantation of a left ventricular lead in case of evolution to a dilated cardiomyopathy without changing the defibrillator itself.

The CRT-D was programmed in DDD mode with a lower rate of 50 and upper tracking rate of 105 beats per minute (bpm) (Table 1). The patient also had a history with paroxysmal atrial fibrillation and an aortic and mitral valve bioprosthesis. During the outpatient visit the initial heart rhythm was sequential atrial-ventricular pacing and frequent premature ventricular beats (PVC) (Figure 1A). Suddenly an arrhythmia developed during the outpatient visit, which stopped spontaneously and recurred multiple times (Figure 1B).

What is the underlying mechanism and etiology of this arrhythmia? What is the solution to prevent this arrhythmia in the future?

KEY TEACHING POINTS

- The rate of a pacemaker-mediated tachycardia (PMT) may be lower than the upper tracking rate of the pacemaker or implantable cardioverter-defibrillator when slow retrograde ventriculoatrial conduction is present.
- Underdiagnosis and undertreatment of PMT may occur in case of slow retrograde ventriculoatrial conduction, since the conventional PMT algorithm is only activated when upper rate tracking is present and may be insufficient if the retrograde conduction is >500ms.
- In the case of very slow retrograde conduction, prevention of PMT may only be possible by prolonging the postventricular atrial refractory period after a premature ventricular beat.

Discussion

The electrocardiogram (Figure 2A) shows an endless-loop, retrograde-dependent pacemaker-mediated rhythm of 80 bpm. The rhythm corresponds with a common pacemaker-mediated tachycardia (PMT) with retrograde P waves (red arrows), but with a significantly prolonged retrograde ventriculoatrial conduction of 520 ms. The cycle length of the PMT was around 750 ms, which equaled the retrograde ventriculoatrial conduction time (520 ms) and programmed AV delay (adaptive 190–270 ms). Therefore, the rate was only 80 bpm, which is formally not a tachycardia. However, we classify this endless-loop,

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Table 1 Device settings for bradytherapy

Pacing mode	DDD
Lower rate	50
Upper rate	105
Atrial output (bipolar)	3.5 V @ 0.4 ms
Ventricular output (bipolar)	3.5 V @ 0.4 ms
Atrial sensitivity (bipolar)	0.25 mV
Ventricular sensitivity (bipolar)	0.6 mV
Paced AV delay	210–300 ms
Sensed AV delay	190–270 ms
PVAB	Smart
PVARP	360–380 ms
PVARP after PVC	400 ms
Ventricular blanking after atrial pace	65 ms
VRP	230–250 ms

PVAB = postventricular atrial blanking; PVARP = postventricular atrial refractory period; PVC = premature ventricular complex; VRP = ventricular refractory period.

retrograde-dependent pacemaker-mediated rhythm as a PMT for reasons of clarity, since no other unifying terminology exists for PMT with a rate below 100 bpm.

PMT is a known complication of CIED. The mechanism of a PMT involves the device repeatedly tracking a retrograde impulse from a previous ventricular paced beat and is often triggered by PVC.[1] It is important to distinguish a classic PMT from a repetitive nonreentrant ventriculoatrial synchrony (RNRVAS), which is closely related to a PMT. However, RNRVAS is a repetitive process with atrial undersensing owing to retrograde atrial activation falling within the postventricular atrial refractory period (PVARP) and subsequent functional atrial noncapture, since the atrial stimulus falls within the absolute atrial refractory period.[2] Therefore, extending the PVARP to prevent PMT may result in increased risk for RNRVAS and vice versa.

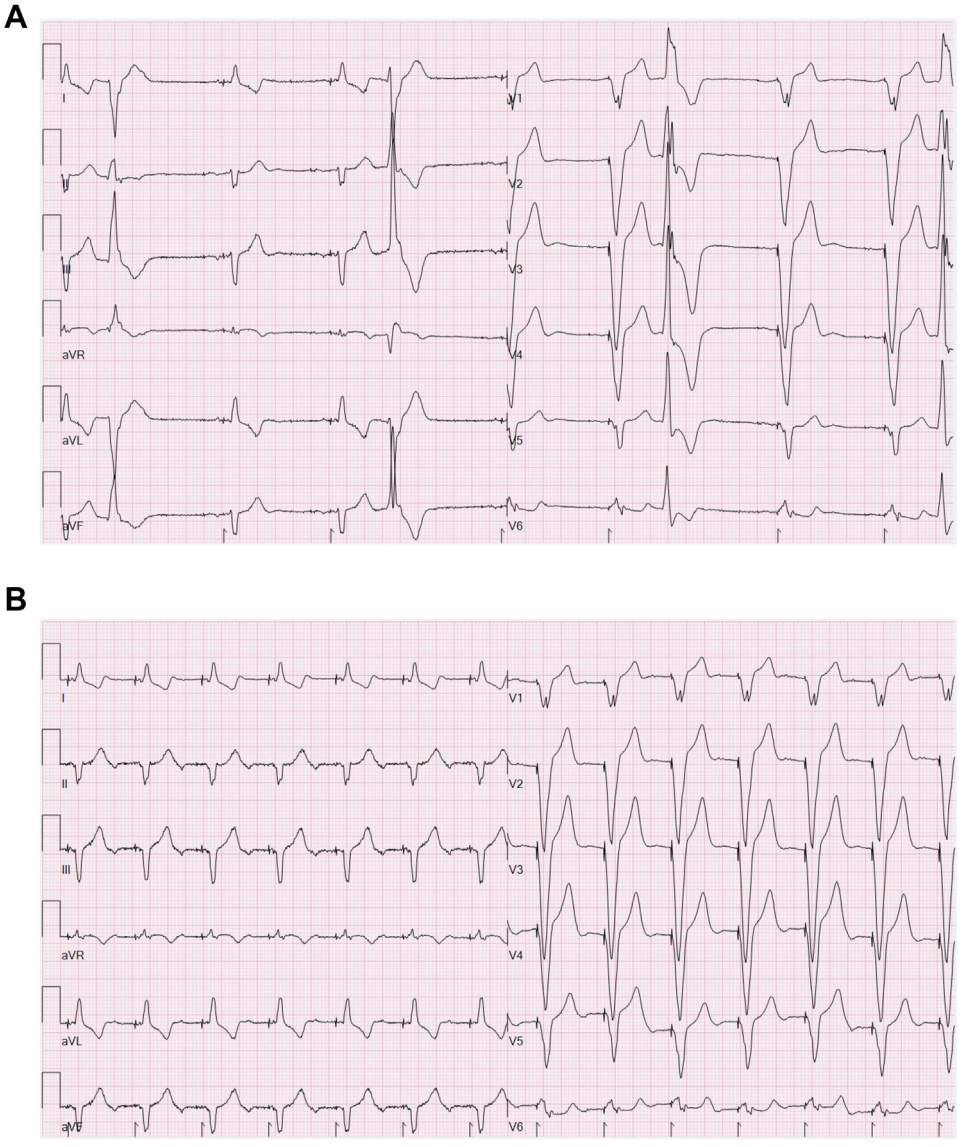

Figure 1 **A:** The initial electrocardiogram during the outpatient visit showed sequential atrial-ventricular pacing and frequent premature ventricular beats. **B:** Suddenly an arrhythmia developed during the outpatient visit.

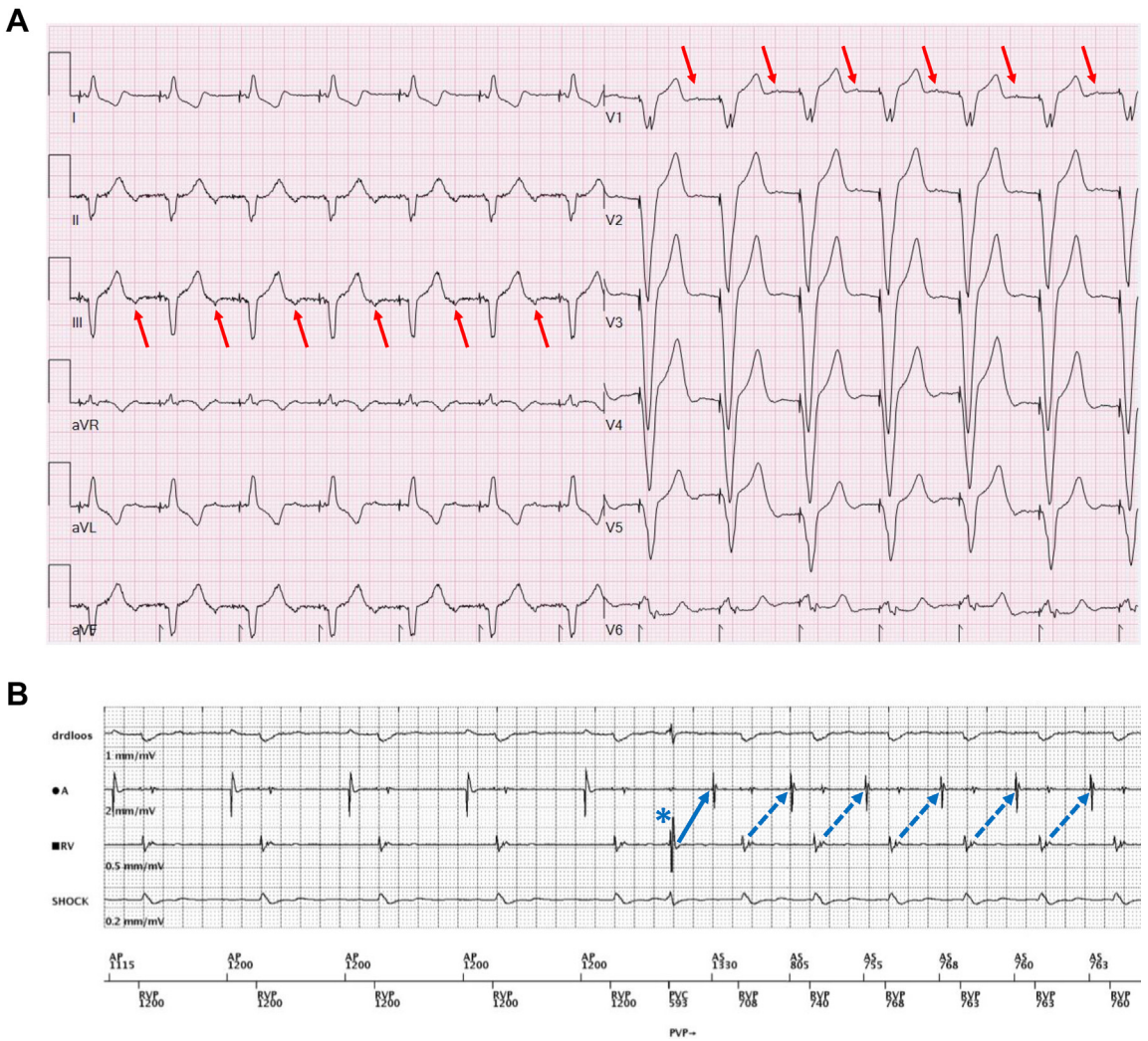

Figure 2 **A:** The electrocardiogram shows an endless-loop, retrograde-dependent pacemaker-mediated rhythm of 80 beats/min with retrograde P waves (*red arrows*) with significantly prolonged retrograde ventriculoatrial conduction of 520 ms, which corresponds with a pacemaker-mediated tachycardia (PMT). **B:** The intracardiac electrogram shows the exact onset and etiology of the PMT. First the tracing shows sequential atrial-ventricular pacing. However, the sixth QRS complex is a premature ventricular beat (*) with retrograde ventriculoatrial conduction of approximately 420 ms (*blue arrow with solid line*), which triggers the PMT. During the PMT the retrograde conduction exceeds the maximal PVARP of 500 ms (*blue arrows with dotted dash line*) and the rate is below the maximal tracking rate.

The exact onset and etiology of this particular PMT could be retrieved from the real-time intracardiac electrograms during device interrogation (Figure 2B). First the electrogram tracing shows sequential atrial-ventricular pacing. However, the sixth QRS complex is a PVC with retrograde ventriculoatrial conduction of approximately 420 ms, which triggers the PMT. There were 2 reasons why this particular PMT could not be diagnosed and terminated by the conventional device algorithms. First a PMT is normally terminated by extending the duration of the PVARP up to 500 ms once the PMT is detected. In this case, only a PVARP of >520 ms would have been sufficient, but the maximum duration of the PVARP is limited to 500 ms. In addition, the device did not recognize the PMT because the rate of the PMT was only 80 bpm owing to the slow retrograde conduction, which was below the upper tracking rate. Therefore, the conventional PMT algorithm was not triggered. Finally, the patient had a "plugged" CRT-D instead of a regular dual-chamber ICD, which may prevent an early replacement of the device in case of a future upgrade. However, one disadvantage of this practice is that it may not be possible to program an AAI-DDD mode (or RHYTHMIQ with AAI and VVI back-up in the case of Boston Scientific) when antegrade conduction is present, since not all CRT devices have this option. An AAI-DDD mode or AAI with VVI back-up mode would have prevented the occurrence of PMT in this case when a long intrinsic AV interval was accepted, since slow antegrade conduction was present. Unfortunately, this possibility was not available in this "plugged" CRT-D.

The initiation of this particular PMT could still be prevented by specifically extending the PVARP after PVC time interval.[3] The PVARP after PVC time interval was extended to the maximum limit of 500 ms and no further PMT occurred. This solution was only possible because the retrograde conduction after a PVC was faster, and therefore

within the limits of the maximum programmable duration of the PVARP, in comparison to the retrograde ventriculoatrial conduction after ventricular pacing during PMT. The difference in the duration of the retrograde conduction after a PVC vs right ventricular pacing can be explained by decremental properties of the retrograde ventriculoatrial conduction or by the location of the PVC, which seemed to be in or close to the anterior fascicle, vs the midseptal position of the right ventricular lead.

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