CASE REPORT

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A sudden change in pacing rate: Normal or malfunction?

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Abstract

A woman with a dual-chamber pacemaker was examined for recurrent chest discomfort and palpitations at our hospital. The Holter monitor test recorded recurrent episodes of a sudden increase in pacing rate from 60 to 105 bpm, which corresponded to the symptoms. Orthodromic pacemaker-mediated arrhythmia (OPMA), caused by ventricular lead dislodgement and atrial far-field sensing, caused the recurrent episodes of a sudden change in pacing rate. The occurrence of OPMA may represent a rare but noteworthy pacemaker problem. To our knowledge, our study reports the first case of PMA that only occurs, and is maintained, in the DDI mode.

KEYWORDS

auto-mode switch, far-field sensing, lead dislodgement, pacemaker-mediated arrhythmia

CASE REPORT

A 66-year-old woman with a history of hypertension was examined for recurrent chest discomfort and palpitations at our hospital. A dual-chamber pacemaker (Abbott/SJM) was implanted due to sick sinus syndrome 1 year ago. The Holter monitor test recorded recurrent episodes of a sudden change in pacing rate, starting with an intrinsic P wave (Figure 1a). In total, 25 episodes were recorded, of which, the longest episode lasted for 3 min. Most episodes corresponded to the patient's symptoms of recurrent chest discomfort and palpitations. Notably, Figure 1b shows pacing pulses in two opposite directions: an upward pulse (Up), followed by a positive P wave, and a downward pulse (Dp), followed by a negative P wave.

The patient offered following information about the last interrogation: DDD mode, base rate 60 bpm, auto-mode switch base rate

(AMSBR) 80 bpm, PAV/SAV interval 300/250 ms, and activity sensor off.

What accounts for the sudden change in pacing rate?

2 | COMMENTARY

The following reasons may be attributed for the sudden change in pacing rate: an auto-mode switch (AMS) (Sharma et al., 2016), atrial overdrive pacing function (Hohnloser et al., 2012), and sensor rate. Every episode started with an intrinsic P wave, which is consistent with the startup characteristics of AMS in the SJM (Barold, 2017). However, no atrial tachycardia was observed before any episode. This suggested that the AMS might be due to atrial oversensing. In contrast, the atrial pacing rate (105 bpm, AP-AP interval 570 ms) did not correspond with AMSBR (80 bpm). The atrial pacing rate to

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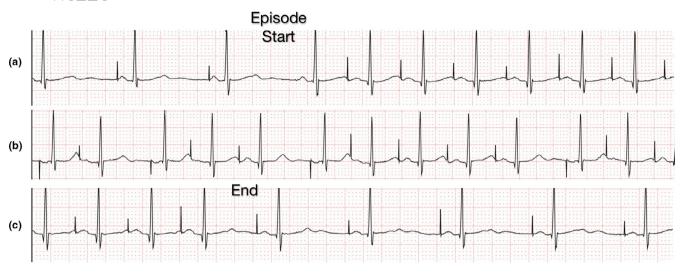


FIGURE 1 (a) A sudden change in pacing rate after an intrinsic P wave; (b) pacing rate at 105 bpm (upward pulses) and pacing rate at 70 bpm (downward pulses). (c) Termination of the rapid pacing

ventricular sensed (AP-VS) sequence in the DDI mode can be faster than AMSBR, as the VS would reset the VV and VA intervals [VV and VA intervals in DDI are equal to 750 and 450 ms, respectively]. In our case, the AP-AP interval of the AP-VS sequence should be 650 ms (92 bpm). Nevertheless, both the actual measured VA interval (320 ms) and AP-AP interval (570 ms) were shorter than the calculated values.

Surprisingly, the Dp-Up interval was precisely equal to the VA interval (Figure 2a). According to variations in P waves after Dp and Up, one of Dp and Up should be a pseudo-atrial pacing pulse (e.g., a dislodged ventricular lead). We assumed that Dp, followed by a negative P wave, was due to ventricular lead dislodgement to the atrium. If so, the lead would sense P waves rather than intrinsic QRS complexes as VS events. If moved backward at 450 ms (VA interval) from Up, we would reach the peak of the recent P wave or Dp. If moved backward at 750 ms (VV interval) from Dp, we would reach the same position of the P wave. Therefore, the measured shorter VA intervals might be due to the repetitive resetting of VA intervals from ventricular lead sensing-paced P waves as in VS events (Figure 2a). In such case, paradoxical pacing (VP failed to pace the ventricle but captured the atrium) should be considered.

Ensuing device interrogation supported this deduction (Figure 2b): (paradoxical sensing) in the upper strip, every VS (blue arrow) event corresponded to the P wave, and the subsequent QRS complex was completely ignored. In our limited observations, both paradoxical sensing and pacing strongly support dislodgement of the ventricular lead to the atrium. In the lower strip, a stable atrial refractory event (AR) corresponded to every J point of the QRS complex (Figure 2b),

which confirmed the intermittent atrial oversensing of the far-field QRS complex. In SJM, AR, followed by an AP, is counted toward FARI. A continuous AP-AR sequence followed by an intrinsic sensed P wave may result in an inappropriate AMS, which is similar to repetitive non-reentrant ventriculoatrial synchrony. Intermittent atrial oversensing and lead dislodgement were fixed during re-operation. The patient did not experience any chest discomfort or palpitations after re-operation.

Pacemaker-mediated arrhythmia (PMA) is a broad term used to describe the abnormal rhythms to which the pacing system contributes. Orthodromic pacemaker-mediated arrhythmia (OPMA) is quite rare in PMA (Alasti et al., 2018), OPMA may lack commonality in triggers because published cases demonstrate varying causes. However, the occurrence of OPMA may represent a rare but noteworthy pacemaker problem (Herczku et al., 2010; Ozeke et al., 2018). To the best of our knowledge, our study reports the first case of OPMA that only occurred, and was maintained, in the DDI mode (most PMA would be terminated immediately in the DDI mode) (Figure 2c). Of note, a 300/250-ms PAV/SAV interval may be below the optimal level in many patients to minimize ventricular pacing rates. Therefore, AAI-DDD switch modes (e.g., SafeR and MVP) and second-generation AV hysteresis (e.g., VIP, Search AV+, and IRS plus) should be considered. Lead dislodgement may occur when paced P waves vary, especially when accompanied with paradoxical sensing and pacing. Device interrogation can visually expose some pacemaker problems, but intermittent tissues may not appear during interrogation. The Holter or continuous ambulatory cardiac monitoring test may help in identifying intermittent malfunctions.

FIGURE 2 (a) An explanation to the rapid atrial pacing. Step 1: Ventricular lead sensed the P wave as a VS event and ignored the subsequent intrinsic QRS; Step 2: The VS triggered a new VA interval, and the subsequent AP continued the cycle. Step 3: If the device sensed an AS event during the VA interval, AP would be inhibited, and the subsequent VP was generated after 750 ms (VV interval). Further, VP captured the atrium, which also continued the cycle. However, we did not note any corresponding P wave that might inhibit the AP, which hinted that the intermittent atrial oversensing might exist. (b) The device interrogation corroborated the working hypothesis. (c) The tachycardia diagram in this case. AP, atrial pacing; AS, atrial sensedVA, ventriculoatrial; VP, ventricular pacing; VS, ventricular sensed

CONFLICT OF INTEREST

Markers

None.

AUTHOR CONTRIBUTIONS

All authors reviewed and approved the manuscript. Directed this study: Yiru Han, Yubin Zhang, Liangrong Zheng. Wrote the manuscript: Yiru Han, Yubin Zhang. Gave suggestions on this study: Liangrong Zheng, Tong Liu.

ETHICAL APPROVAL

The study was approved by the institutional review board of The First Affiliated Hospital, Zhejiang University School of Medicine, Hangzhou, China. The patient provided written informed consent.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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