ECG for Students and Associated Professionals

Pseudofusion in a dual chamber pacemaker: Is this pacemaker working properly?

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\section{1. Case presentation}

A 57-year-old woman with a history of dizziness due to episodes of complete heart block and bradycardia was referred to the electrophysiology ward for pacemaker implantation. A dual chamber pacemaker (Medtronic, Relia, Medtronic, Minneapolis, USA) was implanted. Its analysis showed normal function (atrial lead capture threshold 0.5 V at 0.4 ms, ventricular lead capture threshold 0.75 V at 0.4 ms, and P-wave and R wave amplitude sensing 2 mV and 8 mV, respectively). The pacemaker was programmed in the DDDR mode with a lower rate limit of 60 bpm, atrial and ventricular output of 3.5 V and 3.5 V at 0.4 ms respectively, atrial and ventricular sensitivity of 0.5 mV and 2.8 mV respectively, a paced atioventricular interval (pAVI) of 180 ms, and a sensed atioventricular interval of 150 ms. The post atioventricular blanking period (PAVBP) was 60 ms. In the first day after implantation, 12-lead electrocardiography showed episodes of pseudofusion, leading to suspicion of pacemaker malfunction (Fig. 1).

\section{2. Commentary}

Careful analysis of the electrocardiograph (Fig. 1) reveals narrow and similar QRS complexes in all the beats, showing that they are all intrinsic QRS complexes. Some intrinsic P waves are also visible at the ST-T segment (after R3, R7, R11, and R15); these are the beats without any spike before them. The baseline rhythm is therefore an accelerated junctional rhythm. Some atrial spikes generate P waves (a P wave is clearly visible in R6, especially in lead V1, and with careful attention, it may be seen in beats R1, R4, R5, R8, R9, R12, R13, and R16). These points rule out atrial lead dislodgment into the right ventricle or reverse connection of the atrial and ventricular leads.

In R2, R6, R10, and R14, the first stimuli are from atrial pacing, and existent atrial activity is hidden within the QRS complex. The second stimuli are from ventricular pacing that failed to result in ventricular depolarization (failure to capture). This phenomenon may occur in some situations such as with an elevated pacing threshold, lead dislodgement, or lead fracture. A sufficient output will also fail to capture if it occurs in the physiological refractory period of a spontaneous depolarization because of undersensing.

In R2, R6, R10, and R14, a very short interval between atrial and ventricular spikes is seen, which is because of the ventricular safety pacing (VSP) parameter being activated in the device. In Medtronic devices, when this option is on, a sensed signal during
Fig. 1. Electrocardiograph recorded on the first day after implantation. The pacemaker was programmed to the DDDR mode, with a lower rate limit of 60 bpm. Sixteen continuous beats are marked as R1–R16.

Fig. 2. The programmed pAVI is the time between a paced atrial beat and the subsequent delivery of the ventricular stimulus. This interval begins with a blanking period and is preceded by the VSP window. Any ventricular sensed event that occurs within this period will result in a ventricular pacing at the end of this window. In this example, the intrinsic ventricular beat, sensed after an atrial pacing artifact during VSP window, resulted in delivering a ventricular pacing artifact early at the end of the programmed VSP. pAVI = paced atrioventricular interval; VSP = ventricular safety window; AP = atrial pacing; VS = ventricular sensing; VP = ventricular pacing.
the VSP window and after PAVBP triggers ventricular stimulation at the end of the VSP window (Fig. 2). This feature is designed to prevent ventricular inhibition caused by crosstalk of the atrial spike [1]. If an intrinsic ventricular beat happens exactly after PAVBP, and the pAVI delay setting is prolonged, a spike on the T wave may be induced. However, if the safety pacing is on, pAVI is shortened to 110 ms, avoiding cross talk and the spike on the T wave. In these beats, as the rate of the underlying rhythm was close to the pacing rate, simultaneous atrial pacing and sensed intrinsic QRS caused earlier ventricular pacing. As these stimuli followed too close to the preceding intrinsic QRS complex, they occurred during the ventricular myocardial refractory period and did not produce any QRS complexes.

In other beats (R1, R4, R5, R8, R9, R12, R13, and R16), the intrinsic QRS occurred a little after the atrial spike and were sensed by the ventricular lead after VSP, leading to the inhibition of the ventricular output.

In this case, there was no evidence of atrial or ventricular lead dislodgement on chest radiography and pacemaker analysis was normal. By increasing the lower rate limit of the pacemaker to 65 bpm, the pacemaker suppressed the intrinsic rhythm, following which electrocardiography showed atrial–ventricular sequential pacing.

Conflict of interest

None of the authors have any conflicts of interest to disclose.

Reference

[1] Lloyd MS, El Chami MF, Langberg JJ. Pacing features that mimic malfunction: a review of current programmable and automated device functions that cause confusion in the clinical setting. J Cardiovasc Electrophysiol 2009;20:453–60.