CASE REPORT

Atrioventricular synchrony in the background of ventricular noise and undersensing

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The 24-hr electrocardiogram (ECG) interpretation in patients with double-chamber pacemakers may be challenging. The difficulty increases if not well-known pacemaker algorithm and device malfunction coexist. We show atrial synchronization pace (ASP) in a patient with ventricular lead damage. We provide detailed description of electrocardiogram and intracardiac electrogram. ASP may confuse 24-hr ECG monitoring interpretation, especially in patients with ventricular lead dysfunction.

KEYWORDS

atrial synchronization pace, electrocardiogram, lead failure

1 | CASE REPORT

An 80-year-old woman, with hypertension, atherosclerosis, and chronic heart failure with preserved ejection fraction, was implanted 9 years ago with Vitatron C60 DR and two bipolar leads (ventricular, active fixation lead, Vitatron Crystalline and atrial, passive fixation lead with preformed J-shape, Biotronik Selox JT 53) for sick sinus syndrome. The pacemaker was programmed to DDD mode at a lower rate (also at night) of 65 beats/min (923 ms), maximum paced atrioventricular (AV) delay—160 ms, mode switching (MS)—auto, MS sensitivity—standard, atrial synchronization pace (ASP) interval—300 ms, ventricular refractory period—260 ms, ventricular blanking on atrial pace—30 ms, atrial blanking on ventricular pace/ventricular sense—150 ms/50 ms, maximum tracking rate—140 beats/min (429 ms). Refined atrial and ventricular pacing were off.

The 24-hr electrocardiogram (ECG) showed dominating atrial paced rhythm, periodically dual chamber pacing with programmed lower rate of 65 beats/min. Episodes of sinus bradycardia and pacemaker inhibition up to 34 beats/min were observed (Figure 1a–c). Ventricular loss of capture (Figure 1a, marked with *) and periods of lack of ventricular pacing after programmed AV delay (Figure 1b, marked with **) were also present. Moreover, intermittent sensing disturbances were suspected—no pacemaker inhibition in reaction to ventricular depolarization and paced atrium before paced ventricle (Figure 1c, marked with ***). Several mechanisms could explain this 24-hr ECG recording.

Pacemaker pacing slower than lower rate limit or no stimulus output may be caused by different clinical situations or device functions (Fuentes, Toquero, Arroyo-Espliguero, & Lozano, 2003; Greenspon, Schwartz, & Breecker, 2015; Ząbek, Małecka, Matusik, Boczar, & Lelakowski, 2015). However, stimulus output with no capture strongly suggests lead-related problem. Performed chest x-ray did not suggest lead dislodgement, while echocardiography did not show any signs of heart perforation.

To investigate that problem, detailed pacemaker check was performed. It revealed that electrical parameters in atrial lead were normal (impedance 700 (Uni) – 750 (Bi) Ω , bipolar P wave sensing was >1.5 mV and atrial pacing threshold was 0.625 V @ 0.4 ms). Ventricular lead failure was confirmed through observation of ventricular lead impedance > 2,000 Ω , fluctuations in ventricular sensing in electrograms (EGMs), and variable pacing thresholds (including lack of ventricular output and capture), especially during forceful movements of left upper limb.

Studied EGMs and ECG revealed ventricular noise (VE and RS markers with no corresponding QRS on ECG), ventricular undersensing, and failure of ventricular capture (Figure 2). In our case in the presence of ventricular noise, two atrial depolarizations (AS, atrial sense) were identified by the pacemaker most probably as premature atrial contractions (PAC) and successive ventricular depolarizations were not sensed because of ventricular undersensing. Therefore, ASP was delivered to restore AV synchrony (Figure 2).

FIGURE 1 The 24-hr holter electrocardiogram (ECG) monitoring. Detailed description in text. *Ventricular loss of capture; **lack of ventricular pacing after programmed atrioventricular (AV) delay; ***no pacemaker inhibition in reaction to endogenous atrial and ventricular depolarization

FIGURE 2 The intracardiac electrogram and electrocardiogram during pacemaker check. Detailed description in text. SP, synchronization pace; VRP, ventricular refractory period; AV, atrioventricular; AS, atrial sense; AP, atrial pace; VS, ventricular sense; VP, ventricular pace; VE, premature ventricular contraction; RS, ventricular sense in refractory period (used for diagnostic purposes); AEGM, atrial electrogram; VEGM, ventricular electrogram

It is important to mention that in dual-chamber pacemakers, the timing is often atrial-based (depending on manufacturer and/or pacemaker settings). However, ventricular timing may be used when loss of AV synchrony occurs, e.g., after premature atrial or ventricular contraction (PAC or PVC), a retrograde P-wave, Wenckebach behavior, or mode switching (*Vitatron C-series reference manual*). In ASP algorithm, a ventricular stimulus output is scheduled at the end of the ventricular escape interval, while atrial stimulus output is applied one AV delay before it (the pacemaker preserves AV synchrony), but after programmed SP interval (counted from atrial event, e.g. PAC or retrograde P wave). Ventricular escape interval may be prolonged (by a maximum of 65 or 250 ms in the case of retrograde conduction) and AV delay may be shortened (it should be a minimum of 80 ms) (*Vitatron C-series reference manual*).

In conclusion, atrial synchronization pace algorithm may confuse 24-hr ECG monitoring interpretation, especially in patients with ventricular lead dysfunction. The patient underwent successful transvenous ventricular lead extraction. Simultaneously, new ventricular lead was implanted and pacemaker replacement was performed. Old atrial lead was functioning properly and was not removed. After complex assessment, the patient was discharged home in good condition.

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Explanation of ASP algorithm was based on *Vitatron C-series reference manual*.

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