Contents lists available at ScienceDirect

Journal of Arrhythmia



journal homepage: www.elsevier.com/locate/joa

Case Report

Loss of cardiac resynchronization therapy in a patient with a biventricular implantable cardioverter-defibrillator

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ARTICLE INFO

ABSTRACT

Article history: Received 22 June 2017 Received in revised form 1 August 2017 Accepted 23 August 2017 Available online 30 September 2017

Keywords: Cardiac Resynchronization Therapy Heart failure Inhibition of pacing Left ventricular sensing Loss of resynchronization therapy Here, we discuss the case of a man with a history of ischemic cardiomyopathy and cardiac resynchronization therapy defibrillator implantation, who presented to emergency department with decompensated heart failure due to the loss of resynchronization therapy. The reason for the malfunction was left ventricle upper rate interval lock-in due to inappropriate programming of the device. © 2017 Japanese Heart Rhythm Society. Published by Elsevier B.V. This is an open access article under the

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1. Case presentation

A 75-year-old man with a history of ischemic cardiomyopathy and cardiac resynchronization therapy defibrillator (CRT-D) implantation presented to the emergency department with decompensated cardiac failure.

He had undergone CRT-D implantation in 2010 to treat severe left ventricle (LV) dysfunction with a QRS width of 160 ms and left bundle branch block (LBBB) morphology.

In 2016, the original CRT-D generator was exchanged for an Ilesto 7 HF-T (Biotronik SE & Co. KG, Berlin, Germany) due to battery depletion. Additionally, 3 months prior to hospital presentation, he had undergone atrioventricular node ablation because of frequent episodes of atrial fibrillation. Two months later, he underwent an unsuccessful attempt of ventricular tachycardia (VT) ablation for frequent episodes of sustained monomorphic VT, with a cycle length of 570 ms, despite taking antiarrhythmic medications.

Fig. 1 shows the electrocardiogram (ECG) and device interrogation data at the time of presentation to the emergency department. A rhythm histogram at that time revealed that biventricular pacing was occurring only 21% of the time. Table 1 outlines the device parameters present at hospital presentation. To tackle the problem of inadequate biventricular pacing, we changed

http://dx.doi.org/10.1016/j.joa.2017.08.007

some of the device parameters. Table 2 outlines the modified device parameters.

Fig. 2 shows ECG and device interrogation data one week later, indicating improvements. Furthermore, a 1-month follow-up evaluation performed with home monitoring showed 100% biventricular pacing was present, with significant improvement of the patient's symptoms.

2. Discussion

With widespread usage of devices with LV sensing parameters, a described reason for loss of resynchronization is LV upper rate interval (LVURI) lock-in. This phenomenon occurs when the LV upper rate has not been programmed higher than the right ventricle (RV) upper rate. In our patient's device, for example, both these rates were set at 90 bpm (LVURI: 667 ms, RVURI: 667 ms) [1,2].

LV T wave protection is a parameter whose function is to prevent LV pacing into the vulnerable period of left ventricle. Therefore, when it is on, the device can sense LV premature beats and prevent LV pacing into the vulnerable period with consequent VT or VF induction [1].

Apart from pacemaker time cycling, which is RV-based, CRT devices with LV sensing options have an important LV based-time parameter, which is LVURI. LVURI is the period of time that is started by LV events and in which LV pacing cannot happen. This interval is programmable and corresponds to the programmed maximum trigger rate. RVURI and LVURI can be

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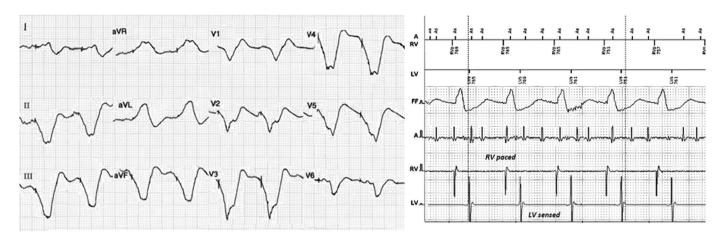


Fig. 1. Left- ECG shows wide QRS complexes with LBBB pattern morphology and bipolar pacing spike in front of each QRS complex without obvious P-waves compatible with right ventricular pacing during atrial fibrillation rhythm. **Right-** Marker channels and intracardiac signals from the atrium (A), far field (FF), RV, and LV shows atrial fibrillation with RV paced and LV sensed events.

Table 1

Device parameters at the time of presentation.

Mode	VVIR, biventricular pacing
First paced chamber	LV with interventricular delay of zero
Lower rate	70 bpm
Upper sensor rate	90 bpm
Sensor threshold/gain	Medium/Medium
LV sensing and pacing	Unipolar
LV refractory period	250 ms
LV T wave protection	On
LV triggering	On
Maximum trigger rate	90 bpm
Tachycardia detection	On
VT ₁ /VT ₂ /VF definition	100/200/240 bpm

Table 2

Modified device parameters.

Mode	VVIR, biventricular pacing
First paced chamber	LV with interventricular delay of zero
Lower rate	70 bpm
Upper sensor rate	90 bpm
Sensor threshold/gain	High/Low
LV sensing and pacing	Unipolar
LV refractory period	250 ms
LV T wave protection	On
LV triggering	On
Maximum trigger rate	100 bpm
Tachycardia detection	On
$VT_1/VT_2/VF$ definition	103/200/240 bpm

programmed individually and only if one of them is shorter than the VT detection rate, will conflict be visible to the programmer [2].

LV pacing always starts an LVURI, regardless of RV pacing offset and/or LV T-wave protection programming. The difference is about LV sensed events. When the left ventricle T-wave protection (LVTP) function is on, LV sensed events will start an LVURI, but when LVTP is programmed off, then the device cannot sense LV events and instead sensed RV events will initiate and reset an LVURI [1,2]. LVURI should be programmed shorter than the RVURI, and the difference should be sufficient in order to compensate for the interventricular delay [2]. Otherwise, it can lead to a form of desynchronization arrhythmia, which is characterized by RV pacing followed by an LV sensed event and loss of LV pacing (like in our patient).

The arrhythmia can be initiated by a single loss of LV capture or by ventricular premature beats (VPBs) (Fig. 3), and is then perpetuated (LVURI lock-in) until a pause from a VPB or a decrease in the RV pacing rate can restore biventricular pacing [2].

In our patient, the problem began after changing of the VT₁ detection rate. Because the patient had frequent episodes of VT, which could be easily terminated with antitachycardia pacing, the VT₁ detection zone had been defined as 100 bpm to 200 bpm, with the upper trigger rate decreased to 90 bpm to prevent programmer conflict. In this patient, the interventricular delay (IVD) was at least 140 ms, and the LVURI had been programmed at 90 bpm/min. Therefore, whenever the sensor increased the pacing rate to more than 75 bpm, this arrhythmia could have happened after the occurrence of VPBs or loss of capture in the left ventricle, and continued on until another VPB could terminate it or until the RV pacing rate fell.

To tackle this problem temporarily (before another ablation procedure), we changed the upper trigger rate to 100 bpm and the VT₁ detection zone to 103 bpm to 200 bpm. On the other hand, as the patient has a sedentary lifestyle and we could not program the maximum trigger rate high enough to compensate for the IVD, we changed the sensor threshold to a less responsive status to maintain resynchronization capabilities, on the expense of a limited sensor-driven rate range.

In conclusion, this case shows an important mechanism of desynchronization in patients with CRT devices with the ability of LV sensing, and emphasizes the importance of proper programming of a device. Overall, in devices with LV sensing parameters—for example, devices from Biotronik (Berlin, Germany) and Boston Scientific (Marlborough, MA, USA)—LV maximum trigger rate should be programmed significantly higher than the maximum sensor rate in order to compensate for the IVD after the occurrence of a desynchronizing event.

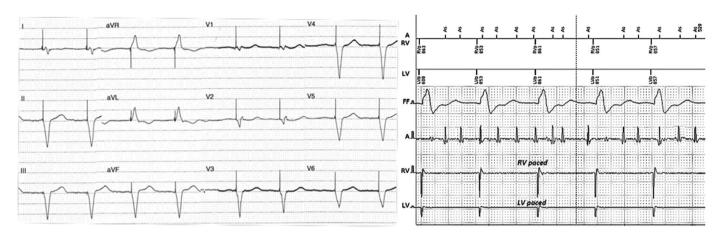


Fig. 2. Left- ECG shows narrower QRS complexes with right bundle branch block pattern morphology and unipolar pacing spike in front of each QRS complex without obvious P-waves compatible with biventricular pacing during atrial fibrillation rhythm. **Right-** Marker channels and intracardiac signals from the atrium (A), far field (FF), RV, and LV shows atrial fibrillation with biventricular pacing (RV paced and LV paced events).

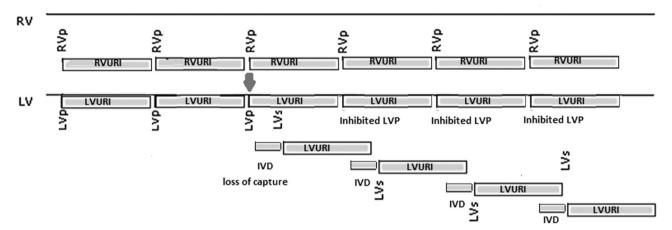


Fig. 3. Initiation of LVURI lock-in after loss of capture in the LV (arrow). LV pacing (LVp) cannot capture LV, but it starts a LVURI. RV pacing (RVp) captures the RV and after IVD and conduction to the left ventricle, the LV signal is sensed (LVs) and restarts LVURI. The RVp is released without an accompanying LVp because the LVp falls into an ongoing LVURI. The inhibited LVp restarts another LVURI and, again after RVp and conduction to the LV, the LVs resets the LVURI, and the cycle will continue.

Disclosures

RS declare no conflicts of interest related to this study.

Conflicts of interest

All authors declare no conflicts of interest related to this study.

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