CASE REPORT

Inhibition of left ventricular stimulation due to left ventricular lead failure and the left ventricular T-wave protection algorithm in patient with cardiac resynchronization therapy and pacemaker dependency

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The electrocardiogram (ECG) interpretation in patients with cardiac resynchronization therapy (CRT) is often a perplexing problem. The difficulty in the device evaluation increases in the presence of unfamiliar timing cycles and a lead dysfunction. Authors describe a special function of a Biotronik CRT devices called the left ventricle T-wave protection (LVTP), and demonstrate its behavior in a patient with left ventricular (LV) lead failure. This report shows that sometimes it might be difficult to understand the loss of resynchronization in 12-lead ECG when LVTP feature is on, and a malfunction of left ventricular lead sensing occurs.

KEYWORDS

cardiac resynchronization therapy, ECG, inhibition of LV stimulation, lead failure, left ventricular T-wave protection

1 | INTRODUCTION

The analysis of 24-h electrocardiogram (ECG) monitoring and 12-lead ECG in patients with cardiac implantable electronic devices might be difficult as it requires a thorough understanding of a variety of mode-switching algorithms, complex timing cycles, and their interrelationships. An interpretation is particularly complicated in the presence of unfamiliar algorithms and a coexisting lead dysfunction (Matusik, Ząbek, Matusik, Małecka, & Lelakowski, 2017). The ECG recordings we present in this article were obtained from a patient with the CRT device in which left ventricular T-wave protection (LVTP) function was turned on, and an intermittent sensing dysfunction occurred.

2 | CASE PRESENTATION

A 60-year-old pacemaker-dependent patient had a dual-chamber pacemaker implanted on the left side of the chest 8 years ago for a complete heart block. Atrial and ventricular Vitatron Cristalline active

fixation leads were fixed in right atrial appendage (RAA) and right ventricular apex (RVA), respectively. Due to congestive heart failure development, New York Heart Association (NYHA) class III symptoms and left ventricular ejection fraction (LVEF) of 35%, an upgrade procedure from DDD pacemaker to CRT-P device was performed 3 years ago. The left bipolar ventricular (LV) lead—St. Jude Medical Quickflex 1258T-86—was positioned in the lateral vein, and the leads were connected with Biotronik Entovis HF-T CRT-P device. With CRT and > 98% of cumulative biventricular pacing, LVEF increased to 45% and the NYHA scale improved to Class I. Patient was CRT responder. This, however, was followed by the failure to capture of the LV lead which was then repaired with A1-N adapter. For that reason LV output polarity was programmed to unipolar mode from LV tip to can with correct capture. During the index hospital admission 4 months later patient presented with heart failure aggravation.

The parameters were as follows: mode DDDR, basic rate = 60 beats per minute (bpm), upper rate response = 120 bpm, ventricular pacing–biventricular (BiV), ventricular pacing triggering–right ventricular (RV) sensing, VV delay after Vp and VV delay after Vs = 0 msec,

RV and LV upper rate = 140 bpm (upper rate interval = 429 msec, RVURI = RV upper rate interval, LVURI = LV upper rate interval), LVTP = on, initially paced chamber = LV, postventricular atrial refractory period (PVARP) = 265 msec (400 msec after a ventricular premature complex [VPC]), RV refractory period = 250 msec, LV refractory period = 200 msec.

The 12-lead ECG showed sinus rhythm at 62 bpm and ventricular paced rhythm after a programmed sensed atrioventricular interval. However, a noticeable variability in paced QRS morphology was observed (Figure 1). The change in paced QRS morphology raised the suspicion of an inadequate CRT delivery.

A subsequent device interrogation revealed the following findings: nearly 100% of RV pacing, a decrease in the cumulative percentage of BiV stimulation to 81.3%, appropriate parameters of atrial and RV lead (bipolar P wave sensing -2.7 mV, unipolar atrial lead impedance -468Ω and atrial pacing threshold 0.4 V @ 0.4 msec; bipolar RV wave sensing -17.0 mV, bipolar RV lead impedance -526Ω and RV pacing threshold 0.7 V @ 0.4 msec). The LV lead failure was confirmed upon the observation of ventricular fluctuations in left ventricular sensing in electrograms (EGMs), especially during forceful movements of the left upper extremity (Figure 2). In unipolar configuration LV lead impedance ($1,443$ – $1,774 \Omega$) and pacing threshold performed in VOO mode (1.5 V @ 1.25 msec) were normal.

Figure 2 shows the analysis of ECG and EGM strips. The intrinsic rhythm is the sinus rhythm at 62 bpm which triggers a paced ventricular response. The majority of paced QRS complexes are narrow (approximately 130 ms), positive (dominant R) in lead V_1 and the frontal QRS plane axis points to the left superior quadrant. Paced QRS complexes marked with * are much wider (approximately 180 msec), negative in lead V_1 (QS) and axis deviation remains in left superior quadrant. Device interrogation and EGM analysis revealed that narrow QRS pattern shows uncomplicated biventricular pacing, broad QRS complexes represent ventricular pacing from RVA and a lack of LV stimulation (marked with *). There was no evidence of ineffectual LV capture episodes.

The mechanism which explains the intermittent loss of resynchronization is an algorithm LVTP set to on. In Biotronik CRT devices, the LVURI controls the delivery of LV stimulation (Barold & Kucher, 2014). LVURI is started by ventricular sensing, either via LV channel when LVTP is turned on, or via RV channel when LVTP is turned off. If LVTP is programmed to be off, LVURI will start upon LV paced events, RV paced events and RV sensed events, whereas LV sensed events are inactive and do not influence any of the LV timing cycles. On the other hand, when LVTP is programmed to be on, all LV paced and sensed events will start the LVURI regardless of which RV events have been detected by the device. Each LV event terminates LVURI in progress

FIGURE 1 ECG. Sinus rhythm at 62 bpm and biventricular pacing after a programmed sensed atrioventricular interval. Two patterns of paced QRS complexes with different width and morphology are visible. Narrower QRS complexes (*) resemble a typical uncomplicated biventricular pacing, whereas broader QRS complexes (**) resemble a typical mono-chamber right ventricular apical stimulation. This ECG strip presumably depicts a malfunction of the left ventricular pacing

FIGURE 2 The intracardiac electrogram and electrocardiogram during pacemaker evaluation. The electrical noise originating from LV lead is visible in LV marker channel and LV electrogram. The inhibition of LV pacing is caused by LV lead oversensing and restarting of LVURI (x). Upper panel: A, RV, LV—atrial, right ventricular and left ventricular marker channel; bottom panel: A, RV, LV—atrial, right ventricular and left ventricular electrogram. AS, atrial sense; VRp, right ventricular pacing; LVp, left ventricular pacing; RVs, right ventricular sensing; LVs, left ventricular sensing; LVrs, left ventricular sensing in left ventricular refractory period; the red color shows LVURI timing cycles during which the LV stimulation was inhibited. See text for details

and reinitiates a new LVURI which in effect produces overlapping LVURIs. Importantly, no LV stimulus can be released during the LVURI. Thus, nonsustained LV lead noise associated with lead fracture potentials and its malfunction caused LV lead oversensing and a recurrent inhibition of LV pacing.

3 | DISCUSSION

Interpretation of the presented ECG strip is a challenging task. We performed differential diagnosis according to the algorithm presented by Barold & Herweg, (2011). Ventricular fusion with the intrinsic QRS complex was ruled out as our patient had complete heart block and was pacemaker-dependent. Furthermore, we could exclude anodal stimulation as LV lead paced in unipolar mode from LV tip to generator can. Other causes which could have produced negative QRS complex in lead V_1 are LV loss of capture, LV lead dislocation, pronounced LV latency and LV intramyocardial disorder. Device interrogation demonstrated that LV capture was correct, however there was LV oversensing and presence of complex CRT timing cycles which were the cause for intermittent BiV pacing loss.

Biotronik and Boston Scientific are the only CRT devices (pacemakers and defibrillators) capable to sense both RV and LV. LV sensing was designed primarily to prevent pacing during the LV vulnerable recovery period potentially inducing a ventricular

tachyarrhythmia. It works by inhibiting the release of an LV stimulus following a sensed LV event. Inhibition of LV pacing in both manufacturers occurs during a programmable period. This feature is referred to as LVTP in Biotronik devices and Left Ventricular Protection Period (LVPP) in Boston Scientific devices. Furthermore, LV sensing can provide useful diagnostic data and permit the diagnosis of dissimilar ventricular rhythms or tachyarrhythmias involving the RV and LV (Barold et al., 2013).

The available literature provides numerous circumstances when LVTP or LVPP affects some of the timing cycles related to antibradycardia pacing and under special circumstances inhibits LV pacing. These include T-wave oversensing by the LV channel and far-field atrial sensing by the LV channel before RV activity during sinus rhythm and atrial tachyarrhythmia (D'Ivernois & Barold, 2015). Moreover, when LVURI interval is longer than RV upper tracking interval resynchronization stops due to repeated LVURI restarting (Barold & Kucher, 2014; Barold, Mello Porto, & Kucher, 2014).

The cessation of LV pacing due to artifacts sensed by LV channel when LVTP is programmed on, has not been reported in the literature to date. Artifacts are divided into external noise / electromagnetic interference, lead / connector noise and muscle noise (Powell et al., 2012). Typical nonphysiological lead fracture potentials are characterized by the following features: have high frequency, occur intermittently, are highly variable in amplitude and frequency, appear in one channel, saturate the sensing amplifier, and are unrelated

to the cardiac cycle (Swerdlow, Kalahasty, Ellenbogen, & , 2016). Oversensing with normal pacing impedance is the initial electrical abnormality in majority of lead failures (Swerdlow et al., 2016; Ząbek, Małecka, Matusik, Boczar, & Lelakowski, 2015). In our case, if LVTP algorithm was turned off the lead noise would not result in a transient loss of resynchronization, a significant decrease in percentage of BiV pacing, and heart failure exacerbation. On the other hand, if not for LVTP programmed to be on, the lead failure might not have been detected in the absence of other pacing and sensing abnormalities. In Biotronik and Boston Scientific CRT devices it is worth to measure LV sensing in order to detect far-field oversensing that is, atrial, T-wave or artifacts and manage LV sensitivity. In conclusion, LVTP feature and LV sensing provide a useful diagnostic tool for the evaluation of CRT function.

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How to cite this article: Ząbek A, Małecka B, Dębski M, Boczar K, Lelakowski J. Inhibition of left ventricular stimulation due to left ventricular lead failure and the left ventricular T-wave protection algorithm in patient with cardiac resynchronization therapy and pacemaker dependency. *Ann Noninvasive Electrocardiol*. 2018;23:e12473. <https://doi.org/10.1111/anec.12473>