CASE REPORT



Suboptimal biventricular pacing. What is the mechanism?

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Abstract

Suboptimal biventricular pacing has deleterious effects on patients with cardiac resynchronization therapy. We describe a unique case of suboptimal biventricular pacing and our approach to overcome it.

KEYWORDS

cardiac resynchronization therapy defibrillator, left atrial far field sensing, left ventricular lead, left ventricular protection period, left ventricular sensing

INTRODUCTION 1

Studies have shown that biventricular pacing of $\ge 97\%^1$ was associated with reduction in recurrent hospitalisation rate and all-cause mortality in patients with systolic heart failure. We describe a unique case of suboptimal bi-ventricular pacing and our approach to overcome it.

2 | CASE REPORT

The patient was a 67-year-old man with a history of dyslipidemia, diabetes mellitus, coronary artery bypass graft surgery 8 years ago and

FIGURE 1 Twelve-lead

electrocardiogram shows intermittent loss of biventricular pacing that is marked with * (see text for details)

ischemic cardiomyopathy (left ventricular systolic function of 35%). He was admitted following symptomatic complete heart block with slow ventricular escape rhythm. He underwent cardiac resynchronization therapy defibrillator (CRTD) implantation (Device: Inogen CRT-D G140, Boston Scientific, Natick, MA, USA; Right ventricular lead: Endotak Reliance, Single Coil, 0293/64 cm, Boston Scientific, Natick, MA, USA; Right atrial lead: Ingevity 7741/52 cm, Boston Scientific, Natick, MA, USA; Left ventricular lead: Easytrack 2 IS-1 4543/90 cm, Boston Scientific, Natick, MA, USA). The left ventricular (LV) lead was placed at the basal posterior branch of coronary sinus. The device was programmed DDD 60-130 bpm with sensed and paced atrioventricular intervals of 100 and 130 ms, respectively. Left ventricular-right ventricular (LV-RV) pacing interval was set at



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FIGURE 2 Intermittent far-field atrial sensing (arrow head) by LV lead of a Boston Scientific Inogen G140 CRT-D device. The marker channels are at bottom, A = atrial, RV = right ventricle, LV = left ventricle, AS = atrial sensed, RVP = right ventricle pacing, LVP = left ventricular pacing, Inh-LVP = left ventricular pace inhibited. The ECG lead, electrograms A, RV and LV are above the marker channels. The bar above the LV channel indicates left ventricular protection period (LVPP). The bar above the RV channel indicates sensed atrial ventricular (AV) interval. There was intermittent far-field atrial sensing on the LV channel due to the programmed sensitivity of the LV lead set at 1 mV but the amplitude of the far-field atrial signal was about 0.9-1.0 mV. Following the AS event, far-field atrial sensing is seen on the LV channel (arrowhead) and this led to inhibition of LV pacing (Inh-LVP), corresponding to loss of biventricular pacing (RV pacing only) on surface ECG. Following sensed AV delay timed out (100 ms, gray bar), RV pacing occurred followed by LV depolarization (asterisk), which was registered but not sensed on LV channel. This was due to LVPP (400 ms) that was initiated after far-field atrial sensing (see text for details)

0 ms (simultaneous biventricular pacing). During subsequent followup, his left ventricular pacing was 72% as compared with right ventricular pacing of 99%. An ECG was done (Figure 1). What was the reason of suboptimal LV pacing?

3 | DISCUSSION

The ECG (Figure 1) showed pacing spike followed by wide QRS complex of two distinct morphologies. The 5th and 6th paced QRS complexes showed wide QS complex in V1, superior axis and were wider than the rest of the paced QRS complexes. The 5th and 6th paced QRS complexes represented RV-only pacing with a left bundle block pacing morphology. The other narrower paced QRS complexes represented biventricular pacing in view of the narrower QRS width and small initial upstroke in lead V1. They were not fusion or pseudofusion beats as patient was in complete heart block.

There was biventricular pacing (simultaneous RV and LV pacing, Figure 2) with intermittent RV pacing only (marked with arrow head, Figure 2), which corresponded to wide QRS complex (RV pacing only) on the surface ECG (Figure 2). This was due to sensing of far-field atrial signal by left ventricular lead which inhibited LV pacing (cross talk between LV and atrial channel). The far-field atrial signal sensed on LV lead occurred about 40 ms later than the atrial signal on atrial lead. This was likely due to time taken for atrial depolarization to arrive at the left atrium from the right atrium. There was intermittent inhibition of LV pacing as the LV sensitivity was set at 1 mV (out of box setting) which was similar to the amplitude of the far-field atrial signal (0.9-1.0 mV). RV pacing occurred 20 ms after LV pacing inhibition as the sensed AV interval was programmed at 100 ms. Subsequently, there was a large electrogram seen on the LV channel due to depolarization of LV following RV pacing. However, this electrogram was not sensed due to left ventricular protection period (LVPP) being programmed at 400 ms. LVPP is the period after a paced or sensed LV event when pacing of the LV is inhibited.

The LVPP was primarily designed to inhibit LV pacing during the vulnerable period of LV. As sensing of LV was not used for tachycardia detection, the programming strategies include (a) reducing LV sensitivity (to avoid far-field atrial sensing); (b) turning off LV sensing; (c) programming left ventricular sensing to unipolar configuration (the distal bipole had a more ventricular position, hence was less likely to have far-field atrial sensing). In our patient, we elected to switch off the LV sensing as the LV signal amplitude was low. Subsequent device interrogation on follow-up showed a biventricular pacing of 99%.

4 | CONCLUSIONS

In our case, the position of the LV lead close to the mitral annulus predisposed to the sensing of far-field left atrial signal. It is important for implanters to be aware of this as a potential cause of intermittent loss of biventricular pacing in particular when the LV sensing is turned on.

CONFLICT OF INTEREST

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

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 Ruwald AC, Kutyifa V, Ruwald MH, Solomon S, Daubert JP, Jons C, et al. The association between biventricular pacing and cardiac resynchronization therapy-defibrillator efficacy when compared with implantable cardioverter defibrillator on outcomes and reverse remodelling. Eur Heart J. 2015;36(7):440–8. How to cite this article: Tan VH, Yeo C, Wong K. Suboptimal biventricular pacing. What is the mechanism? *J Arrhythmia*. 2019;35:673–675. https://doi.org/10.1002/joa3.12207