Interaction between left bundle branch area pacing lead and defibrillator lead: A case report

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Introduction

Heart failure patients with reduced left ventricular (LV) systolic function and wide left bundle branch block can benefit from biventricular pacing.[1] However, factors such as anatomical limitations, phrenic nerve stimulation, and/or high pacing thresholds may hinder LV lead implantation. Although no large randomized controlled studies comparing left bundle branch area pacing (LBBAP) with biventricular pacing have been published to date, clear benefits of LBBAP as cardiac resynchronization therapy (CRT) have been suggested.[2–4] However, while biventricular pacing can be easily combined with a shock lead in patients indicated to receive a defibrillator, the combination of LBBAP with a right ventricular apical shock lead can be more difficult than anticipated. We present an interaction between a LBBAP lead and a defibrillator lead positioned in the right ventricular apex.

KEY TEACHING POINTS

- Conduction system pacing by means of left bundle branch area pacing (LBBAP) is possibly a feasible approach as an alternative for biventricular cardiac resynchronization therapy.
- When an LBBAP lead and an implantable cardiac defibrillator (ICD) lead are combined, there is a risk of possible dangerous oversensing on the LBBAP lead, resulting in pacing inhibition, as well as oversensing on the shock lead, leading to an inappropriate shock.
- We report a case of LBBAP lead interaction when combined with an ICD lead, owing to tricuspid valve closure. Careful analysis of the electrogram and the position of the leads in multiple views during implantation should be made when a shock lead is combined with an LBBAP lead.

Case report

An 80-year-old male patient with nonischemic dilated cardiomyopathy, a left bundle branch block (QRSd 162 ms), and a reduced ejection fraction (ejection fraction = 35%) despite optimal medical treatment was referred for CRT and defibrillator implantation in secondary prevention. The patient had several episodes of near syncope with documented concomitant polymorphic ventricular tachycardia.

In the absence of coronary sinus side branches, conventional biventricular pacing was not deliverable and LBBAP was opted as bailout therapy.[5] LBBAP lead implantation was achieved by using a stylet-driven pacing lead (Solia S60; Biotronik, Berlin, Germany) and a dedicated delivery sheath (Selectra 3D-55-39cm; Biotronik, Berlin, Germany), as previously described.[6,7] During implantation, capture of the conduction system was confirmed by the following: presence of an incomplete right bundle branch block morphology in V_1 with a short LV activation time (LV activation time = 90 ms) stable at differential high and low output, and a full correction of the left bundle branch block with a meaningful reduction of the QRS duration (paced QRS = 136 ms). Next, the shock-lead (Plexa S65, Biotronik, Berlin, Germany) was placed in a right ventricular apical position, while the atrial lead was finally placed in the right atrial appendage. The LBBAP lead was connected to the LV port of the CRT defibrillator device (Acticor 7 HF-T; Biotronik, Berlin, Germany). The shock lead and the atrial leads were connected to the designated ports. No defibrillation threshold test was performed. In the operating theater, both the right atrial and shock leads demonstrated normal sensing, impedance, and threshold values (2.9 mV, 540 ohms, 0.5 V / 0.4 ms; and 18.8 mV, 657 ohms, 0.8 V / 0.4 ms, respectively). The LBBAP lead was tested in unipolar configuration and also demonstrated normal sensing and pacing characteristics (7.1 mV, 423 ohms, 0.3 V / 0.4 ms).

KEYWORDS LBBAP; Left bundle branch area pacing; Physiologic pacing; CRT; Complication; Lead interaction
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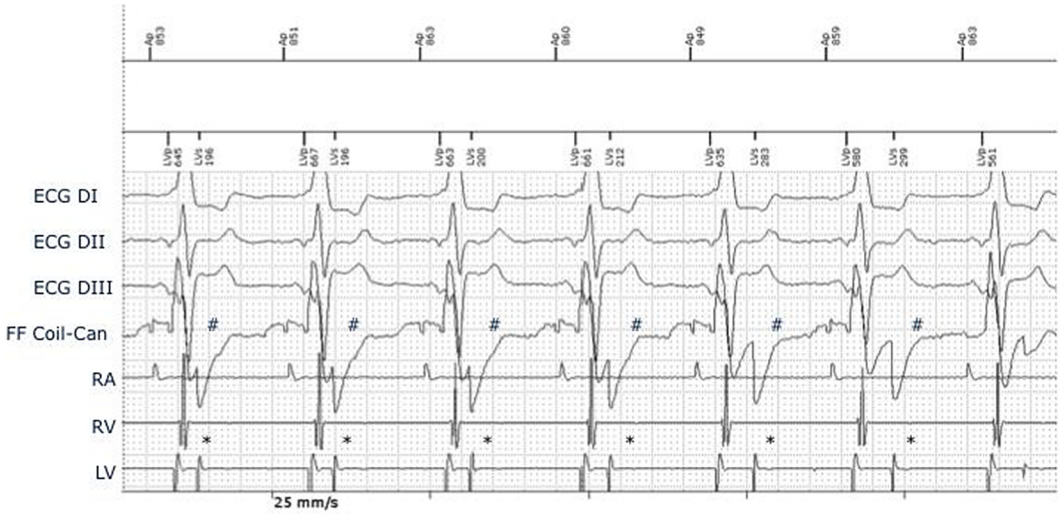

Figure 1 Device in DDD – LV only pacing mode / LV sensing is bipolar. Intermittent sharp oversensing signals (*) recorded on the LV channel with slightly variable LVPace-LVSense intervals. Concomitant artifacts are sensed on the discrimination channel (#). FF coil-can = far-field coil to can; LV= left bundle branch area pacing electrogram; RA = right atrium electrogram; RV = right ventricular (ICD lead) electrogram.

At first device check, the day after implantation, the device was programmed in DDD – LV only pacing mode. The LBBAP lead sensing was set bipolar (LV1-LV2) while the pacing remained programmed unipolar (Tip-Can). Intermittent sharp oversensing signals regularly appeared on the LV channel with slightly variable LVPace-LVSense intervals (Figure 1). There was no corresponding deflection on the surface electrocardiogram. The impedance of the LBBAP lead was unchanged (482 ohms), as well as the thresholds for LBBAP capture and left bundle branch block correction. Characteristics of the shock lead were also unchanged and the shock lead impedance was 70 ohms. Atrial pacing did not interfere with this additional signal. To clearly differentiate both LBBAP and right ventricular (RV) signals, the pacemaker was reprogrammed in biventricular mode with the RV pacing offset 100 ms later than the LBBAP lead (the RV pacing lead being used as backup). This setup was also associated with sharp, noise-like signals of variable amplitude on the LV channel and inappropriate inhibition of the LBBAP (Figure 2A). Concomitant to the additional signal on the LV electrogram (EGM), low-frequency signals of variable amplitude were observed on the "far-field" RV coil-can channel. After reprogramming of the LV sensing polarity to unipolar (Tip-Can), the oversensing on the LV channel immediately disappeared, but noise on the "far-field" coil-can channel remained unchanged. Suspecting an interaction between the implantable cardioverter-defibrillator (ICD) coil and the ring electrode of the LBBAP lead, additional fluoroscopic checks of the leads were performed. No lead dislocation was detected. However, the cinefluoroscopy (Figure 3 and Supplemental Video 1) confirmed intermittent contact between the proximal RV coil and the ring electrode

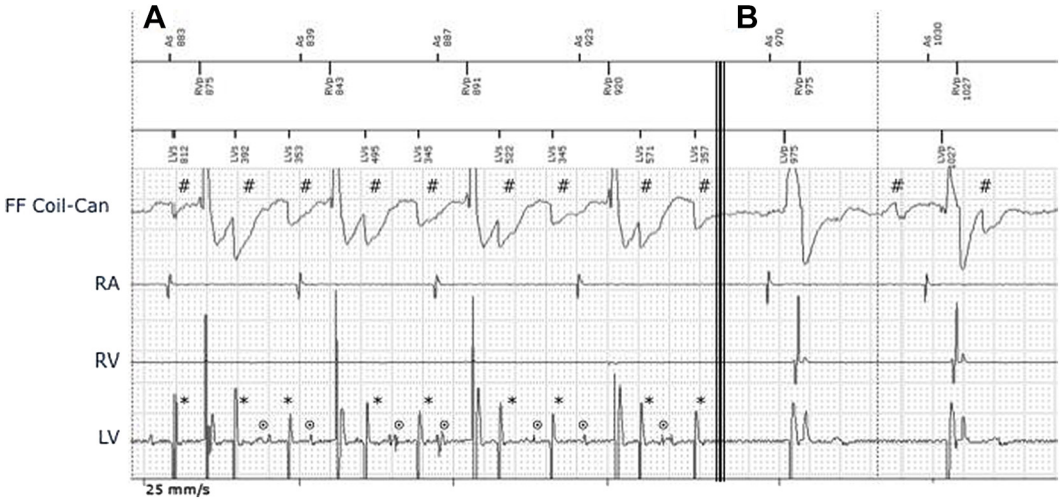

Figure 2 **A:** Device in DDD – Biventricular pacing mode (LV offset 100 ms) / bipolar LV sensing. Noise-like signals of variable amplitude (sensed *, non-sensed ⊙) recorded on the LV channel with inappropriate inhibition of the left bundle branch area pacing and right ventricular backup pacing on the 2 last beats. **B:** Unipolar LV sensing. Missense electrograms disappeared from the LV channel but not from the FF Coil-Can channel (#).

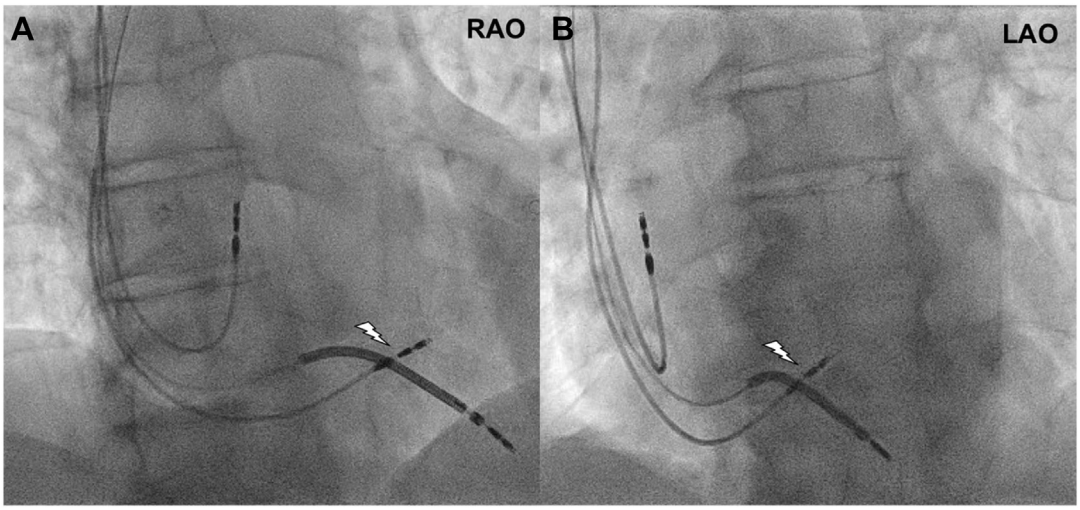

Figure 3 Relative position of the left bundle branch area pacing and shock leads in right anterior (**A**) and left anterior (**B**) fluoroscopic views. Multiple angulation views confirmed the intermittent contact of the ring electrode and the coil following the tricuspid movement.

of the LBBAP lead with every closure of the tricuspid valve, explaining the "missense" EGMs.

Although the most adequate and definite solution consisted of repositioning of the RV lead, the patient refused the revision of the device. As bailout solution, we reprogrammed the device in DDD biventricular RV-sense only mode, with a long VV offset (LV + 100 ms) (Figure 2B), where LV pacing depends on the RV lead sensing. After a follow-up period of 6 months, a remarkable improvement was noted in LV ejection fraction (up to 45%) and patient functional class (NYHA III > NYHA II). The patient being regularly followed by remote monitoring, no report of lead failure was noticed yet.

Discussion

Lead-to-lead interaction is rare but counts among the most dangerous clinical situations for patients implanted with cardiac implantable electronic device. Several publications report on inappropriate sensing and inhibition, inappropriate ICD therapies, noise, insulation breach, and even lead rupture following the repetitive contact between pacing and/or defibrillation leads.[8,9] With the development of conduction system pacing (CSP), cardiac resynchronization can be achieved with only 1 lead instead of 2 leads, as for conventional biventricular pacing. One would therefore expect a lower risk of lead interaction. However, when a defibrillator function is needed, it is still mandatory to add a shock lead to the LBBAP or His bundle lead. In such situation, the risks of lead interaction might increase, as CSP leads are oriented perpendicular and closer to the RV coil. A recent publication already reports on catastrophic complication following LBBAP and ICD lead interaction.[8] In contrast to our case, no EGMs alerted the physician of the possible contact between the 2 leads, and lead fracture was the first clinical manifestation of the interaction. In our case, intraoperative tests of the LBBAP lead were performed using a unipolar configuration where the implanter is blind to EGMs using the ring electrode. Also, far-field (coil-can) EGM was not immediately readable during the procedure, and not routinely scrutinized immediately after implantation. This explains why the close contact between the 2 leads was not immediately recognized. Further, looking at the relative position of the 2 leads using only 1 fluoroscopic orientation can also be misleading and does not always help to suspect the close relation of the 2 leads. Accordingly, multiple angle projection after implant and recording of bipolar (tip-to-ring) signal during implantation may help to early recognize and prevent lead-to-lead interaction. Positioning firstly the ICD apical lead with a lot of slack might also reduce the risk of hooking the shock lead when positioning the LBBAP. Whenever the ICD lead is positioned after the LBBAP, great care should be taken to have the coil placed at an adequate distance from the LBBAP lead insertion site. Finally, we think that this case also illustrates the relevance of the LBBA-Defibrillator concept, where 1 single lead delivers both CSP and defibrillation.[10]

In conclusion, when LBBAP is used as an alternative for biventricular pacing, and is combined with a defibrillator lead, great care should be taken regarding the placement of the RV apical lead. The long-term integrity of the lead and the risk of lead-to-lead interaction need to be put in perspective and evaluated in larger trials.

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Appendix

Supplementary Data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2023.10.026.

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