A case report of far-field P-wave oversensing in left bundle branch area pacing

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Introduction

Since its introduction, left bundle branch area pacing (LBBAP) has gained broad clinical acceptance as an effective way to provide physiological pacing. LBBAP is expected to prevent the deleterious effects of right ventricular pacing and to be an alternative to biventricular pacing. However, experience with the possible complications of this novel technique is still limited. Here, we report the occurrence of far-field P-wave oversensing on the ventricular channel in LBBAP as a cause of presyncope.

Case report

A 68-year-old man with a syncopal attack and second-degree atrioventricular (AV) Möbitz type II block was referred to our hospital for further treatment. The echocardiography showed normal dimensions and a normal left ventricular ejection fraction without other obvious abnormalities.

Implantation of a dual-chamber pacemaker was planned. Because of an anticipated high pacing percentage, conduction system pacing was attempted. First, we positioned an atrial lead (Solia S53; Biotronik, Berlin, Germany) in the right ventricle to function as a backup lead. Next, a ventricular lead (Solia S60; Biotronik, Berlin, Germany) was positioned at the basal right ventricular septum with a dedicated sheath (Selectra 3D-55-39; Biotronik, Berlin, Germany). We used a stylet-driven technique to screw the lead into the septum with continuous monitoring of lead advancement using fluoroscopy and assessment of the unipolar paced QRS morphology on a 12-lead electrocardiogram (ECG).[1] Conduction system pacing was reached at the first attempt, confirmed by the appearance of a terminal R wave in lead V_1 on the electrocardiogram with a V_6-V_1 inter-peak interval of 50 ms (V_6 R-wave peak time 85 ms, V_1 R-wave peak time 135 ms) (Figure 1A). The paced QRS morphology was different than during intrinsic rhythm with a change in QRS axis. There was a negative QRS during pacing in both leads II and III, which suggests left posterior fascicular pacing (Figure 1B and 1C).[2] We did not observe left bundle branch potentials and no QRS morphology transition was detected during threshold testing or pacing at decremental output. Finally, we repositioned the atrial lead to the right atrial appendage.

At implantation, the atrial capture threshold was 0.5 V at 0.4 ms with an impedance of 507 ohms and a P-wave amplitude of 2.0 mV. The ventricular capture threshold was 0.4 V at 0.4 ms with an impedance of 507 ohms and a R-wave amplitude of 4.5 mV. The pacemaker was programmed in DDD mode with a lower rate of 50 beats/min and an upper tracking rate of 130 beats/min. The atrial and ventricular unipolar outputs were 1.4 V and 0.9 V at 0.4 ms, respectively (with Capture Control turned on). The default sensitivity settings were kept with a bipolar sensing configuration and the Automatic Sensitivity Control algorithm turned on (which automatically adjusts the sensitivity according to the last sensed event with a step-wise reduction to a minimal value

KEY TEACHING POINTS

- Syncopal episodes after pacemaker implantation can indicate ventricular oversensing.
- Far-field P-wave oversensing is a known issue in His bundle pacing, but can also occur in left bundle branch area pacing.
- Oversensing should be recognized at implantation. If not corrected for during implantation, possible solutions for the problem are optimizing sensitivity, changing to an asynchronous or triggered pacing mode, or reintervention.

KEYWORDS Pacemaker; Oversensing; Left bundle branch area pacing; Conduction system pacing; Syncope; Atrioventricular block
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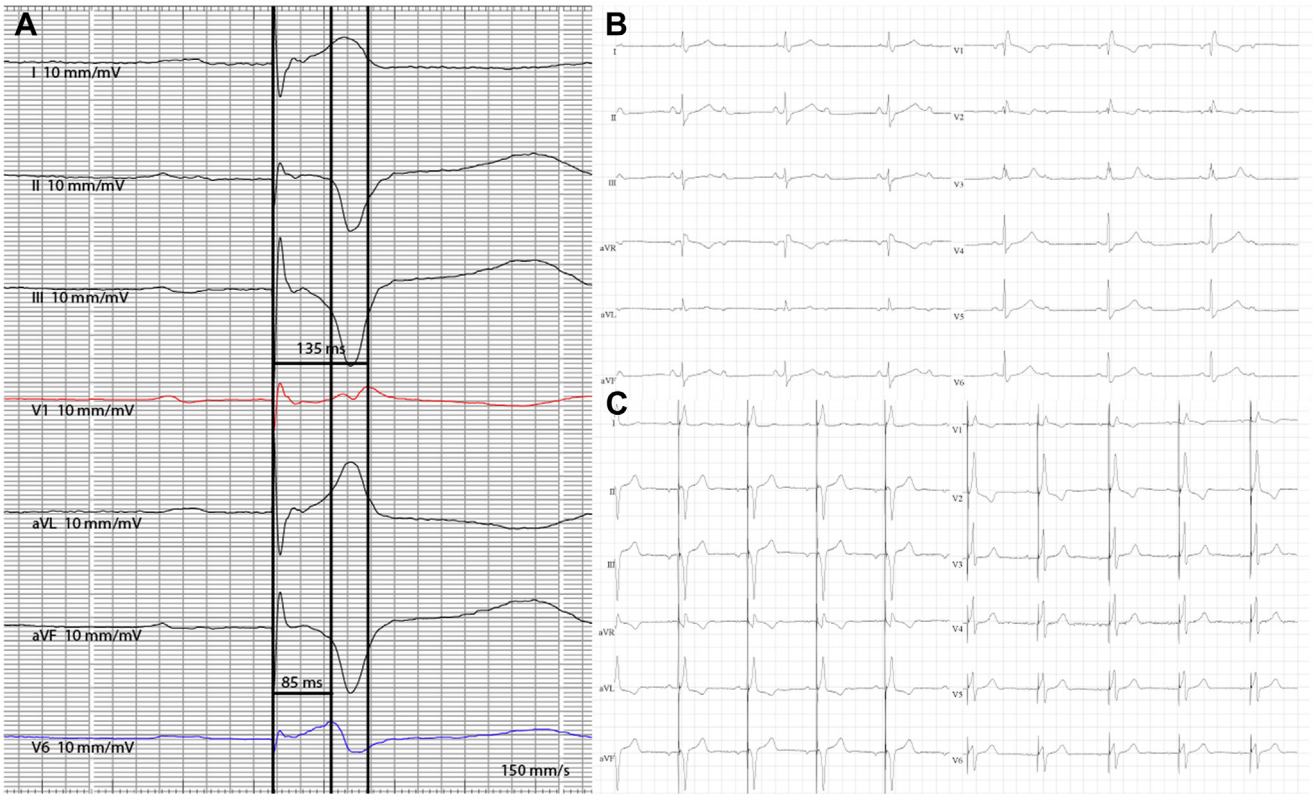

Figure 1 **A:** Paced QRS morphology on the perioperative surface electrocardiogram confirming conduction system pacing with a V_6 R-wave peak time of 85 ms, a V_1 R-wave peak time of 135, and a V_6-V_1 inter-peak interval of 50 ms. **B, C:** Twelve-lead electrocardiogram before (**B**) and after (**C**) pacemaker implantation demonstrating a change from intrinsic right bundle branch block to a QRS morphology consistent with left posterior fascicular pacing.

of 25% of the peak amplitude of this event or a minimal sensing threshold of 2.0 mV in the ventricle).

Seven months after the procedure, the patient reported multiple dizzy spells and 1 near-fainting episode. Device interrogation revealed stable lead measurements with normal pacemaker function and no episodes had been registered in the pacemaker diagnostics. On chest radiography the position of both leads was unchanged (Figure 2A). Provocation maneuvers revealed no oversensing of myopotentials. Twenty-four-hour Holter monitoring was performed, which showed frequent episodes of ventricular asystole for 3–4 seconds owing to total AV block without apparent ventricular pacing spikes, suggesting ventricular oversensing (Figure 2B).

Subsequently, a new device interrogation was performed. Careful analysis of the intracardiac electrocardiograms confirmed intermittent far-field P-wave oversensing on the ventricular channel during both bipolar and unipolar sensing (Figure 2C). The amplitude of the sensed P wave on the ventricular channel was measured and was 1.7 mV, which was near the minimal value of 2.0 mV of the Automatic Sensitivity Control algorithm. The amplitude of the true ventricular signal during bipolar sensing was 7.0 mV (min 6.1 mV, max 7.4 mV). Programming the ventricular sensitivity at a fixed level of 4.5 mV resolved the issue, while keeping a minimal safety margin of 2.0 mV to prevent undersensing.

We reviewed the position of the ventricular lead on transthoracic echocardiography. While difficult to visualize its exact location owing to reverberation artefacts, the lead tip of the ventricular lead appeared to deflect superiorly toward the level of the AV junction, which might suggest inadvertent nonselective His bundle pacing instead of LBBAP (Supplemental Video 1). However, (nonselective) His bundle pacing would not change the QRS axis drastically. Subsequent computed tomography scan revealed a lead tip position 12 mm inferiorly from the insertion point of the septal leaflet of the tricuspid valve and in the posterior third of the interventricular septum, consistent with LBBAP and our suspicion of left posterior fascicular pacing on ECG (Figure 3A and 3B).[3]

Discussion

We report of far-field P-wave oversensing in left bundle branch area pacing, resulting in intermittent inhibition of ventricular pacing and causing presyncope.

Far-field sensing of the atrial activity on the ventricular channel has disastrous consequences if it results in inhibition of ventricular pacing in pacing-dependent patients. If it occurs when the atrial pacing output is sensed on the ventricular channel, this phenomenon is called crosstalk and specific countermeasures protect against this, namely the use of a post–atrial ventricular blanking period and ventricular safety pacing. However, these features are only activated after an atrial pacing stimulus and not after intrinsic atrial activity.

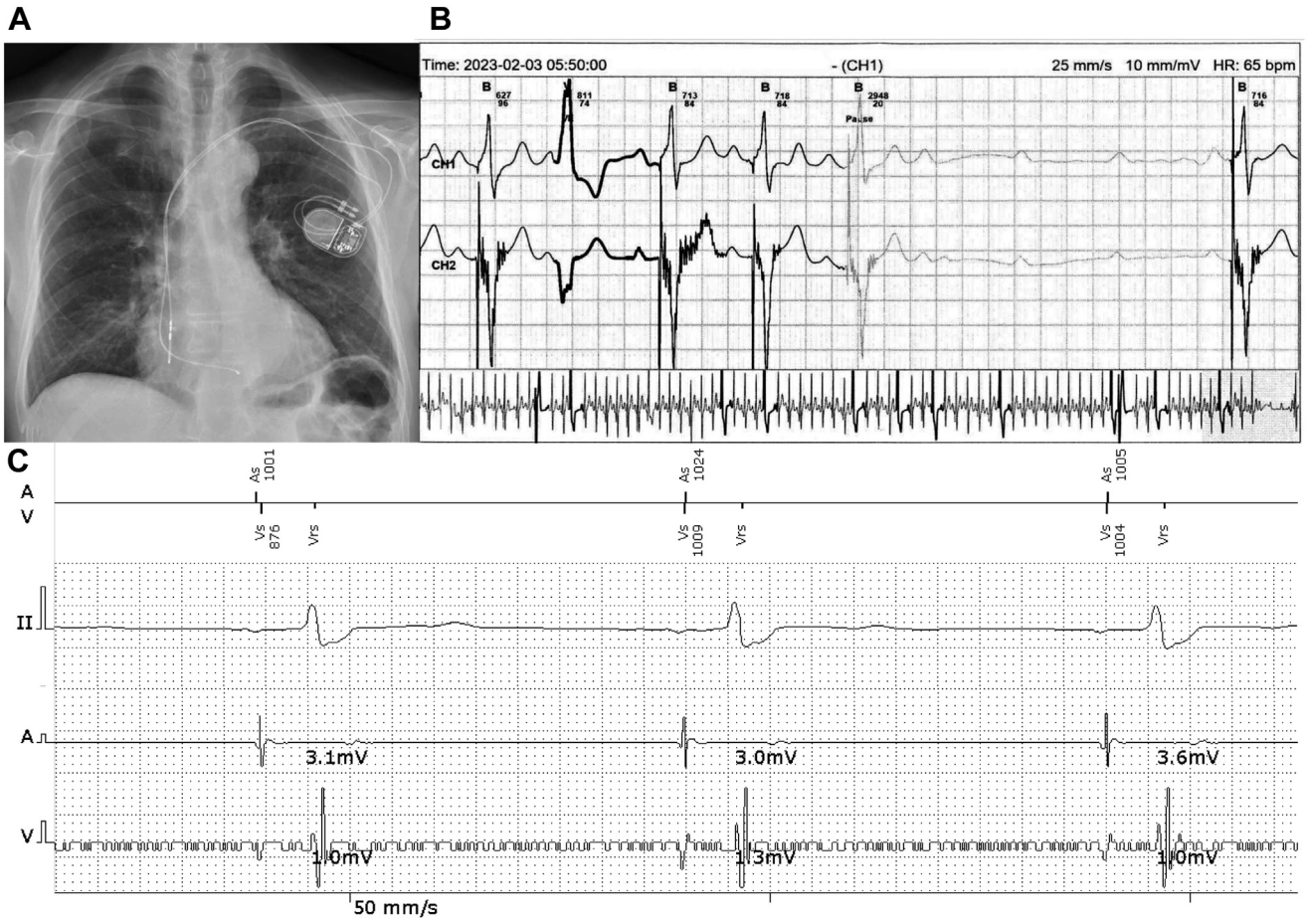

Figure 2 **A:** Anteroposterior chest radiography showing stable lead positions. **B:** Holter tracing demonstrating total atrioventricular block with intermittent absence of ventricular pacing, suggesting ventricular oversensing. **C:** Intracardiac electrogram during a unipolar sensing test revealing episodes of ventricular oversensing owing to far-field sensing of atrial activity. A ventricular-sensed event (Vs) with an amplitude of 1.0–1.3 mV is seen, which coincides with an atrial-sensed event (As) at the moment of the P wave on the electrocardiogram, indicating far-field P-wave oversensing. This is followed by another ventricular-sensed event, which represents the true ventricular activity, evidenced by the concurrent QRS on the surface electrocardiogram and the change on the intracardiac electrogram. This is annotated as a refractory sensed event (Vrs) because it follows the erroneous Vs owing to P-wave oversensing.

Luckily, far-field oversensing of intrinsic activity seldom occurs when the ventricular sensitivity is adequately programmed. Nevertheless, it can happen in certain scenarios, such as dislocation of a ventricular lead to the right ventricular inflow tract, the misplacement of a ventricular lead in the coronary sinus, and the use of an integrated bipolar lead in implantable cardiac defibrillators.[4,5] Additionally, far-field P-wave oversensing can occur in cardiac resynchronization therapy devices with a left ventricular channel capable of sensing. Displacement or malposition of the left ventricular lead toward the AV groove can result in far-field sensing of the left atrial activity and inhibit left ventricular pacing, which impedes adequate biventricular pacing.[6] A related issue is when a lead integrity issue of the ventricular lead at the atrial level causes noise during atrial contraction and mimics far-field P-wave oversensing.[7]

These specific causes of far-field P-wave oversensing are rare, but in His bundle pacing the issue is more common, since the lead is positioned at the atrial or ventricular side of the membranous septum where the atrial activity can more easily be sensed.[8] Therefore, it is recommended in the recent European Heart Rhythm Association consensus paper on conduction system pacing to take care during His bundle pacing to recognize its occurrence and correct for it during implantation.[2]

Lower and more stable capture thresholds have made LBBAP an attractive alternative to His bundle pacing and its rapid adoption has been supported by a small randomized controlled trial and a recent large observational trial comparing LBBAP to biventricular pacing.[9,10] An additional benefit of LBBAP is that far-field P-wave oversensing is less likely to occur compared to His bundle pacing, since the lead is positioned farther away from the atria.

However, our case demonstrates that even in LBBAP we should still keep far-field P-wave oversensing in mind. This is not completely unexpected, since the basal RV septum is targeted for the lead tip insertion. Additionally, in our experience, the intraseptal course of the lead during screwing can be unpredictable. If far-field P-wave oversensing is detected, an alternative lead position should be sought. If it only becomes apparent during follow-up, solutions for this problem are increasing the ventricular sensing amplitude (ie, making

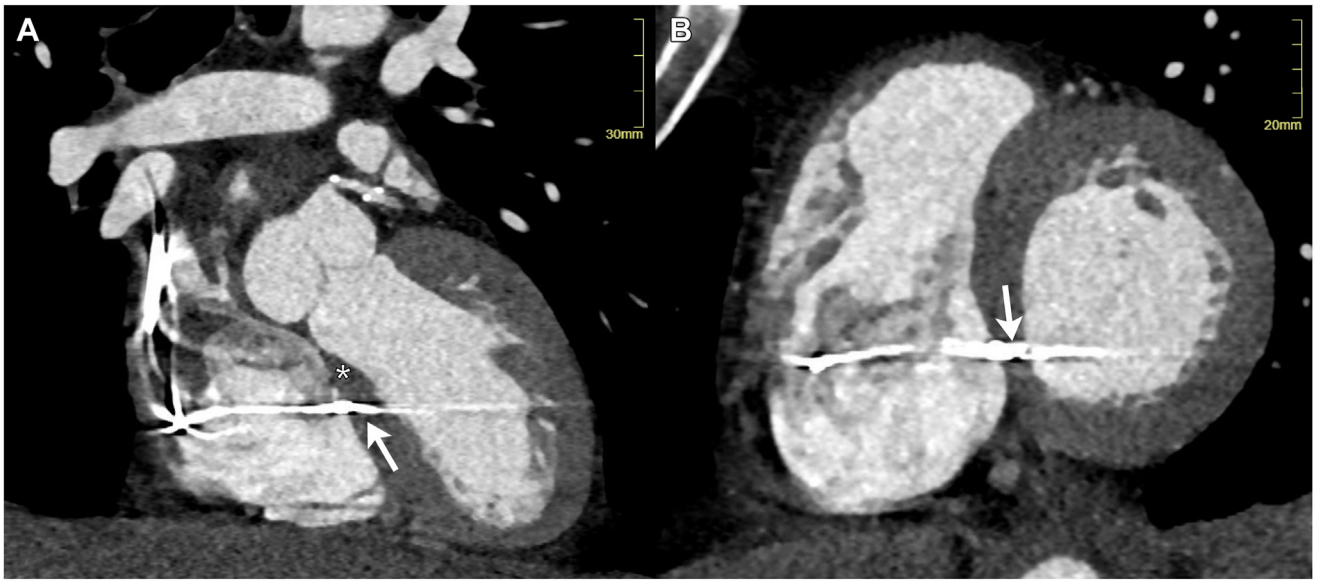

Figure 3 Left anterior oblique cranial (**A**) and short-axis views (**B**) on cardiac computed tomography showing a lead tip position (*arrow*) 12 mm inferiorly from the insertion point of the septal leaflet of the tricuspid valve (*asterisk*) and in the posterior third of the interventricular septum.

the pacemaker less sensitive), changing to an asynchronous or DDT/VVT mode (where sensed events trigger ventricular pacing), or a pacemaker reintervention.

Far-field P-wave oversensing might have been prevented in our case by a lead insertion site closer to the RV apex. Indeed, on cardiac computed tomography a distance of only 12 mm was demonstrated between the lead tip and the insertion of the tricuspid valve on the septum. The optimal technique for LBBAP is still unknown and multiple methods to select the lead insertion site have been described. However, in general it is recommended to aim for at least 15–20 mm between the lead tip insertion site and the tricuspid valve summit in a 20°–30° right anterior oblique view. A smaller distance appears to be associated with more tricuspid valve regurgitation, and in addition the His bundle might be inadvertently stimulated.[2] The use of a right anterior oblique view will also help to detect an unintended atrial orientation of the lead, where the lead tip may be pointing at a 10 to 12 o'clock position instead of pointing at 12 to 2 o'clock. In our case as well, the relatively low R-wave sensing amplitude of 4.5 mV during implantation might suggest nonselective His bundle pacing instead of LBBAP. In LBBAP typically large R waves are observed because the lead is buried deep in the muscular septum. However, as discussed, the paced ECG does not fit with nonselective His bundle capture, since this would not expect to change the QRS axis drastically. The paced ECG morphology and the position of the lead on ECG are more consistent with left posterior fascicular pacing, a type of LBBAP. While in our case far-field P-wave oversensing was seen as a cause of absent pacing spikes, lead dysfunction owing to fracture should be considered as an alternative diagnosis. In stylet-driven LBBAP in particular, delayed conductor fracture has been described.[11] An angulation between the tip and the ring electrode when the latter has not penetrated into the septum might make the lead susceptible to damage from the chronic mechanical stress of the septal contractions.

Conclusion

We report a case of symptomatic far-field P-wave oversensing in LBBAP. Fortunately, in our patient this only caused dizzy episodes, but in pacing-dependent patients syncope or even fatal asystole can occur. Far-field P-wave oversensing should be recognized during implantation and be considered in the case of reappearance of symptoms after LBBAP.

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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.2023.10.027.

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