

Late perforation of a left bundle branch area pacing lead causing ventricular fibrillation: A case report



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Introduction

Conduction system pacing (CSP) is becoming increasingly popular as an alternative to right ventricular (RV) pacing to avoid pacing-induced cardiomyopathy and as an alternative to biventricular pacing to optimize electrical resynchronization of the ventricles. However, large clinical studies are still needed to firmly establish the indications for CSP and to highlight possible downsides. CSP can be achieved either by pacing the His bundle or the left bundle branch area (LBBa). Data suggest that LBBa pacing has several advantages over His bundle pacing, like higher acute success rate, lower thresholds, and more stable pacing values over time. The most frequent complication with LBBa pacing is acute perforation of the septum¹ that, if recognized and the lead is repositioned, has no clinical consequence. Several studies and case reports have reported rare cases of late perforations days to weeks after implantation, with an incidence rate of <1%.²⁻⁵ No serious consequences of these late perforations have been reported other than the need for reintervention.

We present a case of late perforation of an LBBa lead that could have had fatal consequences, if the patient were not equipped with an implantable cardioverter-defibrillator (ICD).

Case report

A 55-year-old male patient (height 187 cm, weight 115 kg) was referred for cardiac resynchronization therapy (CRT) because of dilated cardiomyopathy with left ventricular ejection fraction 35%, NYHA class II despite optimal medical therapy, and an electrocardiogram with sinus rhythm and a typical left bundle branch block with a QRS duration of 180 ms.

The patient had undergone a successful ablation for atrial fibrillation in October 2021 and had type 2 diabetes and sleep apnea.

KEYWORDS Conduction system pacing; LBBa pacing; Complication; Late perforation; Ventricular fibrillation
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KEY TEACHING POINTS

- Late perforation of left bundle branch area (LBBa) leads is a rare but potentially harmful complication.
- Late perforation of LBBa leads can induce ventricular fibrillation in susceptible patients.
- Limited information on lead performance is available via remote monitoring when placing the lead in the left ventricular port of cardiac resynchronization therapy devices.
- During implantation of LBBa leads, attention should be paid to drops in current-of-injury and impedance, as these may indicate imminent perforation risk.

The patient agreed to enter a clinical study on CSP versus biventricular pacing and was randomized to the CSP arm. At implant, a single-coil ICD lead (Durata; Abbott, Chicago, IL) was first implanted to the distal septum of the right ventricle with good electrical values: sense 11.4 mV, threshold 0.5 V/0.4 ms, impedance 460 ohms.

It was then attempted to place a SelectSecure 3830 lead (Medtronic, Minneapolis, MN) in the His bundle position. Capture of the His bundle was obtained but there was only capture of the left bundle branch (LBB) at very high output (~10 V). Then the lead was redirected further into the right ventricle and several attempts were made to penetrate deep into the septum to achieve LBB capture, but penetration was found difficult owing to build-up of torque with rotation but without advancement. It was decided to change the lead to a stylet-driven lead to apply greater force to the tip of the lead and facilitate penetration into the septum. Then, a Solia S60 lead (Biotronik, Berlin, Germany) was successfully implanted with presumed capture of the posterior fascicle and a left ventricular activation time of 70 ms. There was no description of current-of-injury (COI) and no description of transition between selective/nonselective/septal capture. The unipolar threshold in the final position was 0.8 V

at 1.0 ms and the unipolar impedance was 360 ohms. In the device, the pacing vector was programmed tip-RV coil.

Lastly, an atrial lead (OptiSense; Abbott, Chicago, IL) was placed in the right atrial appendage with good electrical values (sense 3.1 mV, threshold 0.5 V/0.5 ms, impedance 410 ohms). The leads were connected to a CRT-defibrillator device from Abbott (Unify Assura). The LBB lead was connected to the left ventricle (LV) port and the V-V timing was set at LV first 80 ms.

Later the same day, the device was interrogated and all leads were reported to work properly, and a chest radiograph confirmed that the leads were in place. The threshold for the LBB lead was reported to be 0.5 V at 1.0 ms, impedance 300 ohms. The AV interval was adjusted to allow fusion between LBB pacing and conduction through the right bundle branch (Figure 1). The patient was feeling very well and was discharged the same evening with a telemonitoring box (Merlin, Abbott, Chicago, IL).

Twelve days postoperatively, a message from the telemonitoring system showed that the patient had received 2 appropriate shocks on VF: the first on Friday evening and the second on Sunday. Both incidents were quite similar, with multiple ventricular premature beats and VF induction by 1 or 2 premature beats with approximate coupling intervals of 290–260 ms and 260–230 ms (Figure 2).

The patient had no symptoms both before and after the shocks. He reported that he was feeling dizzy suddenly and then he observed a thump in the chest. Apart from that he did not think much of it. He was far from the hospital and was very reluctant to come for a check-up even with the risk of further shocks. However, he agreed to come the next day.

The only lead measurement received with the telemonitored transmission was the LBB impedance that was virtually unchanged at 360 ohms during the past 8 days. The device did not report sensing values, since the lead was placed in the LV channel and auto-capture was switched off.

The next day it turned out that the patient had received a third appropriate shock the night before. At interrogation the LBB lead was without capture at maximum output. The measurements for the other leads were unchanged (atrial lead: sense 4.8 mV, threshold 0.625 V/0.5 ms, impedance 400 ohms; RV lead: sense 11.4 mV, threshold 0.625 V/0.5 ms, impedance 400 ohms, shock impedance 69 ohms). A radiograph cineloop showed that the lead had penetrated into the LV cavity (Figure 3, and Supplementary Videos 1 and 2). Echocardiography confirmed that the lead tip was in the LV cavity. It was without evidence of thrombus formation (the apical 4-chamber view is available as Supplemental Video 3). The lead was removed without any complications. Considering the difficulties with the LBB implant, it was chosen to implant an LV lead instead, which was done without any complication.

The patient was discharged the next day without further complications. At 6 months follow-up there had been no more tachyarrhythmias, and the patient was feeling well and had improved clinically. Echocardiography showed improvement in left ventricular ejection fraction to 45% and 6-minute hall-walk distance was improved by 50 m.

Discussion

To our knowledge, a potentially fatal complication to LBBa pacing as the one described in the present case has never

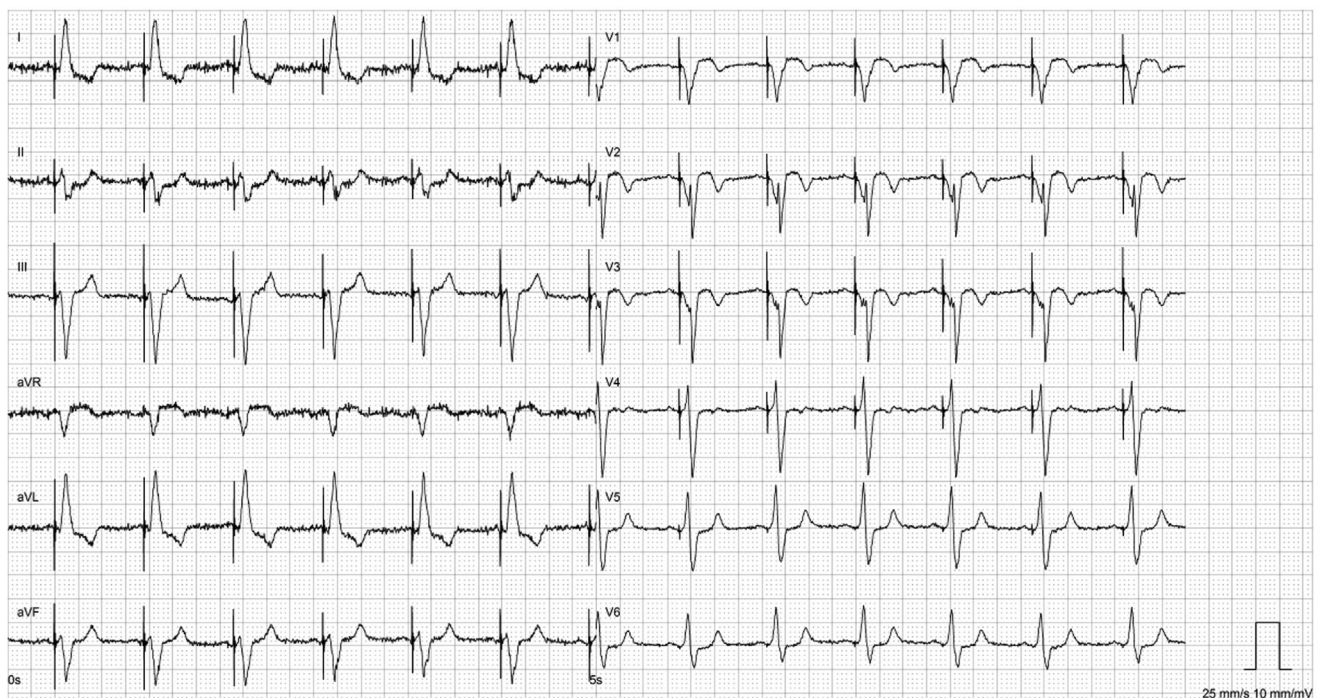


Figure 1 Electrocardiogram at discharge after left bundle branch (LBB) implantation showing fusion between LBB pacing and conduction through the right bundle branch. Left ventricular activation time was measured at 70 ms.

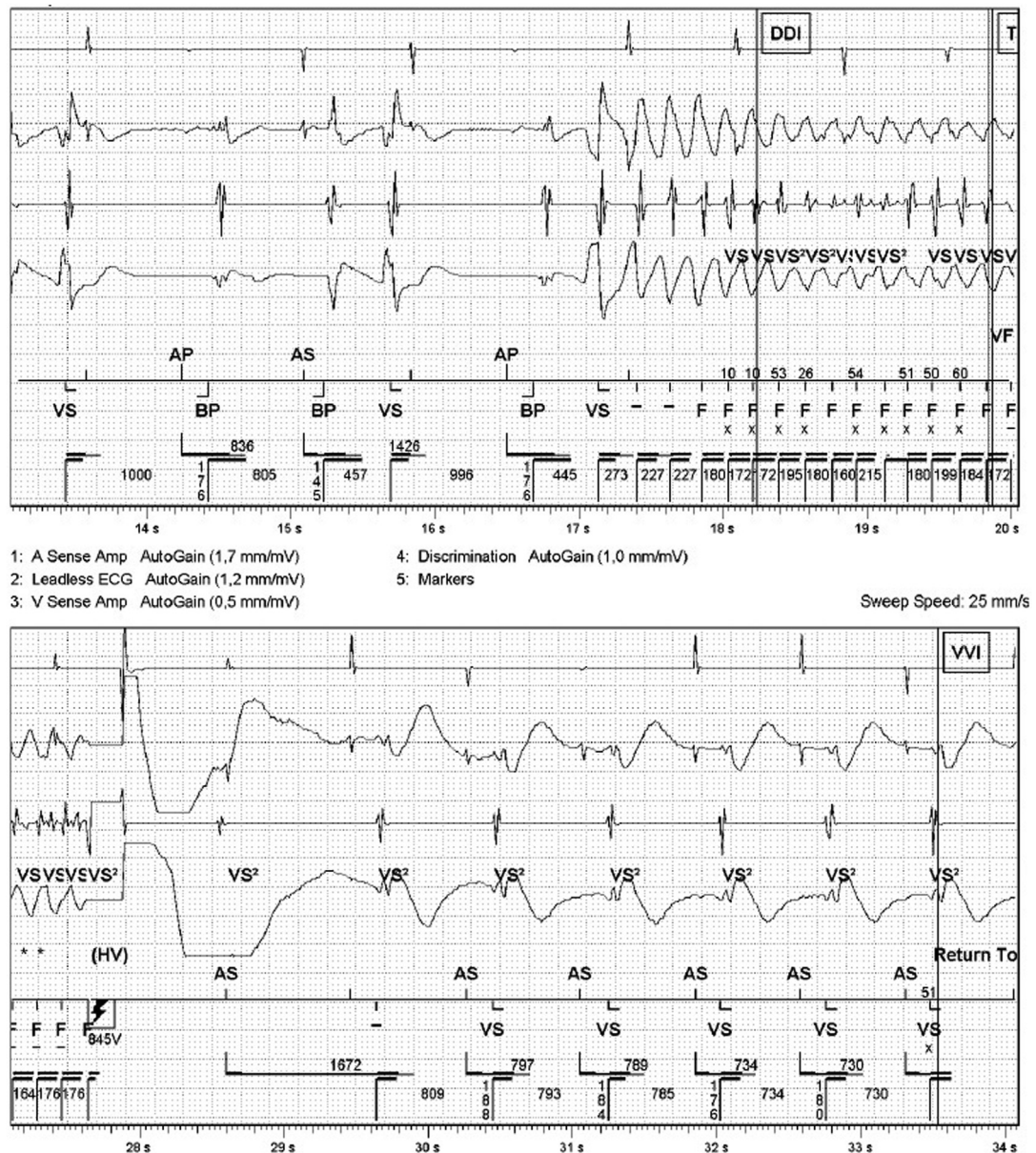


Figure 2 Tracings from the telemonitoring system showing the induction of ventricular fibrillation (VF) by several ventricular premature beats and the shock conversion to sinus rhythm. Not shown is the middle 7 seconds with VF.

been described before. Although the cause-and-effect relationship between lead perforation and VF is not 100% proven in this case, we think that coincidence in time of repeated VF occurrence and lead perforation and the fact that the patient was without malignant arrhythmias both before device implantation and after LBB lead removal provides overwhelming circumstantial evidence. From the MELOS registry,³ late perforation into the LV cavity was described in 2 out of more than 2000 patients without further sequela than the need for repositioning. Chen and colleagues² described 2 cases with late perforations out of a case series of 612 patients. One was on day 2 after implantation and the other was after 1 month. In both cases, the leads could be removed without any complications and new leads implanted. Su and colleagues⁴ reported 1 case of late perforation out of a case series

of 632 patients. Again, the lead could be removed and a new lead placed without complications. De Pooter and colleagues⁵ reported 1 case of late perforation out of 353 patients, resulting in loss of capture. This lead was repositioned without complications. Another case report⁶ described a late LBB lead perforation in a 71-year-old pacemaker patient who had received a pacemaker revision with implantation of an LBB lead for third-degree AV block. Two weeks later he presented with dizziness because of a rise in threshold above the programmed output. Revision was not scheduled until 4 weeks later when the threshold had risen further, and an echocardiogram showed evidence of lead perforation. The lead could be removed without complications.

In all previously reported cases, the focus has been on the risk of thromboembolic complications from having a foreign

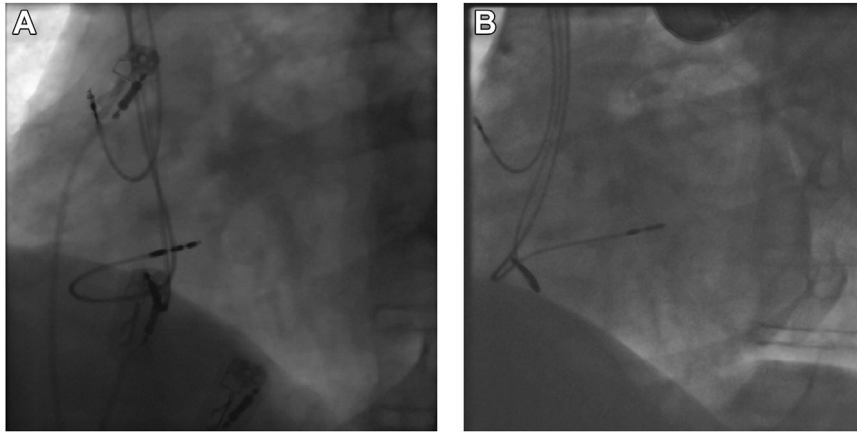


Figure 3 A: Position in left anterior oblique (LAO) 45 of the leads at implant. The left bundle branch area (LBBa) lead is deeply imbedded in the septum. B: Lead positions in LAO 46 at the reintervention procedure. The LBBa lead is advanced several centimeters into the left ventricle cavity.

object in the LV. Such complications had previously been described with LV leads placed using an atrial transseptal approach but never with LBB leads.⁷

The present case indicates that induction of potentially fatal tachyarrhythmias is another possible complication to perforation of the interventricular septum with a pacing lead. Most patients in previous studies have been patients without heart failure with an indication of pacing. Such patients have a lower risk of life-threatening arrhythmias than heart failure patients and this may be the reason it has never been reported or seen before. The induction of ventricular tachyarrhythmia in the present case was done by badly timed premature beats. This rather easy induction of tachyarrhythmia is rarely seen in electrophysiology studies in patients without heart failure.

We cannot neglect the other possibility that fatal complications could be previously under-reported. If the patient in the present case had received a pacemaker and not an ICD, he would likely have died. Only if an autopsy had been performed would the correct diagnosis of a perforated lead have been made. However, there has been no signal in previous studies suggesting a higher mortality among patients receiving CSP compared to patients receiving RV pacing or biventricular pacing; rather to the contrary.⁸ Again, we will know more when the results of the large ongoing randomized trials are available.

The question remains whether the late perforation in the presented patient could have been avoided or foreseen. The only sign of imminent perforation during the case was that the unipolar impedance in the final position was rather low, at 360 ohms. This is in the lower end of acceptable impedances, but impedances had been rather low in all positions and no sudden drop was seen. In addition, the other electrical values were excellent, with a low pacing threshold both at implant and before discharge. At the time, it was not yet standard practice to pay attention to how the COI behaved during implant of the lead. This has since become a primary focus area during implant when determining the proximity to the LV endocardium and trying to avoid

perforating into the LV cavity. In theory, the focus on COI could potentially prevent both acute and late perforations in the future.

The patient's CRT-defibrillator was monitored via telemonitoring, which was an advantage in the present case but is not ideal. In our clinic we have personnel examining the transmissions on weekdays during daytime and in this case, this delayed the detection of arrhythmias, since the patient had not taken any action on his own. In addition, with the LBB lead connected to the LV port in this specific device, there was no automatic detection of sensing values that possibly could have sooner given a clue that something was wrong. The automatic threshold testing was programmed off, which is the traditional way of programming CSP leads, but in this particular case it was maybe not necessary since there was only 1 threshold for LBBa capture. A potential rise in threshold could also have been an early warning sign.

We can hope that devices better designed for His and LBB leads will be available in the near future.

Conclusion

We present the first known case of late perforation of an LBB pacing lead leading to mechanically induced ventricular premature beats that initiated potentially fatal ventricular tachyarrhythmias. Fortunately, this happened in a patient with an ICD who survived without further complications. Although they are rare, the case illustrates that late perforations of these leads can be fatal.

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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.2024.05.002>.

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