Intermittent failure of pacing output caused by algorithm to prevent T-wave oversensing

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Introduction

Oversensing of extracardiac myopotentials can lead to pacemaker inhibition and loss of cardiac output. We describe 2 similar cases in which oversensing of myopotentials and loss of pacemaker output in pacemaker-dependent patients was caused by a proprietary algorithm designed to prevent T-wave sensing. Switching off the algorithm resulted in return of normal pacemaker function. Knowledge of this algorithm is essential for patient safety and avoidance of a potentially unnecessary invasive procedure to reposition a lead.

Case report

In case 1, an 81-year-old man with ischemic cardiomyopathy and a dual-chamber pacemaker implanted for complete atrioventricular block was upgraded to a cardiac resynchronization therapy and defibrillator device. The right ventricular (RV) pacing lead was extracted and replaced with an implantable cardiac defibrillator (ICD) lead (Durata; St Jude Medical) positioned at the RV apex and a left ventricular lead (Quartet; St Jude Medical) in an optimal suitable coronary venous tributary. The pulse generator (Quadra Assura; St Jude Medical) was programmed to DDDR mode (60–130 bpm). The following day the patient was observed to have ventricular pauses lasting over 5 seconds (Figure 1A).

In case 2, a 69-year-old man with ischemic cardiomyopathy and dual-chamber pacemaker (Identity ADx XL DR; St Jude Medical) implanted for complete atrioventricular block underwent an upgrade to a cardiac resynchronization therapy and defibrillator device. An ICD lead (Optisure; St Jude Medical) was placed in the RV apex and a quadripolar left ventricular lead (Quartet; St Jude Medical, St Paul, MN) in the optimal suitable coronary venous branch. The RV pacing lead was capped and abandoned. The pre-existing bipolar right atrial lead (5076; Medtronic) was connected to the new pulse generator (Quadra Assura MP; St Jude Medical) and the system programmed to VVI 60 bpm because the patient remained in atrial fibrillation. The following day, the patient was noted to have ventricular pauses up to 3.8 seconds (Figure 1B).

In both cases, clinical and imaging assessment showed no evidence of lead perforation or dislodgement (Figure 1C). Device interrogation at rest, with pocket manipulation, and with isometric exercise revealed stable capture thresholds, sensing, and impedances (Figures 2A and 3A). However, deep inspiration and coughing reproduced inhibition of pacing output, with high-frequency low-amplitude noise recorded on the near-field channel (Figures 2B and 3B). This effect was independent of pacing polarity, pacing output, and pacing mode. Decreasing the RV channel sensitivity from 0.5 mV (nominal) to 1.0 mV abolished oversensing and pacing inhibition with respiratory maneuvers (Figures 2C and 3C). There was concern that the proprietary low-frequency attenuation (LFA) filter might be responsible for loss of pacing output. Inactivation of this feature without reducing RV channel sensitivity prevented further oversensing at rest and with respiratory maneuvers (Figures 2D and 3D).

Discussion

In both cases, the LFA filter, a proprietary signal-processing algorithm designed to prevent T-wave oversensing (TWOS), resulted in failure of pacing output in pacemaker-dependent patients. The LFA filter was introduced in the St Jude Medical Unify and Fortify devices and is a combination of a high-pass filter and Brady filter.[1] The LFA filter and the Brady filters share a similar center frequency. The LFA filter has a faster roll-off slope below 20 Hz, allowing for additional attenuation of low-frequency content. Compared with the existing Tachy filter, the LFA filter is able to increase R to T wave amplitude ratio by a factor of

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KEY TEACHING POINTS

- When interrogating an apparently malfunctioning pacing system it is important to perform respiratory and physical maneuvers together with imaging assessment.
- In pacemaker-dependent patients myopotential oversensing is an uncommon idiosyncratic problem that can be exacerbated by a proprietary St Jude Medical low-frequency attenuation (LFA) filter, with subsequent inhibition and failure of pacing output.
- Deactivation of the LFA filter can eliminate myopotential oversensing without the need for invasive interventions or a reduction in lead sensitivity.

approximately 2–3. The amplification of high-frequency signals enhances sensing performance but in this case predisposes to diaphragmatic myopotential oversensing. The LFA filter is nominally "ON" in the new generation of St Jude Medical high-voltage devices (including the Fortify/Unify/Quadra Assura and Ellipse models). The nominal sensitivity when the LFA filter is ON is 0.5 mV, whereas when the LFA filter is OFF it is 0.3 mV. In both clinical scenarios, deactivation of the filter resolved the issue without the need for invasive interventions or making the RV channel less sensitive (Figures 2D and 3D).

Our cases illustrate the importance of performing respiratory and physical maneuvers when interrogating an apparently malfunctioning pacing system. An important differential diagnosis is lead perforation, including microperforation by the lead tip screw mechanism. In the context of a recent extraction procedure and/or new RV lead implantation, perforation and lead migration with diaphragmatic myopotential oversensing must be considered. However, high-output pacing through the RV lead did not capture and stimulate the diaphragm, making this scenario unlikely. Careful clinical and imaging assessment is necessary to exclude this serious complication.

Intracardiac or extracardiac oversensing in the ICD population has an estimated prevalence of 7.9%.[2] Pacemaker-dependent patients are particularly vulnerable to far-field myopotential oversensing, with serious consequences such as syncope owing to inhibition of ventricular pacing or inappropriate shocks.[3] The prevalence of diaphragmatic myopotential oversensing in pacemaker-dependent patients has been estimated at 8.6%–18.9% in the cardiac resynchronization therapy population.[3,4] Well-described associations include unipolar or integrated bipolar sensing configurations, inadvertent DF-1 connector inversion at implantation, apical lead placement, and dynamic sensitivity adjustment in the presence of low-amplitude R waves.[4,5] In patients with integrated bipolar sensing, inadvertent inversion of the DF-1 connector creates a larger sensing field, which incorporates upper limb and pectoral myopotential sensing. In that case, myopotential "noise" is noted with upper limb maneuvers, and surgical correction is mandatory. Oversensing of diaphragm myopotentials may be corrected by reducing ventricular sensitivity if ventricular fibrillation (VF) sensing is reliable. Figures 2C and 3C show the persistence of high-frequency low-amplitude noise, with deep inspiration, on the near-field channel. This noise is not sensed when setting the channel at a lower sensitivity of 1.0 mV. However, "blanket" programming the RV channel to

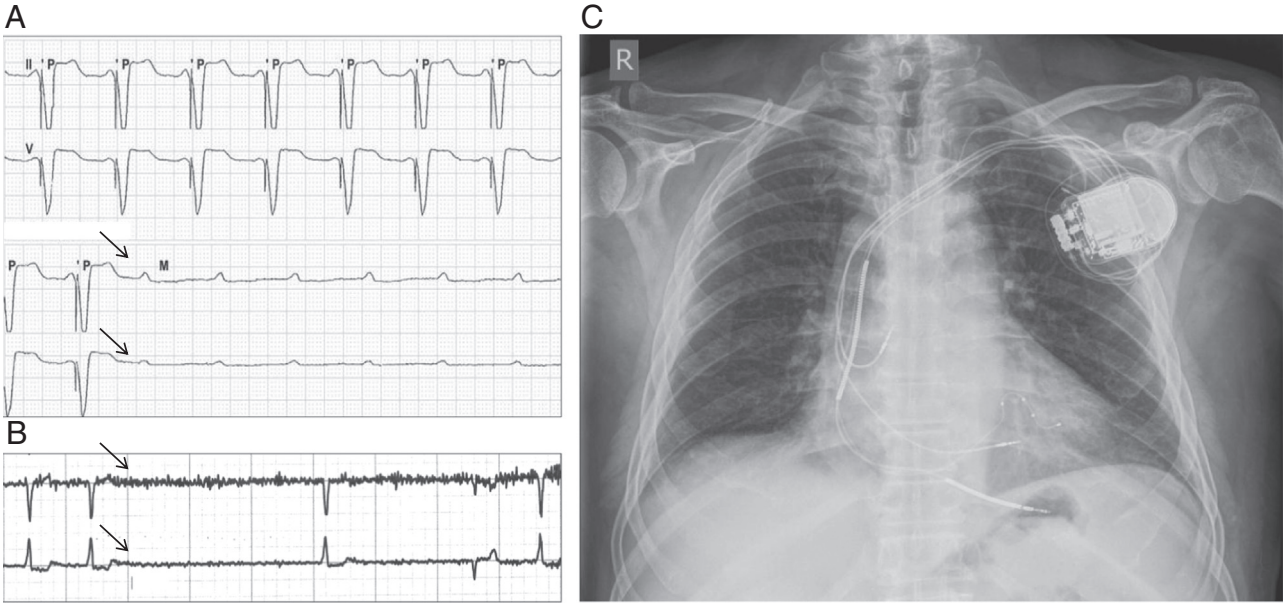

Figure 1 Representative cardiac telemetry recordings show failure of pacing (*arrows*) the day after device implantation in **A:** case 1 and **B:** case 2. **C:** A posteroanterior chest x-ray showing the 3 leads in situ at the locations documented at implant for case 2.

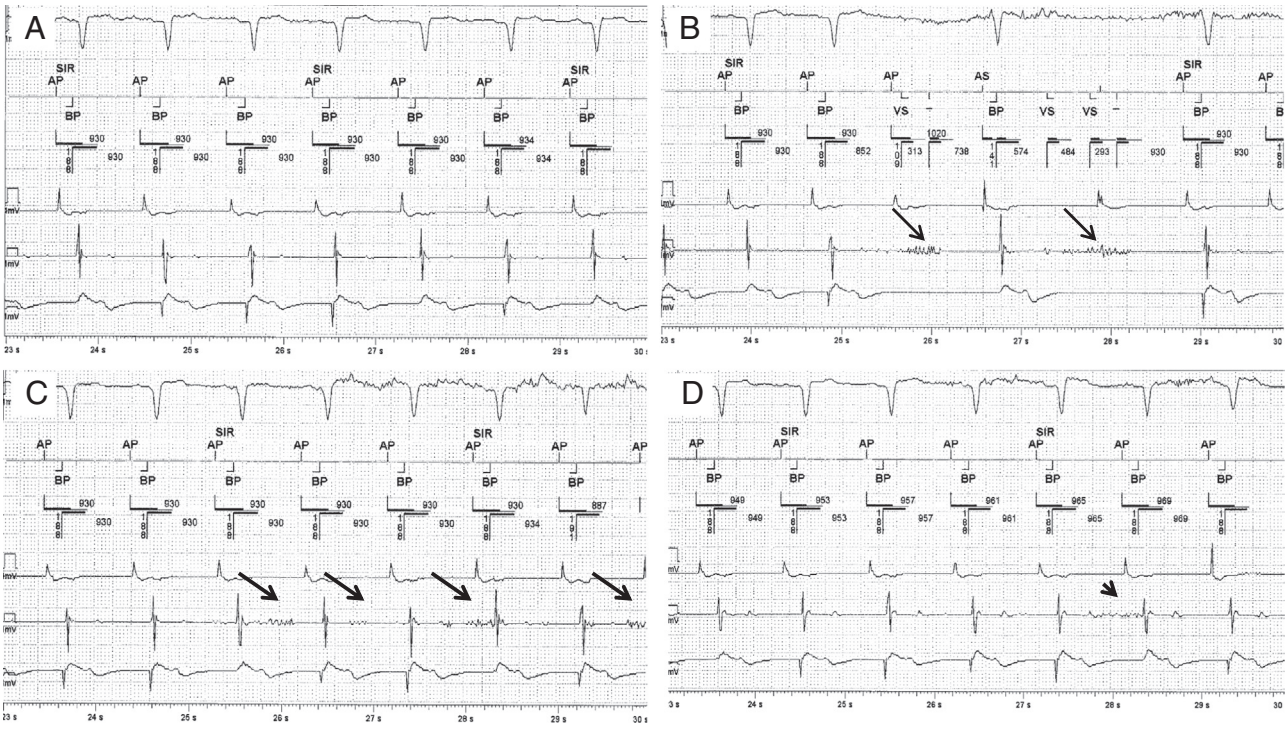

Figure 2 Intracardiac electrograms from patient in case 1. Shown from top to bottom: lead II AutoGain; marker channel; atrial bipolar electrogram; right ventricular near field; and left ventricular distal tip 1 to mid-pole 2. Sweep speed 25 mm/s. **A:** Low-frequency attenuation (LFA) filter ON at nominal sensitivity 0.5 mV with normal quiet breathing. **B:** LFA ON at nominal sensitivity 0.5 mV with deep inspiration maneuvers. High-frequency, low-amplitude "noise" seen on near-field electrogram channel with associated inhibition of pacing (*arrow*). **C:** LFA filter ON at a sensitivity of 1.0 mV (least sensitive) with deep breathing. Noise is seen again on the near field (*arrow*); however, owing to the low sensitivity setting on the right ventricular near field channel (1.0 mV), no oversensing or pacing inhibition is observed. **D:** LFA filter OFF at nominal sensitivity 0.3 mV and deep inspiration. Diaphragmatic myopotential noise (*arrowhead*) is not amplified, owing to absence of LFA filter algorithm operation; therefore, no oversensing is observed.

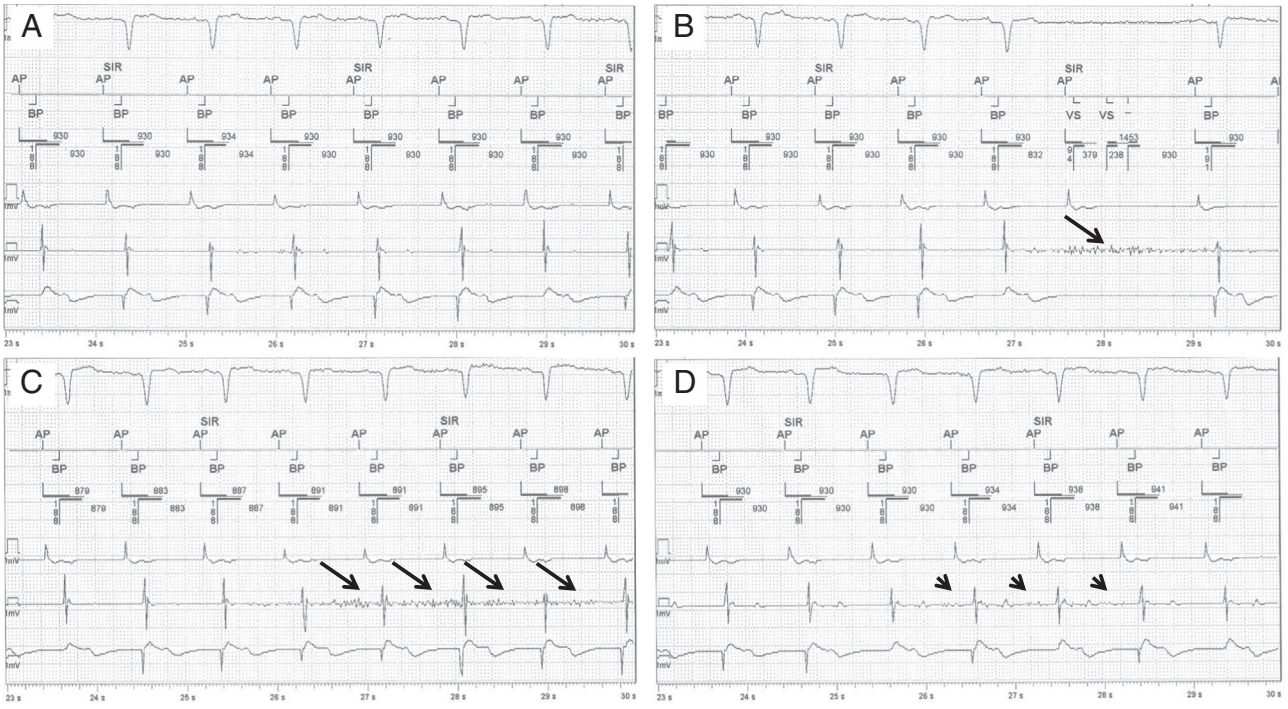

Figure 3 Intracardiac electrograms from patient in case 2. Electrogram set-up and sweep speed is as described in Figure 2. **A:** Low-frequency attenuation (LFA) filter ON at nominal sensitivity 0.5 mV with normal quiet breathing. **B:** LFA filter ON at nominal sensitivity 0.5 mV with deep inspiration maneuvers. High-frequency, low-amplitude "noise" is seen on the right ventricular near-field electrogram channel with associated inhibition of pacing (*arrow*). **C:** LFA filter ON at a sensitivity 1.0 mV (least sensitive) with deep breathing. Noise is seen on the near field (*arrow*); however, owing to the low sensitivity setting on the right ventricular near-field channel (1.0 mV), no oversensing or pacing inhibition is observed. **D:** LFA filter OFF at nominal sensitivity 0.3 mV and deep inspiration. Diaphragmatic myopotential noise (*arrowhead*) is not amplified, owing to absence of LFA filter algorithm operation; therefore, no oversensing is observed.

less sensitive carries a risk of VF undersensing. Defibrillation testing, with its inherent risks, may therefore be prudent. Finally, when the LFA filter is inactivated, the RV near-field sensitivity reverts to its nominal value of 0.3 mV, increasing the risk of TWOS. Very low amplitude noise (diaphragm myopotentials) is seen with deep inspiration; however, this is not sensed, as it is not amplified.

In these cases, we postulate that in the presence of an operative LFA filter, amplification of diaphragmatic myopotentials may result in intermittent oversensing and pacemaker output inhibition. To our knowledge, this has not been described and likely represents an uncommon reaction to this algorithm. The apparent infrequency of this observation is probably a function of anatomic variation and lead position in relation to the diaphragm. It is also likely that this phenomenon is often not recognized, particularly in patients who are not pacemaker dependent. Knowledge of this algorithm is essential for patient safety and avoidance of a likely unnecessary invasive procedure to reposition the lead. In the appropriate clinical context, recognizing the potential role of the LFA filter in apparent failure of pacemaker output is important. Inactivation of the LFA filter should particularly be considered in pacemaker-dependent patients. Device interrogation with respiratory maneuvers should be repeated after its inactivation, prior to making the ventricular channel *less* sensitive or performing an invasive procedure for correction. Conversely, inactivation of the LFA filter algorithm should prompt greater surveillance for TWOS.

In both cases, changing the sensitivity from 0.5 mV to 1.0 mV abolished diaphragmatic myopotential oversensing, but concerns regarding VF undersensing led to the decision to inactivate the LFA filter with reversion to nominal sensitivity (0.3 mV). This prevented further significant oversensing and therefore pacemaker output inhibition, at rest and on deep inspiration. At this new nominal sensitivity, with the LFA filter "OFF," defibrillation testing was performed in case 2 and was successful at 25 J. No TWOS was observed in either case after the programming change.

Conclusion

In clinical practice, the LFA filter appears effective in minimizing TWOS. The observed myopotential oversensing appears to be an uncommon complication of the algorithm, in pacemaker-dependent patients.

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