T-wave oversensing due to left ventricle–only pacing in cardiac resynchronization therapy optimization algorithm

Philippe André, MD, MSc, Benoit Plourde, MD, Franck Molin, MD,
Jean-Francois Sarrazin, MD, FHRS, Jean Champagne, MD, François Philippon, MD, FHRS

From the Division of Electrophysiology, Quebec Heart and Lung Institute, Quebec City, Canada.

Introduction

Despite improved patient selection criteria, the rate of non-responders with cardiac resynchronization therapy (CRT) devices remains significant. After implant, tailored programming could improve resynchronization and achieve a greater responder rate.

With the hypothesis that both left ventricular (LV) and right ventricular (RV) as well as atrioventricular (AV) delays have an impact on LV output and remodeling, device companies have developed several algorithms to enhance and optimize CRT timing. Previous studies have published variable results and benefits from these algorithms.[1,2] Globally, the impact on LV remodeling appears beneficial. LV-only pacing has been found non-inferior to biventricular (BiV) pacing for clinical and echographic outcomes. It might be interesting in CRT devices to increase battery longevity compared to BiV stimulation.[3] Medtronic's AdaptivCRT (Minneapolis, MN) optimizes both AV and interventricular delays to increase LV output. The algorithm incorporates a feature that allows for LV-only pacing[4,5] fusion with the intrinsic conduction and usually produces a narrower QRS than BiV or LV-only pacing. The Adapt Response trial will answer if this algorithm has a favorable impact on clinical outcomes.

We report a case where programming the AdaptivCRT caused T-wave oversensing resulting in decreased LV pacing. Since near 100% resynchronization is an objective, this finding could result in reduced benefit of CRT in this patient.[6,7]

Case report

A 66-year-old man with long-standing ischemic cardiomyopathy, LV ejection fraction at 25%, complete left bundle branch block (QRS width 160 ms), and CRT device was admitted for elective implantable cardioverter-defibrillator and lead extraction procedure because of dysfunctional leads and implant of a new CRT defibrillator. He also had history of high blood pressure, diabetes mellitus, peripheral vascular disease, and paroxysmal atrial fibrillation.

The extraction procedure was successful and uneventful and allowed for a new CRT defibrillator implant. The final system included a Sprint Quattro MRI 6935M DF4 defibrillator lead (Medtronic), a CapSureFix 4076 right atrium lead (Medtronic), and an Attain Performa 4298 LV lead (Medtronic) connected to a Medtronic VIVA QUAD XT CRT-D. The LV lead was positioned in a mid to basal posterolateral vein.

Device settings were DDDR 60–140 with AdaptivCRT ON (adaptive BiV and LV). Postventricular atrial refractory period (PVARP) was set on Partial + (minimal PVARP 250 ms), V blanking post ventricle sense (VS) was 120 ms, and V blanking post ventricle pace (VP) was 200 ms. There was an excellent R-wave detection at 18.8 mV. Premature ventricle capture (PVC) response was programmed ON, meaning that after an intrinsic beat considered by the device as a PVC, the PVARP is extended up to 400 ms to avoid pacemaker-mediated tachycardia.

Immediate postoperative telemetry showed BiV pacing alternating systematically with an intrinsic left bundle branch block QRS complex. Device analysis showed a VS marker following a VP and occurring concomitantly to the T wave (Figure 1).

Postoperative intracardiac electrogram with Adaptiv BiV ON showed AS marker (sinus P wave) followed by VP marker (LV lead pacing), VS (T-wave oversensing with the RV lead), and atrial refractory (sinus P wave post VS, in PVARP because of the extension of the PVARP to 400 ms due to the PVC response algorithm). Since atrial refractory does not trigger BiV or VP, this explains the VS resulting in spontaneous conduction.

T-wave oversensing is manifested on these tracings but requires further analysis. In fact, despite that the T-wave detection algorithm was programmed ON and the R-wave sensitivity was decreased to 1 mV, the phenomenon persisted. We also chose not to increase ventricular blanking

KEY TEACHING POINTS

- In cardiac resynchronization therapy (CRT), loss of biventricular pacing and a 1-on-2 biventricular pacing pattern can be caused by T-wave oversensing.

- T-wave oversensing can be induced by the CRT optimization algorithm AdaptivCRT.

- If usual T-wave oversensing troubleshooting is inefficient, turning this algorithm off could restore resynchronization under certain conditions.

post VS to avoid undersensing of ventricular arrhythmia, since VS events occurred 400 ms after VP. Of note, Figure 1 shows that this finding coincided with VPs that actually were LV-only pacing events. Thus, with the AdaptivCRT algorithm activated allowing LV-only pacing, suboptimal resynchronization occurred.

After the device was set in nonadaptive CRT mode (with nominal R wave at 0.3 mV and same V blanking period),

allowing constant BiV pacing, the problem was solved and the patient was continuously paced (Figure 2).

Discussion

Usual management of T-wave oversensing post VP includes the following: (1) extension of post-VP blanking (may have to decrease the upper tracking, ventricular sense response, and sensor rates to allow for post-VP blanking extension), (2) programming an auto-adjusting sensitivity (begins after the post-VP blanking interval, so by extending blanking, the T wave often will not be sensed), (3) reducing the ventricular sensitivity while analyzing the real-time electrogram to see when T-wave oversensing disappears, and (4) trying a different V sensing vector (RV tip to RV coil). If sensing vector is changed, or V sensitivity > 0.6 mV is required, it may be appropriate to perform a defibrillation safety margin test.

All those usual troubleshooting strategies were inefficient because of the peculiar origin of the T-wave oversensing.

Medtronic AdaptivCRT requires a normal AV conduction (up to 250 ms in paced events) and a heart rate less than 100 beats/min for LV-only pacing. It paces the LV after 70% of the intrinsic AV delay.[8]
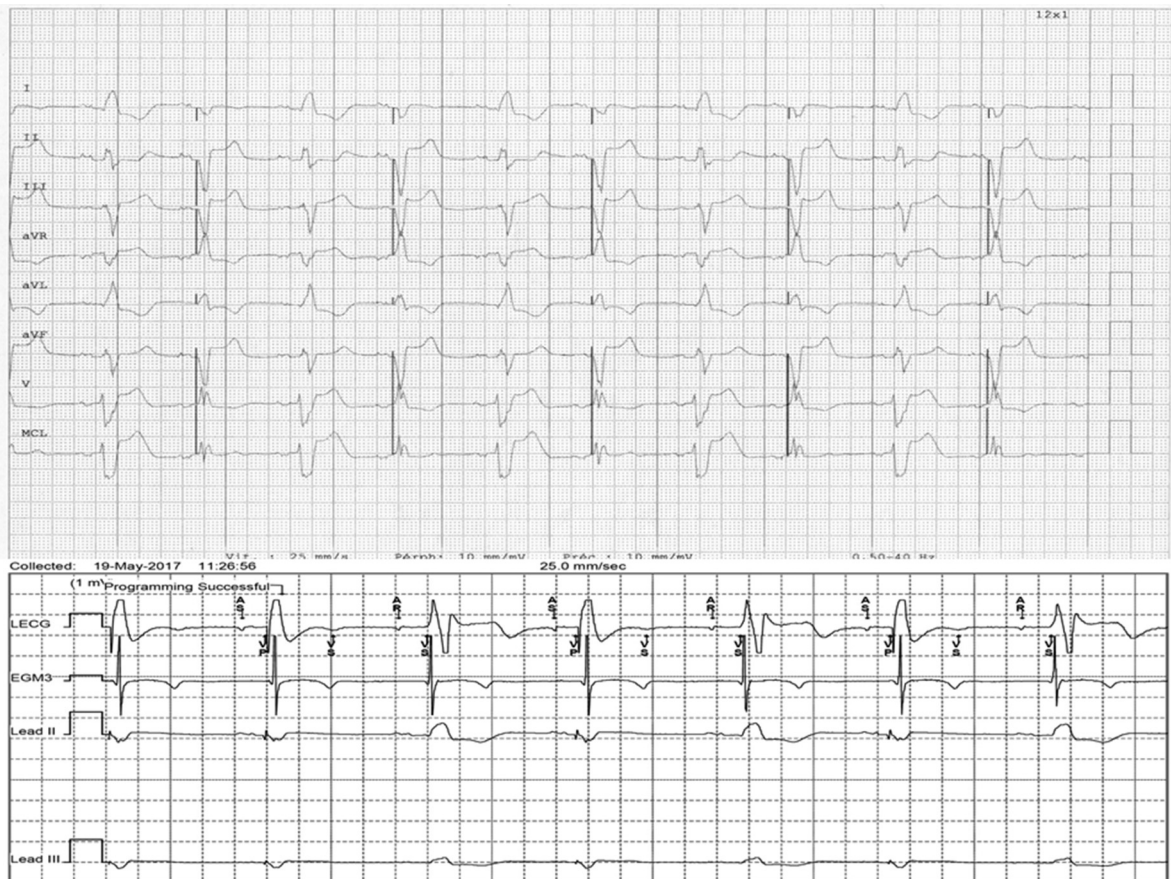
Figure 1 Occurrence of an episode of T-wave oversensing after a left ventricular (LV)-only pacing (AdaptivCRT ON). Top panel shows a telemetry strip with alternating biventricular (BiV) pacing and intrinsic QRS. Bottom panel shows an intracardiac electrogram (EGM) and markers. AR = sinus P wave in postventricular atrial refractory period; AS = sinus P wave with ventricular pacing (VP) (LV only); VS = oversensed T wave approximately 400 ms after VP; VS following AR = native conduction with left bundle branch block through the atrioventricular node, since no BiV/VP trigger by the AR.
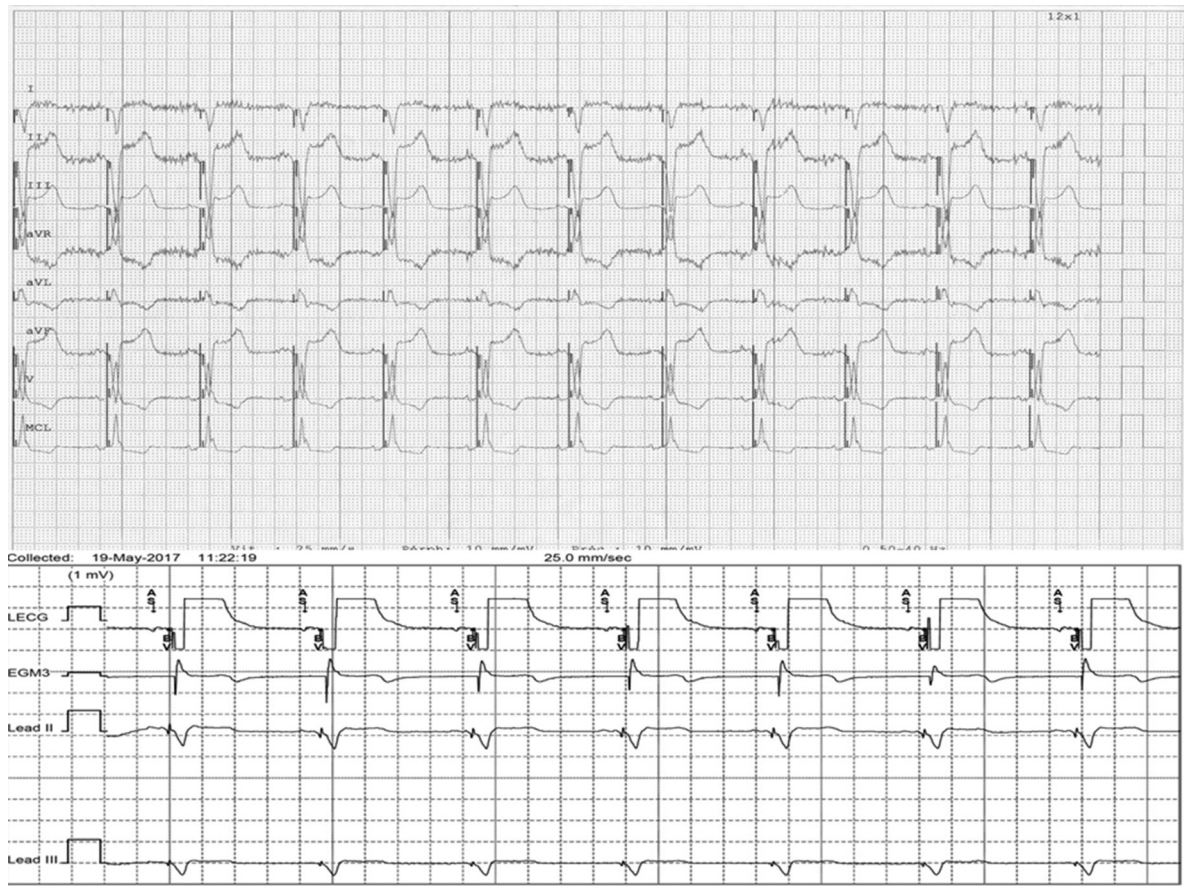
Figure 2   Adequate biventricular pacing following turning off AdaptivCRT with left ventricular (LV)-only pacing. T-wave oversensing disappeared compared to LV-only stimulation. Top panel shows a telemetry strip following device modification. Bottom panel shows intracardiac electrogram (EGM) after turning Adaptiv LV to OFF. AS = sinus P wave.

In this patient with normal AV conduction, the algorithm was initially set on Adaptiv LV and BiV but since it fulfilled all criteria, it operated in Adaptiv LV only.

The T-wave oversensing algorithm is the same for Adaptiv BiV or Adaptiv LV only. In the presence of a very dilated LV and compared to BiV pacing, it is possible that sensing from the right ventricle allowed the detection of a wavefront activation of a very late T wave that was misclassified as an R wave, resulting in T-wave oversensing not correctly identified by the algorithm.

Simultaneous RV and LV pacing makes the depolarization more synchronous in both ventricles. Therefore, repolarization (T wave) happens earlier compared to LV pacing only, allowing the T-wave oversensing algorithm to detect any possible T wave.

T-wave oversensing post pacing will not result in an inappropriate shock. The VT counters must be consecutive and with pacing occurring, the tachycardia count will not reach the detection criteria.

On the other hand, postpacing T-wave oversensing will result in the heart rate falling below the programmed limit and, similar to our case,[9,10] the absence of benefits from CRT with loss of resynchronization 50% of the time.

The clue to this phenomenon is when resynchronization happens every other beat. Clinicians must be aware of this possibility and ways to reprogram the device to achieve adequate resynchronization.

Conclusion
In the case of a Medtronic device with AdaptivCRT with LV-only setting, exhibiting a BiV paced beat followed by an intrinsic beat, if all the usual troubleshooting for T-wave oversensing mentioned above failed, turning off this algorithm could restore permanent BiV pacing.

References
1. Abraham WT, Gras D, Yu CM, Guzzo L, Gupta MS, FREEDOM Steering Committee. Rationale and design of a randomized clinical trial to assess the safety and efficacy of frequent optimization of cardiac resynchronization therapy: the Frequent Optimization Study Using the QuickOpt Method (FREEDOM) trial. Am Heart J 2010;159:944–948.e1.
2. Ellenbogen KA, Gold MR, Meyer TE, et al. Primary results from the SmartDelay determined AV optimization: a comparison to other AV delay methods used in cardiac resynchronization therapy (SMART-AV) trial: a randomized trial comparing empirical, echocardiography-guided, and algorithmic atrioventricular delay programming in cardiac resynchronization therapy. Circulation 2010; 122:2660–2668.

3. Liang Y, Pan W, Su Y, Ge J. Meta-analysis of randomized controlled trials comparing isolated left ventricular and biventricular pacing in patients with chronic heart failure. Am J Cardiol 2011;108:1160–1165.

4. Starling RC, Krum H, Bril S, Tsintzos SI, Rogers T, Hudnall JH, Martin DO. Impact of a novel adaptive optimization algorithm on 30-day readmissions: evidence from the Adaptive CRT Trial. JACC Heart Fail 2015;3:565–572.

5. Martin DO, Lemke B, Birnie D, et al. Investigation of a novel algorithm for synchronized left-ventricular pacing and ambulatory optimization of cardiac resynchronization therapy: results of the adaptive CRT trial. Heart Rhythm 2012; 9:1807–1814.

6. Hayes DL, Boehmer JP, Day JD, Gilliam FR, Heidenreich PA, Seth M, Jones PW, Saxon LA. Cardiac resynchronization therapy and the relationship of percent biventricular pacing to symptoms and survival. Heart Rhythm 2011;8:1469–1475.

7. Koplan BA, Kaplan AJ, Weiner S, Jones PW, Seth M, Christman SA. Heart failure decompensation and all-cause mortality in relation to percent biventricular pacing in patients with heart failure: is a goal of 100% biventricular pacing necessary? J Am Coll Cardiol 2009;53:355–360.

8. Krum H, Lemke B, Birnie D, Lee KL-F, Aonuma K, Starling RC, Gasparini M, Gorcsan J, Rogers T, Sambelashvili A, Kalmes A, Martin D. A novel algorithm for individualized cardiac resynchronization therapy: rationale and design of the adaptive cardiac resynchronization therapy trial. Am Heart J 2012;163:747–752.e1.

9. Lezcano AOL, Aracama JMP, Ayestaran VU, Urra FG. T wave oversensing and low percentage of biventricular pacing in cardiac resynchronization therapy. Cardiol J 2009;16:580–581.

10. Kawata H, Noda T, Yamada Y, Okamura H, Nakajima H, Kobayashi J, Kamakura S. Abrupt heart rate fallings in a patient with biventricular pacing: latent risk for exacerbation of heart failure. Pacing Clin Electrophysiol 2012;35:e55–58.