Published in final edited form as:

Pacing Clin Electrophysiol. 2013 August ; 36(8): 1024–1026. doi:10.1111/pace.12194.

Electrophysiologic mechanism of deteriorating cardiac function in a patient with inappropriate CRT indication and frequent ventricular ectopy

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Keywords

Electrocardiographic Imaging, ECGI; Cardiac Resynchronization therapy; Premature ventricular contraction

An 84 y/o man had worsened symptoms (NYHA class III, LVEF 25%) following implant of a biventricular (BiV) cardioverter defibrillator (Biotronik, Lumax 540) at another institution. He also had frequent premature ventricular contractions (PVCs, 35%) that interfered with cardiac resynchronization therapy (CRT) and triggered BiV pacing. At electrophysiology (EP) study, the left ventricular (LV) lead was discovered to be within the anterior interventricular vein. His native QRS (113 ms) was much narrower than during CRT; thus, BiV pacing was disabled to allow for native conduction. The dominant clinical PVC was mapped and successfully ablated from the coronary cusps. His symptoms improved dramatically within days of the procedure and programming change. Prior to ablation, Electrocardiographic Imaging (ECGI)¹⁻² was applied to noninvasively map and study the mechanisms of pacing induced dyssynchrony (Figure 1) and ineffectiveness of PVC-triggered pacing (Figure 2).

This patient had a narrow QRS and consequently a predictable lack of benefit from CRT and inappropriate CRT indication. Moreover, his LV lead was anterior, which several studies have demonstrated not to be optimal for CRT. Inappropriate CRT in patients with synchronous LV activation could cause pacing induced dyssynchrony, leading to deteriorating LV function.^{1, 3} In the context of heart failure, ECGI has been used to map the EP substrate², to identify potential responders/nonresponders to CRT¹, and to guide electrode placement⁴. Unlike the global body-surface QRSd, the ECGI derived cardiac electrical dyssynchrony (ED) index, computed as the standard deviation of all activation times on the LV, quantifies the spatial dispersion of activation over the LV (ED>28 ms is a marker of electrical dyssynchrony¹).

As shown in the ECGI image of Figure 1, pacing from the early-activated LV anterior wall during BiV created a functional line-of-block. This forced the activation front to propagate

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Disclosures:

Dr. Rudy co-chairs the scientific advisory board of and holds equity in CardioInsight Technologies. CardioInsight Technologies does not support any research conducted by Dr. Rudy, including that presented here. Dr. Cooper has received consultant fees or speaker honoraria from Boston Scientific, St. Jude Medical, Medtronic, and Biotronik.

in a U-shaped pattern around the line-of-block, causing late dyssynchronous activation of the lateral LV base. The image in Figure 2 shows that during PVC-triggered pacing, the PVC site of origin was in close proximity to the anterior LV lead, but far from the RV sensing lead, which caused delayed sensing and pacing. Thus, BiV pacing triggered by PVC failed to capture the (already activated) ventricles and was ineffective. Because in many similar cases PVCs can impede the effective delivery of continuous CRT, PVC treatment (catheter ablation or antiarrhythmic drugs) to ensure high percentage of effective BiV pacing might be considered.

Acknowledgments

Funding Sources:

This study was supported by NIH-NHLBI grants R01-HL-033343-28 and R01-HL-049054-20 (to Yoram Rudy) and by the Washington University Institute of Clinical and Translational Sciences grant UL1 TR000448 from the National Center for Advancing Translational Sciences (NCATS) of the NIH.

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Figure 1.

QRS morphologies (12-lead ECG) and ECGI-reconstructed epicardial activation isochrones under native sinus rhythm (Panel A, left, and Panel B) and BiV Pacing (Panel A, right, and Panel C). Note that QRS in native sinus rhythm is much narrower than for BiV pacing. The activation maps are shown in right anterior oblique (top, RAO) and lateral (bottom, LAT) views. Pacing sites are indicated by plus signs(+). QRSd and ED index are computed for each rhythm. The white arrow highlights the U-shaped propagation pattern around a functional line of block (black arrows) during BiV Pacing, which induced dyssynchrony. Inset: Fluoroscopy images showing the pacing lead positions in RAO and left anterior oblique (LAO) views. RA= right atrium; LA= left atrium; RV= right ventricle; LV= left ventricle.

Figure 2.

QRS morphologies (12-lead ECG) and ECGI activation Isochrone maps during PVC-triggered BiV pacing (Panel A, left, and Panel B) and spontaneous PVC without pacing (Panel A, right, and Panel C). The activation maps are shown in posterior (top, POS) and left anterior oblique (bottom, LAO) views. Pacing sites are indicated by plus signs (+). The earliest epicardial activation is marked with an asterisk (*). RA= right atrium; LA= left atrium; MV= mitral valve. PVC-triggered BiV pacing did not change the QRS morphology of the PVC (pacing occurred during the PVC, too late to capture). There was no appreciable effect of pacing on the PVC (Panel B, compare to Panel C). Both rhythms had identical activation patterns and similar ED and QRSd values.