Optimization of cardiac resynchronisation therapy: LV Lead position, qLV, or paced effects?

Niraj Varma, Raja Ghanem, and Ping Jia
Cardiac Bioelectricity Research and Training Center, Case Western Reserve University and University Hospitals of Cleveland, Cleveland, OH, USA
* Corresponding author. Tel: +1 216 444 2142; fax: +1 216 445 6161. E-mail address: varman@ccf.org

A New York Heart Association (NYHA) Class III female patient with ischaemic cardiomyopathy (left ventricular ejection fraction 20%) and left bundle branch block with QRS duration 160 ms received cardiac resynchronisation therapy (CRT). The left ventricular (LV) lead was placed anteriorly, since this was the only available tributary. Biventricular pacing narrowed QRS duration to 120 ms, but without the emergence of an R wave in lead V1. Electrocardiographic imaging was undertaken 20 days later (Figure 1). She was a ‘responder’, regaining NYHA Class 1 function and sustaining echocardiographic reverse remodelling over the next 12 months.

Manoeuvres to improve CRT efficacy at implant are directed to LV lead deployment to a posterolateral region associated with a long activation time (‘qLV’). However, no chronic difference in outcomes were noted among patient groups with lateral, inferior, or anterior leads, and qLV > 95 ms is only modestly predictive of CRT efficacy (AUC 0.63). These results expose the limitations of current notions of ‘ideal’ LV lead positioning, which miss the assessment of LV paced effect, although this is the delivered treatment. Thus, haemodynamically optimal LV pacing sites were not predictable from anatomical positions, or qLV, and had to be individualized. In contrast, post-implant structural remodelling was associated with restoration of electrical resynchronization, independently of lead position or qLV.

The current case indicates that successful lead location should be adjudicated on results of individualized paced effects that restore electrical resynchronization, irrespective of anatomical position and/or qLV. In the future, electrocardiographic imaging may guide electrode deployment and timing of stimulation (LV/RV), especially to overcome nonconventional lead locations and functional or scar-related conduction barriers, to yield best electrical resynchronization.

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References
Figure 1 (A) Typical LBBB, depicting early RV activation (red), slow trans-septal conduction and delayed lateral LV activation (dark blue, terminal activation 173 ms). LV lead is located anterobasally (*) associated with qLV 75 ms (light green isochrones). (B) RV pacing alone—the paced wavefront reduces trans-septal delay inferoapically and accelerates LV activation—resolving the area of very late activation [replacement of dark (seen in A) with light blue areas]. (C) LV pacing pre-excites the anterolateral LV but inferior late activated regions persist. (LV only pacing results in optimal resynchronization in only a minority of CRT recipients with LBBB.) During simultaneous biventricular pacing (D), RV and LV paced wavefronts synergize to resynchronize biventricular and intra-LV activation (complete resolution of dark blue areas seen in A). These features illustrate successful electrical optimization achieved by coordinated LV and RV paced wavefronts—despite a ‘non-ideal’ LV lead position by current anatomical or electrical convention. LBBB, left bundle branch block; LV, left ventricle; RV, right ventricle.