Therapy for cardiac resynchronization: When left ventricular–only "fusion" pacing is not enough

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Introduction

In patients with left bundle branch block (LBBB), the right bundle branch (RBB) is responsible for left ventricular (LV) activation, following slow transseptal conduction (Figure, left). Once initiated, propagation across the LV free wall is usually rapid.[1] Cardiac resynchronization therapy aims to restore electrical synchronization. Conventionally, this is delivered by LV pacing to the site of latest LV activation. It is appealing to time this with intrinsic RBB conduction ("LV fusion" pacing) to avoid deleterious right ventricular (RV) effects of RV pacing. However, this assumes that LV stimulation elicits rapid and complete LV depolarization.

Case report

A 53-year-old man with ischemic cardiomyopathy (LV ejection fraction 10%), typical LBBB (QRS 184 ms), and PR 200 ms received an atriobiventricular pacing system. The LV lead was placed laterally (qLV 160 ms). Electrocardiographic image examination was conducted (Figure). In this case, LV pacing failed to generate confluent rapid LV activation. As a result, LV fusion pacing (at 180 ms atrioventricular [AV] delay) was insufficient in restoring electrical synchrony, which however was accomplished by adding RV pacing (biventricular pacing at the same AV delay).

Discussion

The case illustrates that pacing from the latest site of LV activation may not elicit optimal paced effect. This may limit the success of LV-only "fusion" pacing. However, biventricular stimulation at longer AV intervals may optimize LV activation.

LV pacing is the core component of cardiac resynchronization therapy. It aims to preexcite delayed LV activation. It is logical to time this with intrinsic RBB conduction ("fusion pacing") to attempt restoration of physiological biventricular activation. What is missing from this concept is that LV paced effects (and transseptal conduction via the RBB) vary from patient to patient.[1,2] Moreover, these cannot be predicted from baseline QRS morphology or qLV.[3] The interaction of pacing and conduction barriers is important. When LV pacing facilitates conduction block, it will retard LV activation compared to intrinsic conduction (Figure, middle). This effect is associated (unsurprisingly) with nonresponse. On the other hand, resolution of conduction delay permits recruitment of a greater proportion of the LV and diminished total activation time. This is associated with improved hemodynamic function.[4]

RV pacing may contribute to resynchronization. A randomized trial showed the benefits of baseline programming to achieve electrical synchrony using individualized AV and V-V timing on chronic structural remodeling.[5] Best results were achieved most often by biventricular pacing at critical AV intervals. In comparison, LV-only pacing was optimal in only a minority. This result appears puzzling, since it is known that committing ventricular depolarization to RV pacing only in heart failure patients is deleterious for both RV and LV function, and for clinical outcomes. Our results may explain these findings. Transseptal delay—the source of LV delay in LBBB—may have a functional basis that can be mitigated by RV pacing.[1,2] Hence the initial effects of RV pacing on septal conduction may be useful. We depict this condition with an

KEY TEACHING POINTS

- Pacing from the latest site of left ventricular (LV) activation in heart failure patients with left bundle branch block may not elicit confluent activation of the entire left ventricle owing to conduction barriers.
- Slow LV activation limits ability to restore cardiac resynchronization with LV pacing timed to intrinsic right bundle conduction ("LV fusion" pacing).
- Incorporation of timed right ventricular pacing with right bundle branch conduction and LV pacing ("triple fusion") may improve electrical resynchronization.

KEYWORDS CRT; ECG imaging; Fusion; LBBB; Left ventricle; Right bundle branch; Right ventricular pacing; Triple fusion
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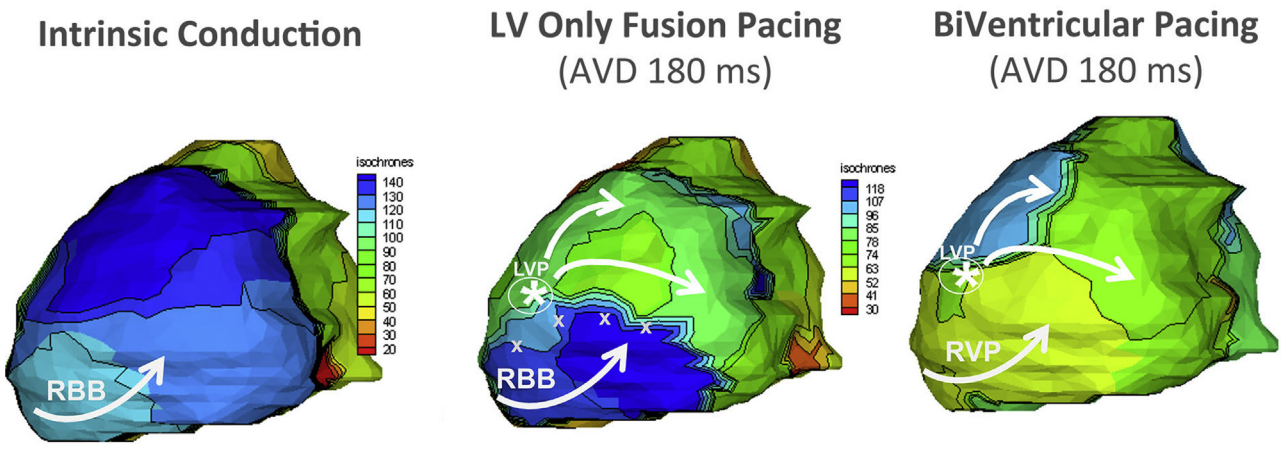

Figure Electrocardiographic imaged (ECGI) isochronal maps (timed to earliest onset of ventricular activation, ie, right ventricular [RV]) depicting left ventricular (LV) free wall activation in the left posterior oblique view under 3 conditions. **Left:** The LV free wall is activated by preserved right bundle branch (RBB) conduction and is delayed (dark blue isochrones) relative to RV activation. Following onset, LV activation is rapid (no isochronal crowding).[1] **Middle:** LV pacing (LVP; *) from point of latest LV activation during intrinsic conduction (qLV 160 ms) only partially restores rapid LV free wall depolarization. A conduction barrier (xxxx – isochronal crowding) prevents preexcitation of the inferolateral LV. **Right:** Biventricular pacing. The RV paced wavefront (RVP) depolarizes the inferolateral LV to synergize with the LVP effect to result in confluent LV depolarization.

electrocardiographic image (Figure right). Thus, biventricular stimulation at longer AV intervals (in this case 90% of the PR interval) introduces the initial effects of RV pacing as well as capturing intrinsic RBB conduction to deliver "triple fusion."

Conclusion

Our case illustrates a condition in which LV fusion pacing induces LV conduction barriers but the addition of RV pacing promotes more confluent LV activation. Individualized programming may be key to successful electrical resynchronization[5] (www.clinicaltrials.gov identifier NCT04100148).

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