LETTERS TO THE EDITOR

To the Editor — Multisite pacing strategies: Solutions looking for a problem?

A key indication for cardiac resynchronization therapy (CRT) is a broad QRS duration, indicating a dyssynchronous activation pattern. Recovering a synchronous activation with CRT is expected to improve cardiac function. Multipole (pacing from multiple electrodes on the same lead) and multipoint (pacing from 2 electrodes on different leads) studies, where initiating activation from multiple locations in the left ventricle (LV), is proposed to achieve a more synchronous activation and improve CRT response. Consistent with this synchronous activation hypothesis, the recent study by Heckman et al[1] finds that pacing from widely separated electrodes was beneficial and that no change in total activation time with multipole/multipoint pacing caused no improvement in acute hemodynamic response. However, while changes in LV activation times were reported in the abstract, their relationship with the acute hemodynamic response was not explored. This is unfortunate, because total activation times are harder to interpret, as they can be dominated by late right ventricle activation. Furthermore, the results may have been affected by attenuated underlying electrical dyssynchrony, smaller porcine hearts limiting electrode separation, and faster conductivity in a healthy compared to a failing heart reducing dyssynchrony. However, the conclusions reinforce the limited benefit of stimulating noninfarct LV from multiple sites.[2]

There is now compelling evidence that in noninfarct LV, multipole/multipoint pacing has limited benefit when the optimal pacing site can be accessed. In addition, multipole pacing has failed to show benefit in nonresponders to CRT in a large randomized trial (MORE CRT MPP study). However, there remain open questions about CRT response with multipole/multipoint pacing. Specifically, under what conditions do multipoint/multipole pacing benefit infarct patients, are lead designs optimized for multipole/multipoint pacing, and how far from an optimal pacing location must a lead be for a multipoint/multipole strategy to be of benefit? We hope that the excellent work coming out of Maastricht is able to help in answering these questions.

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Disclosures

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References

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2. Antoniadis AP, Sieniewicz B, Gould J, et al. Updates in cardiac resynchronization therapy for chronic heart failure: Review of multisite pacing. Curr Heart Fail Rep 2017;14:376–383.

Reply to the Editor — Regarding Multisite pacing strategies: Solutions looking for a problem?

The extensive intraindividual comparison of multisite pacing strategies requires the use of a preclinical animal model. We used large (~70 kg) pigs, who do not have smaller hearts than man, but admittedly develop less dyssynchrony from radiofrequency ablation–induced left bundle branch block as compared to humans.[1] However, we do believe that using the systematic intraindividual comparison of a large number of pacing regimes (varying sites and combination of sites) provides results that can at least qualitatively be compared to human patients.

Niederer et al rightly state that the relationship between acute hemodynamic response (AHR) and left ventricular activation time (LVAT) would be interesting. Although it was not described in the original publication, we did evaluate the relationship of the AHR with LVAT. The correlation between (normalized) AHR and total activation time (TAT) ($R = 0.47$) was somewhat stronger than the correlation between AHR and LVAT ($R = 0.40$). Also, we evaluated the correlation of AHR with the Q-LV interval, which was very poor (<0.1). This poor correlation is the result of the fact that Q-LV is more suitable as a patient selection criterion (identifying late LV lateral wall activation) and less for precise lead positioning on that LV lateral wall.[2] Therefore, we chose to elaborate on the interaction of AHR and TAT in the last section of the Results paragraph. Also, the use of LVAT in daily clinical practice is limited compared to TAT, as this is measured as QRS duration on the standard 12-lead electrocardiogram.

Since in our study multipoint pacing and multizone pacing failed to produce a significantly larger hemodynamic improvement compared to optimal biventricular pacing (highest 25% group), we agree with the authors that multi-LV pacing has limited benefit when the optimal pacing site can be accessed. The authors state that this is particularly true in the noninfarcted LV. However, we would like to emphasize that there is no solid proof that multipoint or multizone pacing are of benefit, particularly in ischemic patients. In fact, there is only 1 publication on direct comparison of multiple LV configurations, where in fact no difference in AHR was found between multipoint pacing and multivein pacing, although ischemic cardiomyopathy patients were included.[3]

Lastly, we do agree with the authors that questions remain on the effectiveness of multi-LV activation in the infarcted LV.

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