Comment on the article by Trolese T et al.

The recent introduction of quadripolar leads for cardiac resynchronization therapy raises the question which pacing vector is most beneficial to response and which parameter gives insight into the optimal vector. The study conducted by Trolese et al. is therefore of important value and unique in its kind.\(^1\) The found correlation between maximal difference of the QRS-width ($\Delta$QRS) and acute haemodynamic response (AHR) ($\Delta$LV $dP/dt_{max}$) is an important finding to aid vector selection. However, their results give rise to questions.

Figure 1A of Trolese et al. shows an equal QRS morphology comparing M3M2 and M3P4 indicating cathodal stimulation from M3 and no effect of the anodal electrode. However, comparing D1M2 and D1P4 shows a clear difference in morphology. Because the cathodal electrode (D1) is the same in both configurations, this can be explained by anodal capture from either M2 or P4, implicating that in one of the configurations left ventricular (LV) pacing is a dual site and a single site in the other. This will affect QRS-width as well as AHR and makes the comparison between configurations problematic. Figure 1 of this letter gives an example of changes in QRS-morphology with anodal capture. Most probably, there is also a typographical error in Figure 1A of Trolese et al. regarding the QRS-width of baseline compared with D1M2.

Second, the change in QRS-morphology between vectors P4M2, P4RV, M3M2 and all preceding vectors in Figure 1B of Trolese et al. suggests that the LV pacing wavefront becomes less dominant (lead I less negative and lead V1 less positive) when the proximal electrodes are used. Electrodes M3 and P4 are possibly closer to the base of the ventricle, and would need pre-excitation (changes in VV-delay) to depolarize a substantial part of the LV when pacing biventricular. Acute haemodynamic response of these vectors could be underestimated by a reduced contribution of LV depolarization by the LV pacing wavefront. To show the true effect of LV pacing vectors on AHR, LV pre-excitation had to be programmed while pacing at proximal electrodes. Unfortunately implementation of VV-delays in the study protocol is shear impossible, as it would result in long procedures.

Third, the analysis of Trolese et al. rises methodological questions. Although randomization of the pacing sequence and baseline measurements in-between pacing were performed,\(^2\) outliers have large effect on findings, even while considering the small sample size ($n = 16$).\(^2\) Repetition of measurements and curve fitting with implementation of several AV-delays could be a better approach, however leading to an undesired longer procedure. Increasing the sample size could also decrease the effects of outliers.

Lastly, based on the above-mentioned arguments and known intra-individual spread in correlation of inter-ventricular delays (IVDs) and AHR, the conclusion that vector selection based on IVD is not supported, could be premature. Zanon et al.\(^4\) demonstrated that the correlation of QLV-interval (comparable with IVD) and LV $dP/dt_{max}$ is quite variable between patients and strongly correlated intra-individually. We are, however, delighted by the articles published by Trolese and colleagues, as the introduction of quadripolar LV leads raise questions on optimal vector selection. However, the methodology and conclusions of their articles are up for debate.

References


Wouter M. van Everdingen\(^1\)*, Berry van Gelder\(^2\) and Mathias Meine\(^1\)

1. Department of Cardiology, University Medical Centre Utrecht, Utrecht, The Netherlands; 2. Department of Cardiology, Catharina Ziekenhuis Eindhoven, Eindhoven, The Netherlands. *Corresponding author. Tel: +31 887553097; fax: +31 88755471. E-mail address: w.m.vaneverdingen@umcutrecht.nl

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Figure 1Stimulation on vector P4M3. Continuous electrocardiogram registration of stimulation on vector P4M3, with increased output from 2.8 to 3.3 V. Note the changes in QRS-morphology after increased output (especially lead I, II, aVR, V5, and V6), indicating anodal capture.