Biventricular pacing and transmural dispersion of the repolarization

In 2003, Medina-Ravell et al.\textsuperscript{1} published a study that suggested that biventricular pacing could be arrhythmogenic in a subset of patients because of the reversal of the normal endocardial-to-epicardial activation sequence by left-ventricular epicardial pacing. As epicardial action potentials are briefer than endocardial action potentials, transmural dispersion of the repolarization (TDR) is larger with an epicardial-to-endocardial activation sequence than with an endocardial-to-epicardial activation sequence. Medina-Ravell et al. demonstrated this in an isolated arterially perfused rabbit left-ventricular wedge preparation. They also showed, in a quasi-ECG derived from this preparation, that TDR was faithfully reflected by the $T_{\text{peak-end}}$ interval and that $T_{\text{peak-end}}$ was larger with epicardial pacing than with endocardial pacing of the preparation. Their conclusion that a similar effect occurs in intact hearts in humans was substantiated by the observation that in 29 heart failure patients with a biventricular pacemaker, the $T_{\text{peak-end}}$ interval was larger during left-ventricular epicardial pacing than during right-ventricular endocardial pacing. However, no $T_{\text{peak-end}}$ Values with sinus rhythm and with biventricular pacing were reported, due to measurement difficulties.

With interest, we read the recently published study by Santangelo et al.\textsuperscript{2} They describe how left-ventricular, right-ventricular, and bi-ventricular pacing in heart failure patients influences a number of ECG indexes of ventricular dispersion of the repolarization, among others the $T_{\text{peak-end}}$ interval. Compared with sinus rhythm, the $T_{\text{peak-end}}$ interval increased with left-ventricular epicardial and with right-ventricular endocardial pacing, but it decreased with bi-ventricular pacing. The observations by Santangelo et al. confirm nicely the findings of our recent study\textsuperscript{2} in which we evaluated, in a similar way, the effect of left-, right-, and bi-ventricular pacing on a set of ECG indexes thought to represent ventricular dispersion of repolarization, among which was $T_{\text{peak-end}}$. We also found the briefest $T_{\text{peak-end}}$ interval to occur with biventricular pacing.

What should be the conclusion from these observations? Medina-Ravell et al.\textsuperscript{1} conclude that, in their patients, epicardial left-ventricular pacing increases TDR with respect to right-ventricular pacing, because the $T_{\text{peak-end}}$ interval is the largest with left-ventricular pacing. Santangelo et al.\textsuperscript{2} conclude, among others, on the basis of the behaviour of the $T_{\text{peak-end}}$ interval, that left-ventricular and right-ventricular pacing increase TDR with respect to sinus rhythm, whereas bi-ventricular pacing decreases TDR. The implications of such a conclusion would be that any single-lead ventricular pacing, be it sole endocardial right-ventricular or sole epicardial left-ventricular pacing, would be undesirable because it increases TDR. It also implies that the thus-created disadvantageous situation can be turned to being advantageous by simultaneous biventricular pacing. Would that mean that the inversion of the endocardial-to-epicardial activation by epicardial left-ventricular pacing is undone by simultaneous right-ventricular pacing? The BELIEVE study,\textsuperscript{3} in which 74 heart failure patients who were randomized to left-ventricular only or to bi-ventricular pacing were followed for 1 year, was not able to detect any evidence for a proarrhythmic effect of left-ventricular pacing.

Our conclusion, supported by computer simulations\textsuperscript{3} is that the $T_{\text{peak-end}}$ interval in the ECG of intact humans is not reflecting TDR, in contrast to the $T_{\text{peak-end}}$ interval in the quasi-ECG made in the left-ventricular wedge-preparation. The surface ECG made from a whole heart and the quasi-ECG derived from a preparation of the left-ventricular free wall are not analogous because surface ECG electrodes record, from a distance, electrical activity in the whole heart; in the above studies, cancellation plays a prominent role.\textsuperscript{2} The quasi-ECG is, however, recorded close to a small preparation in which cancellation plays virtually no role. The peak in the T-wave in the surface ECG reflects septal repolarization rather than, e.g., repolarization of the endocardium in the case of left-ventricular epicardial pacing.

Although it may well be true that in intact human hearts with left-ventricular and with biventricular pacing, TDR may be locally increased under the left-ventricular epicardial electrode, this is not reflected in the $T_{\text{peak-end}}$ interval on the surface ECG.

References


