Biventricular pacing and transmural dispersion of the repolarization

In 2003, Medina-Ravell et al. 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_peak-end interval and that T_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_peak-end interval was larger during left-ventricular epicardial pacing than during right-ventricular endocardial pacing. However, no T_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. They describe how left-ventricular, right-ventricular, and biventricular pacing in heart failure patients influences a number of ECG indexes of ventricular dispersion of the repolarization, among others the T_peak-end interval. Compared with sinus rhythm, the T_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 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_peak-end. We also found the briefest T_peak-end interval to occur with biventricular pacing.

What should be the conclusion from these observations? Medina-Ravell et al. conclude that, in their patients, epicardial left-ventricular pacing increases TDR with respect to right-ventricular pacing, because the T_peak-end interval is the largest with left-ventricular pacing. Santangelo et al. conclude, among others, on the basis of the behaviour of the T_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, 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 is that the T_peak-end interval in the ECG of intact humans is not reflecting TDR, in contrast to the T_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. 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_peak-end interval on the surface ECG.

References


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