Modulation of ECG Atrial Flutter Wave Amplitude by Heart Motion: A Model-based and a Bedside Estimate

V Jacquemet, B Dubé, P van Dam, AR Leblanc, R Nadeau, M Sturmer, T Kus, A Vinet

Université de Montréal
Montréal, QC, Canada

During atrial arrhythmia, ventricular contraction may modulate the morphology of the atrial contribution to the ECG by moving or constraining atrial geometry. We hypothesized that ventricular contraction induces only small changes in atrial signal amplitude on the thorax. Our aim is to estimate an upper bound for this effect as it could affect the interpretation of QRST cancellation.

Pseudo-orthogonal 3-lead ECG/VCG was recorded during 1 hour at rest in a patient with stable atrial flutter, atrio-ventricular block and a pacemaker programmed to deliver ventricular stimuli at a fixed rate of 40 bpm. Intervals between R waves and flutter waves were uniformly distributed (p=0.28; Kolmogorov-Smirnov test). Under the assumption that atrial signals are amplitude-modulated periodic waves, average beat subtraction eliminates the ventricular contribution to the ECG. Flutter wave amplitudes were extracted from the corrected VCG using min/max filters with window length of one flutter period. These amplitudes were analyzed over three intervals: during the T wave (320 ms), during the U wave (400 ms) and in the subsequent isoelectric interval (400 ms).

Flutter waves were also simulated using a previously-developed atrial model embedded into a time-dependent volume conductor model of the thorax in which atrial geometry was adjusted to MRI data acquired at 50 time instants during normal rhythm (worst-case estimate of geometrical effects).

Average flutter wave VCG magnitude in the three intervals was 309 ± 6 μV vs 308 ± 5 μV vs 314 ± 4 μV in the simulation, and 335 ± 36 μV vs 329 ± 28 μV vs 332 ± 24 μV in clinical data. Although variations of $>10\%$ were observed of some leads, results suggests that heart motion may be neglected for atrial activity cancellation.