Interactive Simulation of the Activation Sequence: replacing effect by cause

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Electrocardiographic wave forms during the QRS interval reflect the activation sequence of the heart. This can be clearly demonstrated in ECGSIM, an interactive software package aimed to support ECG education and re-search. In this package, the timing of local depolarization may be changed interactively, either locally or globally and the resulting QRS changes are displayed instantaneously. The activation sequence itself depends on the distribution of the sites of initiation of the activity, the initiation stemming from bundle branches and Purkinje system, as well as on the tissue properties setting the propagation velocity of the activation process. In a recent paper (van Dam et al. MBEC 2009), we have shown that high-ly realistic activation sequences can be generated by defining a limited num-bers of initiation (focus-like) sites in a numerical model of the propagation in the myocardium. The anisotropic velocity of the propagating wave front was assumed to be uniform throughout the myocardium, with the transmural velocity set at one third of the velocity parallel to the ventricular surface. Inspired by the high quality of these simulations, we have explored the usefulness of interactively changing the intrinsic, anisotropic, propagation velocity values at selectable regions (non-uniform distribution of intrinsic propagation velocity). Combined with specified sites of initiation, such changes (the cause) produce, in a direct, natural manner, changes in the acti-vation pattern (the effect). The velocities that are currently considered for implementation in ECGSIM are their spatial distribution at the endocardium, epicardium propagation velocity as well as the transmural distribution. When simulating local ischemia, next the effects already incorporated in ECGSIM: increased resting potential and shortened action potential duration, the effect of the accompanying reduced propagation velocity may now be studied. Other applications are the study of effects of activation delays as occurring in left ventricular hypertrophy and Brugada syndrome.