of transmural distribution of the intrinsic action potential durations of the myocytes, 1 homogeneous, and the other 2, heterogeneous. The following markers of local repolarization are considered: RT_v = time of minimum TAP derivative during the downstroke, and RT90_v = time when TAP reaches 90% of the resting value during the downstroke. These 2 markers are generally considered to be the best standards for evaluating other markers that assess RTs from EG recordings, such as RT_ue = time of maximum EG derivative during the T wave. However, several authors use the variant RT_ue+ = time of minimum EG derivative when the unipolar T wave is positive. We also considered the marker RTd2_ue = time of minimum second derivative during the T wave.

Results: Our results show that the mean discrepancy between RT_ue (RTd2_ue) and RT_v (RT90_v) is 2.1 (1.2) milliseconds with SD of 1.4 (0.9) milliseconds. The variant RT_ue+ produced worse estimates of RT90_v than RTd2_ue, with average discrepancy 6.26 milliseconds and SD 1.45 milliseconds, yielding 2 incoherent estimates of RT90_v.

Conclusions: We conclude that, in a normal tissue, RT_ue and RTd2_ue are very accurate estimates of RT_v and RT90_v, respectively, independently of T-wave polarity, repolarization sequence, and transmural distribution of intrinsic properties of the cell membrane.

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MOD-2

Investigations of 3-dimensional approximated thorax model applied to simulation of cardiac electrical field[☆]

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Introduction: The work presents the results of investigations of 3-dimensional approximated model of thorax applied to the simulation of cardiac electrical field to determine the epicardial maps on a basis of measured body surface potential maps solving the inverse problem. The model is created using the image data referred to the contours of the radiographic images combined with the external chest sizes of each subject. The aim was to find the influence of speeding up the process of creation of the model on the accuracy of epicardial maps.

Methods: The main factor affecting the time of creation of the model is the process of segmentation of image data. The examined models were created applying two methods of segmentation: (*a*) semiautomatic—based on the split and merge technique and (*b*) automatic—based on threshold and edge detection operations. Contours for creation the reference model were made in manual way.

Results: For evaluation of effects of speeding up the creation of the model, determination of epicardial maps was carried out for 5 healthy subjects based on body surface potential maps applying 3 models of thorax: the reference model, the model created applying semiautomatic method, and the model created applying the automatic method. Differences between maps determined using the reference model and maps using the model created applying the semiautomatic method were less than the differences between maps determined using the reference model and maps using the model created applying the automatic method.

Conclusions: In both cases, the locations of negative and positive extremes are similar. However, differences between the values of global extremes are greater in the case referred to automatic method (about 7%) then in the case referred to semiautomatic method (about 4%). However, this 7% accuracy was sufficient for the proper diagnosis. It is convenient in cardiac diagnosis carried out for large number of examined patients.

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MOD-3

Effect of ischemic action potentials on T and U Waves in the electrocardiogram

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Introduction: The T and U waves reflect the repolarization process of the cardiac ventricles. The repolarization process is influenced by ischemia through the ionic exchanges across the cardiac membranes. The effect is a shortening of the action potentials (APs). Here, we investigate the effect of location and size of ischemic areas on T and U wave.

Methods: The model is a stylized slice of the left ventricle of 1961 hexagonal cells in a single layer. Action potentials are assigned to each cell; their timing follows a simulated excitation sequence. The potential differences between the APs of adjacent cells produce time-varying electrical sources, which contribute to the potential in point P in proportion to its transfer function. The electrocardiogram at P is the sum of all potential contributions. In the anterior wall, 3 areas were studied: endo-, mid, and epicardial. Size of the ischemic areas varied from 1×16 to 10×26 mm. Ischemia was simulated by shortening of the APs in the 3 areas. The APs were shortened in steps of 20 ms, with a maximum of 80-millisecond shortening. Observation point P was located anteriorly at 4 cm from the epicardium, that is, comparable to a V_2 location.

Results: Epicardial ischemia increased peak T amplitude with very little effect on the U wave. The end T amplitude increased proportional to the peak T amplitude, obfuscating the TU nadir. Endocardial ischemia gave a size-dependent reduction in T amplitude and very little effect on the U wave. With increasing midmyocardial ischemia, the effects are far less dramatic and only seen in a subtle decrease of T amplitude and reduction in TU nadir amplitude to zero or negative. Simulated ischemias never led to a QT duration change, except for the endocardial largest area, which produced a QT reduction of 12 milliseconds. Paradoxical QT lengthening was not seen.

Conclusions: The gross TU wave changes of ischemia are dominated by the endo- and epicardial changes, and midmyocardial ischemia is more readily seen in subtle T and U wave changes.

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MOD-4

Why we need supercomputers to understand the electrocardiographic T wave?

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Introduction: Propagation of depolarization and repolarization in myocardium result from an interplay of membrane potential, transmembrane current, and current flow between the cells ("electrotonic coupling"). This process can be represented mathematically with a reaction-diffusion (RD) equation. Even today, solving RD equations for a whole heart still requires a supercomputer. Earlier models relied on predefined action potential (AP) shapes and fixed propagation velocities. It is our purpose to explain the difference between these models for a nonspecialized audience and to show why RD models are important when electrocardiographic T waves are studied.

Methods: We simulated propagating AP with an RD model of the human heart, which included transmural and left ventricle–right ventricle heterogeneity of membrane properties. Computed activation times served as input to a model that used predefined AP and to a "hybrid model" that computed AP only in the repolarization phase. Fixed APs were obtained from simulations of isolated cells and represented the same heterogeneity of cell types as the heart model. The RD model had a spatial resolution of 0.25 mm. The hybrid model was tested with different spatial resolutions. Electrocardiograms were computed with all 3 models.

Results: As expected, computed QRS complexes were practically identical in all models. T waves in the fixed-AP model had 20% to 40% larger amplitudes in the precordial leads V_1 to V_3, became biphasic in lead III, and notched in leads II and aVF. In contrast, the hybrid model produced the same T waves as the RD model at 0.25-mm resolution but underestimated T-wave amplitude in V_1 to V_{3u} at lower resolutions.

Conclusions: Fixed AP waveforms in a forward electrocardiographic model lead to exaggerated T waves. Hybrid models only equal RD models when used with the same spatial resolution. Thus, a model that can be trusted to smulate electrocardiographic T waves correctly still requires a supercomputer.

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