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Medical physics

Explaining the T-wave shape in the ECG

The heartbeat is recorded on an electrocardiogram (ECG) as a characteristic trace determined by changes in the electrical activity of the heart muscle. The T wave is a component of this waveform that is associated with the repolarization phase of the action potentials¹. It is asymmetrical in healthy subjects, but tends to become symmetrical with heart disease². The reason for the T-wave shape is not clear³. Here we show that T waves become more symmetrical as a result of an increase in the dispersion of the regional repolarization of cardiac muscle.

The exact sequence of depolarization and repolarization of the action potential (the potential difference that arises between the intracellular and extracellular fluid) in three-dimensional heart muscle is complex, but to model the electrocardiogram on the body surface, only the heart surface potentials need to be known⁴, assuming that the myocardium has uniform and isotropic conductivity. Although the repolarization

on any individual cell is timed to the initiation of its action potential, repolarization occurs smoothly and systematically across the epicardial surface^{5,6}. Repolarization of the surface of the left ventricle occurs first epicardially in the postero-basal region, then at the septal wall and apex⁶, and finally at the endocardial surface⁷.

We used this spatial sequence on a simple model of the left ventricle which allowed the body-surface 12-lead T waves of the ECG to be calculated with the standard repolarization phases of an action potential⁸ and standard modelling equations⁴. The body was represented by an elliptical cylinder⁹ and the heart by a truncated ellipsoid (axis diameters, 7.0 and 6.6 cm; height from base, 7.4 cm; displaced 4 cm left and 6 cm forward of the body axis, and rotated 25 degrees forward and 40 degrees to the left).

We evaluated this model twice: by using a fixed, normal shape for the action potential, and then by using three regions, each with a different shape for the action potential, with a 10% increase in model parameters⁸ at the endocardium and with a 10% decrease at the apex. These changes to the parameters generated abnormally shaped action potentials. To make sure that the results did not depend on the specific model values, we calculated the form of T waves for the initial heart position and for separate shifts of the axes of ± 2 cm and rotations of ± 10 degrees, to calculate error bars.

To represent differences in the regional dispersion of repolarization times, we varied the time delay between the first and last action potentials, and thus between the earliest and latest region to repolarize, from 10 to 150 ms in steps of 10 ms, which ranges from normal to abnormal dispersion, and linearly interpolated the time delays for intermediate regions. We calculated a symmetry ratio from the body-surface T waves as the ratio of the areas under the two sections of the T curves

(beginning-to-peak compared with peak-to-end).

Our results show that low dispersion is represented by asymmetrical T waves, and high dispersion by increasingly tall and symmetrical, clinically hyperacute T waves that tend to a symmetry ratio of unity (Fig. 1). This agrees with the accepted symmetry ratio for normal T waves of 1.5 (ref. 10) and with the expected relation between tall, symmetrical T waves and abnormal repolarization. Abnormal shapes in the action potential result in differences in the T-wave symmetry ratio, but still tend to produce symmetrical T waves for high dispersion.

This finding can be explained by considering a simplified situation, based on the assumption that the heart contains only two regions, which give rise to action potentials that have identical shapes but are displaced in time. The resulting potential gradients, and ultimately the shape of the T wave, can be approximated to the difference between the two action potentials at each instant. When dispersion, or time displacement, is small, this difference will result in an asymmetrical waveform (as a consequence of the shape of the action-potential repolarization phase), and when it is large (with the second action potential beginning to repolarize after the first has mostly repolarized), a symmetrical waveform will tend to be produced.

Such a simple model can be expressed mathematically and computed easily but is not convincing without the calculations used here, with more realistic heart and torso geometries and gradual differences in the initiation of action-potential repolarization across the myocardium. Confidence in the model is increased by the resulting T-wave shapes in the 12 ECG leads.

Analysis of T-wave symmetry offers a new clinical indication of dispersion, which would be valuable for patients with high dispersion, who are more likely to die suddenly¹¹; the current methods used to measure dispersion are unsatisfactory and are prone to errors¹². Our results indicate that there is a link between T-wave symmetry and the abnormal regional dispersion of repolarization.

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Figure 1 T-wave symmetry ratios for a range of dispersions of repolarization for the two implementations of the model (blue circles, single action potential; red triangles, three regions with different APs) for the mean of precordial leads V2 to V6. V1 was omitted as it was sometimes biphasic. Lines are drawn through the symmetry ratios for the initial heart position; error bars give the maximum range calculated for the different heart positions. The mean difference between limb and precordial leads for the symmetry ratio was 0.026 for the model of the single action potential, and 0.005 for three action potentials. The graphs at the top show the T waves for the single normal action potential in an example lead V2; each grid square is 0.5 mV tall and 200 ms wide, as in standard ECG paper recordings.

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