# THE EQUIVALENT SURFACE SOURCE MODEL IN ITS APPLICATION TO THE T WAVE

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## INTRODUCTION

The recent revival of interest in the diagnostic properties of the T wave calls for a model of the genesis of the normal T wave against which observed wave forms in cases of disease can be contrasted. The ECG wave forms as observed at some distance from the active tissues can only be interpreted correctly in a physiological sense if appropriate models are incorporated to describe both he source and the volume conduction effects of the surrounding tissues.

Most ECG textbooks include some notions related to the genesis of the T wave. Correct as these may be in a general sense, they do not do justice to the full complexity of the problem. Cardiac sources have a spatio-temporal character. The temporal features dominate the observed potentials and tend to obscure the spatial features.

In this paper the full spatio-temporal character of the sources is represented by an equivalent surface source situated at the boundary of ventricular tissue. The local source strength is assigned the typical shape of the transmembrane potentials of ventricular muscle cells. The wave form as such is taken to be the same at all points of the ventricular surface, but the timing of local depolarization and of repolarization at these points is specific. When combined with a realistic volume conductor model of the thorax, this model has previously been shown to yield an accurate description of the potentials during QRS. More recently it was demonstrated that this model could also be applied to the genesis of the T wave [1]. This approach is similar to the one of Harumi *et al.* [2].

In the present study the required "timing" of repolarization was obtained by means of an inverse procedure applied to measured body surface potentials. The view on the repolarization "sequence" that one obtains in this way is discussed, in particular with respect to the discussion about the significance of the QT dispersion [3].

## THEORY

Any simulation of ECG- or electrogram wave forms requires a specification in physical terms of the electric sources, preferably linked directly to the underlying electrophysiological phenomena: one needs a *source model*. In addition one needs to specify the way in which these sources feed their current into the surrounding tissues, thus setting up the potential differences that are recorded by means of the electrodes used (the lead system): one needs to specify a *volume conductor model*. Both models need to be specified completely: an incomplete specification leads to confusion regarding the electrophysiological interpretation of the observed phenomena.

### The Equivalent Surface Source Model

The source model used in this work is the equivalent surface source model (ESS). In this model, the entire electrical activity within the ventricles is expressed by means of an electric double layer source situated on the closed surface $S_h$ bounding all ventricular cells. For any position on $S_h$ the time course of the local source strength is taken to be proportional to the transmembrane potential, $\phi_m(t)$, of the cells near the boundary. This type of source model is directly related to the classical uniform double layer model. In its application to the depolarization phase, we previously arrived at the equivalence of the sources at the actual wave front of depolarization and model sources on $S_h$ . At that stage [4] the equivalence was derived from the solid angle theory [5,6].

The application of the ESS to the repolarization phase, and to the genesis of the T wave derives its justification from an analysis by Geselowitz. In 1989 Geselowitz [7] introduced the ESS as based on analysis of the electrical properties of ventricular cells treated as a homogeneous syncytium by means of the bidomain approach. In that paper Geselowitz stipulated that this approach is only valid if the bidomain is isotropic, but in a subsequent paper [8] he showed that this restriction is lifted in situations where the ratio of extracellular to intracellular conductivity in the fiber direction can be taken to be the same as the corresponding ratio in the cross-fiber direction. This so-called *equal anisotropy ratios assumption* dominates the application of the equivalent surface source model. The justification for this approach stems from the fact that the measured wave front strength during depolarization in the extracellular part of the ventricles in the fiber direction is approximately the same as that in the cross-fiber direction [9]. The equivalent surface source model has a direct link to the underlying electrophysiology. It does justice to the fact that:

*if all ventricular cells would depolarize in full synchrony, following the same time course, the amplitudes of the resulting QRS complexes would be zero*.

Similarly,

*if all ventricular cells would repolarize in full synchrony, following the same time course, the amplitudes of the resulting T waves would be zero*.

This fundamental property of the ESS relates to the fact that a closed double layer does not set up any potential difference (gradient) in the medium surrounding it. The corresponding statement related to a uniformly polarized single cell is well known. For the T wave, as is shown below, it has the direct implication that the amplitude of the T wave is proportional to the dispersion of repolarization times.

The validity of the ESS model can be disputed, in particular regarding the assumptions of homogeneity and the equal anisotropy ratios. In this paper its practical value will be demonstrated. Simulated T waves based on the ESS that have previously been reported in the literature can be found in [10,11,12].

**The Timing Of Repolarization**

In this work the entire ventricular surface $S_h$ carrying the ESS has been sub-divided into small elements $\Delta S_h$. The nature of a source at each element is that of a small current dipole that is oriented normal to $S_h$ and points towards the ventricular tissue. The positions of these elementary dipoles are referred to as the *nodes*. The strength of the dipoles are proportional to the area of $\Delta S_h$ around the nodes. The local time course of the source strength of any node $n$, ($n$=1, …., $N$), has been assigned the shape shown in Fig. 1. This shape approximates the time course of the transmembrane potential of ventricular muscle cells.

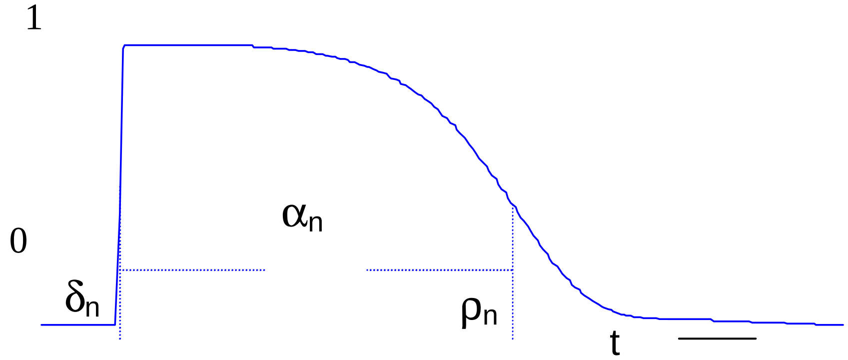


***Figure 1***. *Time course of the normalized source strength* $S_n(t)$ *at node* $n$.

The general shape of this curve is identical for all nodes. The time instant of the maximum positive slope specifies the moment of local depolarization. For node $n$ it is denoted as $\delta_n$. Similarly, the time instant of the maximum negative slope is taken as a marker for the timing of local repolarization. For node n it is denoted as $\rho_n$. This is the most accurate single parameter for specifying the timing of repolarization, short of some parameter related to any specific ion transport across the membrane. It can be identified with far greater accuracy than, say, the 'end' of the depolarization phase.
The interval $\alpha_n = \rho_n - \delta_n$ is a measure of the local action potential duration (APD) [13]. Because of the scaling applied to the the local source strength (see below) the time integral of the curve shown in Fig. 1 closely approximates $\alpha_n$.

## MATERIALS AND METHODS

### Source Specification

As discussed above, the ESS can be represented by elementary current dipoles located on the ventricular surface $S_h$. In this study the shape of $S_h$ was obtained from magnetic resonance images (MRI) of a healthy subject. In all $N$=257 nodes were evenly distributed over $S_h$. Since a closed double layer does not generate any external field, the minimum source strength can be assigned the value zero. The maximum double layer strength around each node was 40 mV. This value stems from the amplitude of the up-stroke of the transmembrane potential, scaled by the local conductivities of the tissues [9]. The magnitude of the source strength was incorporated in the lead field expressing the volume conductor (see below). As a consequence, a normalized (dimensionless) maximum value of 1 can be used for the local source strength around the nodes, the value shown in Fig. 1. The values specifying the local timing of depolarization, $\delta_{n,}$ as well as those of repolarization, $\rho_n$, were found by means of an inverse procedure applied to measured body surface potentials. For details, see [1].
The set of $N$ depolarization times $\delta_n$ constitute a vector $\delta$. The set of repolarization times, $\rho_n$, can similarly be interpreted as elements of a vector $\rho$, and the values $\alpha_n$ as elements of a vector $\alpha$, (vectors in the sense of linear algebra). Following this notation we may write the time course of the source strength of node $n$ as

(1) $s_n(t)=s_n(t\,;\,\delta_n,\,\rho_n)$.

During depolarization the 'turning on' of the local source strength of surface elements follows the wave form of the local transmembrane potential. The duration of this phenomenon is much smaller than that of the QRS-complex; the precise wave form of the upstroke plays only a minor role. In contrast, the duration of the repolarization process is much longer than the dispersion in the timing taken to represent 'the' moment of repolarization. As a consequence, the shape of the transmembrane potential during repolarization has to be chosen with care. In the present study the shape of the down-slope shown in Fig. 1 was derived from the integral over time of a weighted mean of the T waves measured on the subject; see [1].

### Measured potentials

The body surface potentials of the subject were recorded using the $L$=64 leads of the Nijmegen lead system [14]. This lead system has the standard 12-lead ECG as a subset. A 500 ms interval of QRST signals was sampled at a rate of 500 samples/s. Linear baseline correction was applied. The data were stored in a matrix $\mathbf{V}$ of dimension $(64\times250)$.

### Volume Conductor Model

A piecewise homogeneous, multi-compartmental volume conductor model was used. It included the inhomogeneities of the lungs and the blood-filled cavities. The conductivity values of these compartments relative to the overall conductivity of the body, were: lungs 0.2, cavities 3.0. Their geometry was extracted from MRI data of the (healthy, male) subject. Based on this volume conductor model the transfer was computed between the source strength of all of the $N$ nodes and all of the L=64 leads on the thorax. This computation was carried out by means of the Boundary Element Method. For more details, see [1,5,6]. The resulting transfer can be denoted by means of a matrix, $\mathbf{A}$, comprising the $(64\times257)$ transfer coefficients involved. Element $a_{n,t}$ of this matrix expresses the contribution of the instantaneous source strength $s_n(t)$ to the potential $\phi_l(t)$ at lead I on the thorax. The total value of the potential, $\phi_l(t)$ is found by adding up all contributions of the individual nodes. This summation can be denoted as

(2) $\phi_l(t) = \Sigma_n \, a_{l,n} \, s_n(t).$

By evaluating this expression at $t = 1 \ldots T$ discrete time instances the entire implied forward computation is denoted as

(3) $\mathbf{\Phi} = \mathbf{A}\,\mathbf{S} = \mathbf{A}\,\mathbf{S}\{\delta\,,\,\rho\}$

with **S** a matrix having (257×250) elements $s_{n,t}$, each representing the source strength around node $n$ at time instant $t$.

## RESULTS

The inversely computed timing on $S_h$ of depolarization, δ, and of repolarization, ρ, when introduced to the source specification in Eqn.3, produced QRST wave forms that closely resembled the measured data. The relative mean square difference, computed over all time instances and over all of the 64 leads involved was 0.17. Figure 2 depicts the standard 12 lead signals, both of the measured data, 12 signals selected from **V**, and of the simulated data, the corresponding subset of $\mathbf{\Phi} = \mathbf{A}\,\mathbf{S}$. The layout of this figure, a "map montage", has been designed such that the position of the signals relative to one another is similar to the corresponding lead placement on the thorax. Shown are the standard leads V1 to V6 as well as Vra, Vla and Vlf.

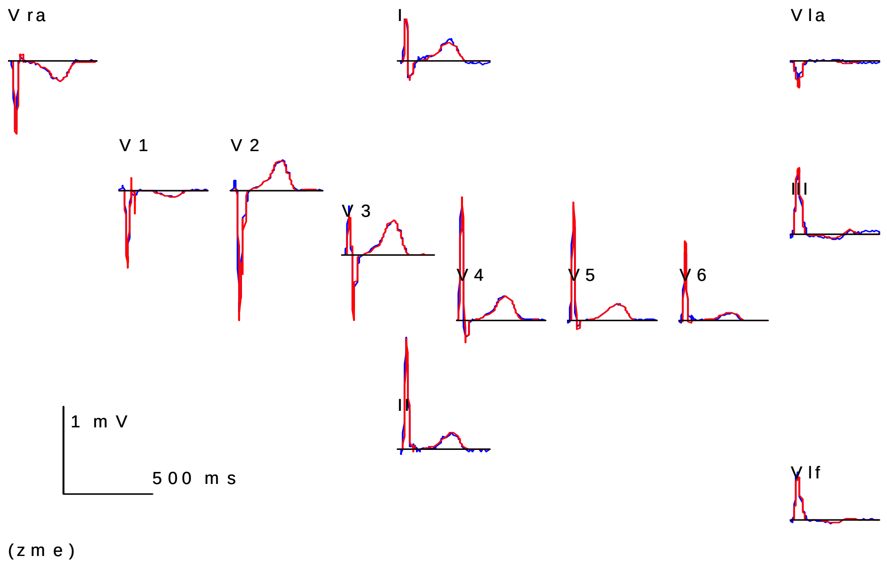


***Figure 2***. *Standard 12-lead ECG shown in a "map montage".*
*Solid lines: measured data; dashed lines: simulated data. The fact that differences between these line types can hardly be distinguished demonstrates the high quality of the ESS based simulations.*

These leads are unipolar leads, recorded with reference to Wilson's Central Terminal. The bipolar leads I, II, III are shown as well, placed at positions similar to those of the corresponding potential gradients on the thorax. This type of display depicts the signals in a manner that stresses their link with the underlying topology of the cardiac generator.
The inversely computed timing of depolarization used in this simulation showed a qualitative nature that resembles that of invasive measurements [15]. For the repolarization timing no complete set of invasively

obtained data is available. In the first publication on this topic [1] details of the inversely computed timing of depolarization and of repolarization were presented in the form of isochrone maps on $S_h$. Here the results are shown of a regression analysis on the individual elements of timing . Figure 3. depicts the scattergram of the elements of the APD, $\alpha_n = \rho_n - \delta_n$ , plotted as a function of the corresponding depolarization times $\delta_n$ . Different symbols are used for the data of nodes on different subsections of $S_h$ . The slopes of the linear regression lines for these subsections (not shown) were -1.75, -1.45, -1.45 and -0.98 for nodes of $S_h$ in contact with the left ventricular cavity (LVC), those in contact with the right ventricular cavity (RVC), those on the epicardium of the left ventricle (epiLV) and those on the epicardiun of the right ventricle (epiRV), respectively. For all data points the slope of the regression line, shown in Fig. 3, was -1.44.

## DISCUSSION

The results shown in this paper, Fig. 2, demonstrate that the Equivalent Surface Source model of the cardiac electric generator is capable of producing high quality simulations of the electrocardiographic wave forms. The timing of the depolarization is very similar to what is known from invasive electrophysiology [14,1]. The general nature of the repolarization time as shown by the regression lines of APD versus the depolarization timing, Fig. 3, is in full agreement with what is known from the scarce, fragmented, invasive data that have been published in the literature [13,16], in particular in [16], where a mean value of -1.44 was reported for the slope of the regression of measured APD values *vs.* depolarization times.

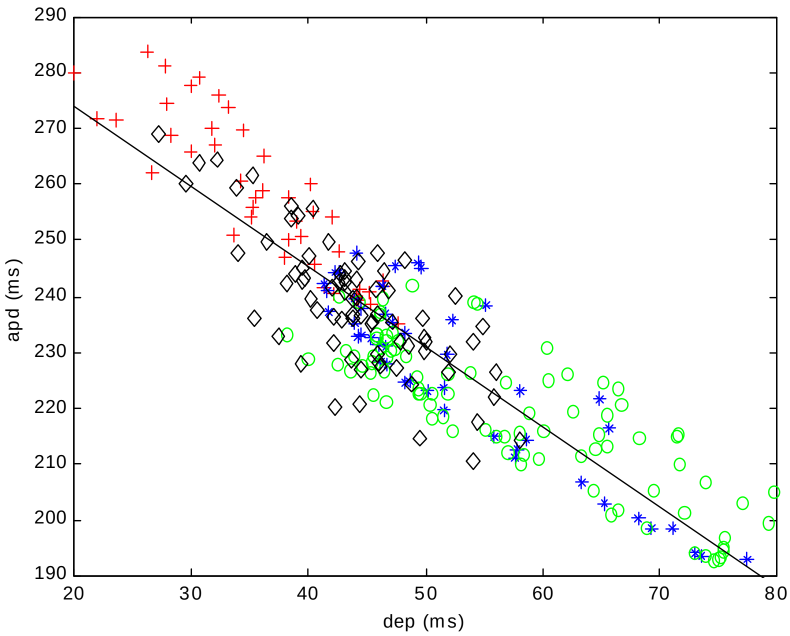


**Figure 3.** *Scattergram of the inversely computed APD values, $\alpha_{n,}$ at all nodes of $S_h$ as a function of the inversely computed depolarization times, $\delta_{n,}$ at these nodes. The + symbols relate to the nodes (LVC), the * symbols to nodes (RVC), the diamonds to nodes (epiLV); the circles relate to nodes (epiRV) (see text ).*

In an analysis based on this model it was shown that the peak of the T wave corresponds to the mean repolarization time over $S_h$. Moreover, the peak amplitude of the mean T wave is directly proportional to the dispersion of repolarization times. Its timing coincides with the mean repolarization time over $S_h$ (see [1]). In this analysis the range of depolarization times on $S_h$, the range of the $\delta_n$ values, is treated analogous to that of the repolarization `times', $\rho_n$ .

The current confusion related to the significance of QT dispersion stems from the fact that what one would like to estimate from recorded body surface potentials is the dispersion of the action potential durations on $S_h$ , the dispersion of the individual APD ($\alpha_n = \rho_n - \delta_n$) values, quantified by their range or by their standard deviation. However, meaningful estimate of this measure can only be obtained after solving, on an individual basis, the timing of depolarization, $\delta_n$ , and that of repolarization, $\rho_n$ , from observed body surface potentials. Both problems are so-called Inverse Problems. Although progress has been made toward solving these types of problems, unfortunately, the current quality of the inverse solutions does not provide an estimate of sufficient high quality to justify their introduction into a clinical application. The spreading out of the electric currents through the thorax prohibits the direct interpretation of the dispersion of QT intervals observed on the thorax, as representing the dispersion of APD values on the heart surface.

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