The Effects of Variations in Conductivity and Geometrical Parameters on the Electrocardiogram, Using an Eccentric Spheres Model

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SUMMARY The effects of variations in the volume conductor properties of the torso on the electrocardiogram were studied by means of a theoretical eccentric spheres model. The model includes a blood cavity, cardiac muscle layer, pericardium, lung region, skeletal muscle layer, and subcutaneous fat. The source of the field is a double-layer spherical cap located within the myocardium. The following effects regarding the electrocardiogram (ECG) potentials were determined: (1) blood augments the potential, but less than predicted by simpler published models; (2) in anemia, high potentials are expected, whereas in polycythemia, voltages are reduced; (3) abnormally low lung conductivity (emphysema) causes low surface potentials whose magnitude is controlled by the low conductivity skeletal muscle layer; (4) low voltages result both from low and high pericardial conductivities; (5) the surface potential increases with increasing myocardial conductivity; (6) low skeletal muscle conductivity (Pompe's disease) causes high surface potentials; (7) obesity lowers the potential only slightly; (8) a thick myocardium, protruding into the lung region, slightly augments the potential; (9) an increase in the thickness of the myocardium at the expense of the blood cavity causes a decrease in potential; (10) the potential increases with increasing heart size; and (11) the location of the heart within the torso has a very significant effect on the surface potential distribution.

THE electrical activity of cardiac muscle cells is manifest at the surface of the torso through the action of the intervening conducting medium. The surface potentials which are recorded as electrocardiograms (ECGs) reflect, therefore, both the heart generators and the properties of the surrounding volume conductor. To investigate the effects of the volume conductor on the surface potentials, we constructed a mathematical model which includes the conductivity and relative geometry of all important torso compartments, and yet is simple enough to permit analytical solutions (Rudy and Plonsey, 1979). The model consists of a spherical heart eccentrically located within a spherical torso, and includes the following discrete regions: intracavitary blood mass, myocardium, pericardium, lungs, skeletal muscle, and subcutaneous fat.

In contrast to prior theoretical models concerned with internal inhomogeneities, this model includes all important torso components, permitting a study of their integrated effects on the surface potential. An earlier attempt to do so (Bayley and Berry, 1966; Bayley et al., 1969), which was based on a comprehensive model similar to the one used here, was found in error (Rudy and Plonsey, 1979).

The objective of this paper is to determine the behavior of the surface potential magnitude and distribution resulting from variations in geometry and conductivity that reflect both normal and abnormal behavior. In some cases the variations simulate specific disease states. Specifically, we shall consider variations in: (1) conductivity of the intracavitary blood, (2) lung conductivity, (3) pericardial conductivity, (4) myocardial conductivity, (5) conductivity of the skeletal muscle layer, (6) fat layer thickness, (7) the location of the heart within the torso and of the activation source within the myocardium, (8) thickness of the ventricular wall, and (9) the size of the heart. Since the model includes a number of inhomogeneous torso compartments, the variations in geometry and/or conductivity of a single compartment are studied against some suitable choice of the remaining parameters. That is, the interactions of effects are considered.

Methods

The effects of the volume conductor properties on the ECG were studied by computer simulation based on a mathematical solution of a suitable volume conductor model (Rudy and Plonsey, 1979). The model used consists of two eccentric systems of concentric spheres (Fig. 1). In this model, the heart is represented as a sphere consisting of a central blood volume bounded by a spherical heart-muscle shell and pericardium; the heart, in turn is placed eccentrically within a spherical torso which includes a lung region bounded by spherical muscle
and fat layers. The source of the field is a double-layer spherical cap lying concentrically within the heart muscle which represents an activation wave. The direction of the double layer is radial, and since the spread of activation in the left ventricular wall is mainly from endocardium to epicardium, this is a realistic representation of the source during most of the QRS. The idealized spherical geometry is necessary for obtaining an analytic solution to the problem. Such a solution makes possible the inclusion of many inhomogeneous compartments in the model and the manipulation of geometrical parameters (such as the relative size of the heart or its location, the dimensions of any other compartment, etc.) without difficulty. In addition, the model permits alteration of the electrical conductivities of the various compartments present. The method of determining the analytic expression for the surface potentials on the spherical "torso" is described in a former paper (Rudy and Plonsey, 1979). It should be mentioned that, although the expression obtained for the surface potential is analytic, its functional dependence on the conductivities and geometry is complicated, and numerical computations must be made to investigate the effect of variations in each one of these parameters on the surface potential distribution. For simplicity, most of the potential values presented in this paper were calculated at a single point \((r_6, 0)\) (see Fig. 1), since the behavior of the potential at other anteriorly located sites is qualitatively the same.

The following values are used in the computations to represent typical geometrical and conductivity parameters (see Fig. 1 for identification of parameters):

**Conductivity Parameters** (Rudy and Plonsey, 1979; Rush et al., 1963)

- \(\sigma_1\) (blood) = 0.006 mho/cm, \(\sigma_2\) (myocardium) = 0.002 mho/cm, \(\sigma_3\) (lung) = 0.0065 mho/cm, \(\sigma_4\) (skeletal muscle) = 0.00125 mho/cm, \(\sigma_5\) (fat) = 0.0004 mho/cm, \(\rho_p\) (pericardial resistance) = 1000 ohm-cm².

**Geometrical Parameters** (Eycleshymer and Schoemaker, 1911)

- \(r_1\) (radius of blood cavity) = 4 cm, \(r_3\) (radius of spherical "heart") = 5 cm, \(r_5\) (external radius of lung region) = 11 cm, \(r_4\) (external radius of muscle region) = 12 cm, \(r_6\) (radius of spherical "torso") = 12.5 cm, \(d\) (eccentricity; distance of heart center from torso center) = 5 cm.

The value assigned to the conductivity of the myocardium is an average of the "high" and "low" conductivities of this slightly anisotropic tissue. The conductivity of the lung region is an average over a respiratory cycle. The highly anisotropic surface muscle layer is replaced by an equivalent layer of isotropic conductivity \(\sigma_4\), obtained by using a scale transformation (McFee and Rush, 1968). Under this transformation, the thickness of the muscle layer is modified, being multiplied by a factor of 3, so that the effective thickness used in the computations is 3 cm.

Unless otherwise stated, the central angle of the double-layer spherical cap was taken to be 60°. For this angle, the surface area of the source is sufficiently extensive to represent a "typical" activation wave. In most of the calculations, the double-layer radius was set equal to 4.5 cm. This radius corresponds to a double layer located midway between the endocardium and the epicardium. The strength of the double-layer source was taken to be unity. In the study of the effects of variations in an individual parameter, the remaining parameters were held constant at the typical values given above.

**Results**

**Variations in Blood Conductivity**

Figure 2 describes the surface potential at \((r_6, 0)\) (see Fig. 1) as a function of the intracavitary blood conductivity \(\sigma_i\). Since blood conductivity and hematocrit are related (Cole and Curtis, 1944; Hirsch et al. 1950), the latter is also included in the figure. "Normal" refers to the value of conductivity at normal hematocrit. Two different locations of the source are considered, namely at the endocardium and the epicardium. The condition \(\sigma_1 = 0.002\) mho/cm describes the case in which the blood region is homogeneous with the surrounding myocardium. We can conclude that the surface potential increases with increasing blood conductivity (decreasing hematocrit); the effect is more pronounced for an endocardial double-layer source.

The effect of the intracavitary blood mass can be appreciated by comparing the potential obtained for \(\sigma_1 = 0.006\) mho/cm (normal conductivity of blood), to that obtained under homogeneous conditions \((\sigma_1 = \sigma_2 = 0.002\) mho/cm). For a double-layer source located endocardially, the potential increases by 46.4% when the intracavitary blood is...
Variations in Lung Conductivity

The effect of variations in the conductivity of the lungs on the potential magnitude at \( (r_5, 0) \) is described in Figure 3. Three cases are considered: In A, the skeletal muscle and subcutaneous fat layers are made homogeneous with the underlying lung region (i.e., \( \sigma_5 = \sigma_4 = \sigma_3 = 0.0005 \text{ mho/cm} \)). Under these conditions, the surface potential increases as the lung conductivity decreases. In B, the surface muscle layer is assigned its typical conductivity value \( (\sigma = 0.00125 \text{ mho/cm}) \), as a result of which the functional dependence of the potential on the conductivity of the lungs changes completely, so that low voltages are obtained for abnormally low lung conductivities. The effect of adding the subcutaneous fat layer to the model defined by B is very small (C in Fig. 3), and the behavior of the potential is essentially the same as in B.

The modification in the behavior of the model brought about by inclusion of the surface muscle layer (B), in comparison to the behavior when this high conductivity layer is absent (A), demonstrates its dominant effect on the potential distribution. In particular, the effect of variations in lung conductivity on the surface potential cannot be evaluated without the inclusion of the surface muscle layer.

A clinical abnormality which produces abnormally low lung conductivity is obstructive lung disease (pulmonary emphysema, cystic fibrosis). In this condition, air—which is a nonconductor of electricity—is trapped in the lungs, and as a result the average lung conductivity decreases. According to our complete inhomogeneous model (curve C in Fig. 3), abnormally low surface potentials are to be expected. Low voltages measured in patients with obstructive lung disease are reported in the literature (Burch and DePasquale, 1963; Wasserburger et al., 1959; Selvester and Rubin, 1965; Littman,
Variations in Pericardial Conductivity

The dependence of the potential at \((r_0, 0)\) on the conductivity of the pericardium is shown in Figure 4. Two different cases are considered: in A, the pericardium is treated as an infinitely thin resistive membrane, whereas, in B, it is a layer of finite thickness (a thickness of 0.5 cm was chosen). The abscissa is a logarithmic scale of pericardial conductivity (normalized, log scale). The potentials obtained here demonstrate that decreased lung conductivity may indeed be a cause for the reduction in the surface potential, and that an important role is played by the muscle layer in determining this behavior. This last observation adds to our understanding of the process. The intuitive explanations which have been given in the literature consider the lungs to be the only cause for the low potentials detected in patients with obstructive lung disease. This conclusion is reached by arguing that the high resistance lungs impede current flow to the surface and, as a result, low voltages are obtained. In fact, were the surface muscle layer absent, an abnormally high ECG potential field would result from low values of lung conductivity (A in Fig. 3).

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Variations in Myocardial Conductivity

The behavior of the potential at \((r_\text{s}, 0)\) as a function of the myocardial conductivity is shown in Figure 5. The potential increases with increasing myocardial conductivity; for an increase in conductivity from 0.0005 mho/cm (homogeneous with the surrounding lung region) to 0.002 mho/cm (the typical value), the potential increases by 21.9%.

Variations in Skeletal Muscle Conductivity

The high conductivity surface muscle layer plays an important role in determining the behavior of the body surface potential distribution. As described above, the functional dependence of the surface potential on the conductivity of the lung is controlled by the effect of the skeletal muscle, and this surface layer is responsible for the abnormally low voltages obtained in cases of abnormally low lung conductivities (Fig. 3).

The high conductivity surface muscle layer attenuates the potentials at the surface of the torso. The decrease in the potential at \((r_\text{s}, 0)\) caused by the normal surface muscle in the presence of normal inhomogeneities is 22.56% (relative to the case in which the surface muscle layer is made homogeneous with the underlying lung region, i.e., \(\sigma_\text{M} = \sigma_\text{L} = 0.0005\) mho/cm). This layer also acts to reduce potential differences between points on the torso. The potential difference between \((r_\text{s}, 0)\) and \((r_\text{s}, 180)\) is reduced from 2.75 to 1.95 when the surface muscle is included. This “short-circuiting” effect explains the low voltages detected in cases of abnormally low lung conductivities, since under this condition most of the tangential current is confined to the high conductivity surface muscle layer.

The potential at \((r_\text{s}, 0)\), as a function of the skeletal muscle conductivity is shown in Figure 6. The potential decreases with increasing muscle conductivity, and a 5-fold increase in the conductivity (from 0.0005 mho/cm to 0.0025 mho/cm) causes the potential to drop from 2.05 to 1.22 (a 40.5% decrease).

A clinical condition which results in an abnormally low skeletal muscle conductivity is Pompe’s disease. According to the behavior described in Figure 6, abnormally high surface potentials are to be expected with this abnormality. The clinical findings conform to this prediction; R voltages of 66 mm in V5, 84 mm in V6, and 88 mm in V7 are observed (Potter, J. L., and Kramer, J. D., The Children’s Hospital Medical Center of Akron, Ohio, personal communication). Since a high degree of cardiomegaly is observed in patients with Pompe’s disease, and since the effect of a dilated heart, according to our model, is to augment the surface potentials (the effect of an increase in the size of the heart is discussed below), it is likely that the high ECG voltages result from a combination of two effects: a dilated heart and an abnormally low skeletal muscle conductivity.

Variations in the Fat Layer Thickness

The outermost compartment of our model, namely, the subcutaneous fat layer, does not influence the surface potential distribution significantly. Its inclusion in the model causes the potential at \((r_\text{s}, 0)\) to increase by only 6.8%, whereas the potentials at other locations on the torso are affected hardly at all.

An attempt to simulate the effect of obesity on the surface potential is shown in Figure 7. The potential at \((r_\text{s}, 0)\) decreases with increasing fat
thickess so that low potentials are expected in cases of obesity. The effect is not very significant, and an increase in fat thickness of 1 cm (from 0.5 to 1.5 cm) causes the potential to decrease by only 9.2%.

**Variations in the Location of the Heart, and of the Double-Layer Source**

A geometrical parameter which plays a very important role in determining the surface potential distribution is eccentricity (the distance from the "torso" center to the "heart" center). In the model, for an eccentricity of 1 cm, the anterior wall of the ventricle is 5 cm from the inner boundary of the anterior chest wall, and for an eccentricity of 5 cm, the distance between the heart and the anterior chest wall is 1 cm.

The potential at (r, 0) is shown in Figure 8 as a function of the eccentricity. When the eccentricity is increased from 1 to 5 cm, the potential is almost doubled (the increase in potential is 97%). When the eccentricity is increased by 1 cm, from 4 to 5 cm (this can represent a normal variation in the location of the heart), the potential increases by 24.2%.

The effect of variations in the double-layer source location on the surface potential at (r, 0) is described in Figure 9. The potential is plotted as a function of the distance of the double layer from the endocardium (a distance of 0.0 cm represents an endocardial location of the source, while 1.0 cm refers to a source located epicardially). The double layer is moved from the endocardium to the epicardium, while the eccentricity of the heart is kept constant at 5 cm. Two situations are described in the figure: variations in the location of a double layer having a constant angle of opening (θ₀ = CONST.), and variations in the location of a double layer with a constant area (AREA = CONST.). In both cases, the potential increases as the source is advanced toward the epicardium. In the area = const. case, the increase is caused by the decreased distance between the source and the surface point where the potential is calculated [i.e., the point (r, 0)]. Under the condition of a constant angle of opening, an additional factor which contributes to the augmentation of the surface potential is the increase in the area of the double layer for increasing distance from the endocardium. This is the reason for the enhanced augmentation in the case θ₀ = const. as compared to the area = const. case. The increase in potential caused by the change in source location from endocardium to epicardium is 24.8% in the θ₀ = const. case, whereas in the area = const. case, the potential increases by 16% only.

An interesting observation is that the operation of the "Brody effect" (Brody, 1956) is opposite to that of the proximity effect described above. The Brody effect augments endocardial sources more than it augments sources located epicardially. The results obtained here show that the proximity effect overwhelms the Brody effect even when the area of the double layer is kept constant. Therefore, the net result is an increase in the surface potential at (r, 0). This observation strengthens our conclusion that the Brody effect is not as significant when considered together with all other torso effects.

**Variations in the Thickness of the Ventricular Wall**

In an attempt to simulate hypertrophy, the myocardial thickness was varied and the effect of these variations on the surface potential investigated. Three situations are illustrated in Figure 10. A is the typical case, with normal thickness of the ventricular wall (a thickness of 1 cm). When the thickness of the wall is doubled at the expense of the lung region (this situation is described in B), the potential at (r, 0) increases by 13.6% (from 1.000 to 1.138) relative to the typical case. On the other hand, when the thickness of the wall is doubled at the expense of the blood chamber (C in the figure), the potential decreases by 12% (from 1.000 to 0.881). The potential values are normalized so that a potential of unity is obtained for the typical case. The
FIGURE 10 The effect of a thick ventricular wall on the surface potential. A defines the typical case. The increase in myocardial thickness described in B is compensated by a reduction in the lung region, and in C it is compensated by a reduction in the intracavitary blood compartment.

area of the double-layer source and its distance from the anterior chest wall were kept constant in the three cases described.

The increase of 13.6% described in B is small, and cannot account by itself for the high ECG voltages measured in cases of hypertrophy (in these cases, an increase of 100% in potential values, relative to the normal, is common). It seems, therefore, that factors other than those under consideration here are influenced by hypertrophy. The factors might include the strength of the source and/or the area it occupies.

Variations in the Size of the Heart

The size of the spherical heart was increased to simulate dilation and to investigate its effect on the surface potential. Three cases are considered (Fig. 11). Figure 11A describes the typical normal case; a heart of radius 5 cm located within a torso of radius 12.5 cm. In B, the radius of the heart is increased from 5 to 8 cm, while the angle of opening of the double-layer source is kept constant. The enlargement of the blood chamber is accompanied in this case by an increase in the area of the double-layer source. The enlargement of the blood chamber is accompanied in this case by an increase in the area of the double-layer source. The situation described in B suggests a possible effect of the dilation on the source, assuming normal action potentials in the distended myocardial fibers, and normal initial spread of the electrical activity (i.e., a normal Purkinje system). In C, while the heart radius was increased from 5 to 8 cm (as in B), the area of the double-layer source was kept constant (as in A). This simulates a situation in which the source is not affected by the dilation, and allows isolation of the effect of the increased blood chamber on the surface potential arising solely from its volume conductor properties. In all three situations, the minimal distance from the anterior chest wall to the double-layer source and to the anterior wall of the spherical "heart" are the same. The following potential values are obtained at \( r = 0 \): In B, the potential is 1.8232; an increase of 82.3% relative to the typical case described in A. In C, there is also an increase in the surface potential, but by 35.3% only; a potential value of 1.3535 is obtained. (The potential values are normalized so that a potential of unity is obtained for the typical case.)

Since in C the area of the source is equal to its area in A, whereas in B its area is 3.8 times the area it occupies in A, we can conclude that the main contribution to the augmentation of the potential in B comes from the enlarged source itself.

The results obtained here show that a dilated heart is expected to augment the magnitude of the surface potentials due to the increase in the size of the blood compartment per se (C), while a possible contribution may arise from modifications in the source itself (B).

The theoretical predictions of these simulations are opposite to the results of a clinical study by Ishikawa et al. (1971). In their study of patients with congestive heart failure, the spatial maximal magnitude of the QRS vector was found to decrease as a function of increased cardiothoracic ratio (estimated from chest x-rays). Since the volume-conductor properties of a large blood cavity result in the augmentation of the surface potentials (C), other factors must be operative in congestive heart failure to account for the net attenuation of ECG voltage. It is possible that the excitation pattern and the source configuration and/or strength are modified in this abnormality in such a way that low potentials result in spite of the large blood cavity. The effect of this alteration in the source on the surface potential is opposite to the effect of the change simulated in B. Another possibility is in-
creased lung conductivity due to edema caused by the congestive heart failure. According to the model simulation, the potential at \((r_0, 0)\) is reduced by 49.5% when the lung conductivity is set equal to that of blood. The effect of high lung conductivity is, therefore, to attenuate the surface potentials, and is probably the cause of the low voltages measured in patients with congestive heart failure.

**Discussion**

The model simulations described above provide insight into the effects of various torso inhomogeneities on potentials at the surface of the body. An advantage of a theoretical model (compared to animal experiments or human studies) is the ability to investigate the effects of variations in a single parameter on the potential distribution, while all the other parameters are well controlled and kept constant at some chosen value. For example, the study of the effect of dilation on the surface potential shows that an increase in the volume of the blood chamber alone is to augment the potentials at the surface. This finding allows us to conclude that the low potentials detected in cases of congestive heart failure cannot result from the dilated heart per se, and that other factors must be operative. Other such examples are the findings that the thickening of the ventricular wall cannot be the sole factor responsible for the high surface potentials detected in cases of hypertrophy, whereas, on the other hand, the low potentials measured in pericardial effusion can be explained on the basis of variations in a single parameter, namely, the pericardial conductivity.

Another important conclusion is that interactions between the various torso compartments play an important role in determining the surface potential distribution. This is demonstrated by the dominant effect of the surface-muscle layer on the behavior of the potential as a function of lung conductivity, and by the reduction in the Brody effect due to interactions of the blood cavity with other torso inhomogeneities. These observations show that combined models, rather than models which isolate portions of the torso volume conductor, should be utilized in the investigation of the effects of variations in conductivity and/or geometrical parameters on the surface potential.

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_Circ Res._ 1979;44:104-111
doi: 10.1161/01.RES.44.1.104

_Circulation Research_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7330. Online ISSN: 1524-4571

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