A Comparison of Volume Conductor and Source Geometry Effects on Body Surface and Epicardial Potentials

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SUMMARY Using an analytical mathematical model, we studied and contrasted the effects of variations in geometry and volume conductor properties of the torso on epicardial and body surface potentials. The model consists of a spherical heart (blood cavity bounded by a spherical muscle shell that includes a double layer source, and pericardium) eccentrically placed in a spherical torso (lung region bounded by muscle and fat layers). The effects of the following parameters on body surface and epicardial potentials were studied: (1) separation of the cardiac sources; (2) location of the heart within the torso; (3) combined effects of all torso inhomogeneities; (4) "internal" inhomogeneities (intracavitary blood, pericardium); (5) "external" inhomogeneities (lung region, skeletal muscle, subcutaneous fat); and (6) hypertrophy and dilation. It was determined that, although internal inhomogeneities affect both epicardial and surface potentials similarly, the effect of external inhomogeneities on body surface potentials is different from their effect on epicardial potentials. The effects of hypertrophy and dilation are seen to depend on specific details regarding alterations in size and shape of blood cavity, heart, and activation surface. The most important conclusion of the study is that epicardial potential maps accurately reflect the underlying source configuration, are free of the effects of body shape and size, and are affected significantly by only one extracardiac inhomogeneity—namely, the lung region. Such maps, therefore, can enhance our capability to interpret and diagnose electrophysiological events within the heart.


As a consequence of the electrical activity of the heart, electrical potentials appear throughout the volume conductor in which the heart is embedded. In addition to body surface potentials which constitute the data in electrocardiography, potential distributions over the epicardium are also of great interest since they may reflect events within the heart that are not distinctly reflected at the body surface. Studies conducted on closed (intact) animals in which epicardial and intramural electrodes have previously been implanted (Spach et al., 1969; Spach and Barr, 1975a, 1975b, 1976) demonstrate that epicardial potential maps contain a considerable amount of information about the underlying intramural electrophysiological events.

A very important property, arising from potential theory, is that, in principle, epicardial potentials can be recovered from body surface data (Martin and Pilkington, 1972; Martin et al., 1975). Based on this capability, there is hope that epicardial data may be available noninvasively through computations based on body surface potentials and body geometry. This inverse solution, in contrast with a solution which represents the activity of the heart in terms of dipoles, can be evaluated by a direct comparison with epicardial measurements, such as those obtained simultaneously with surface potentials in an experimental animal (Barr and Spach, 1978). The objective of this paper is to study, using a mathematical analytical model, various effects of volume conductor properties (inhomogeneities and geometry) and of source configuration on epicardial potentials and to contrast them with the effects of the same parameters on body surface distribution. The results help elucidate the particular advantages in the use of epicardial maps.

Methods

To investigate and contrast the effects of the volume conductor and source geometry on both epicardial and body surface potentials, we use a theoretical eccentric-spheres model (Rudy and Plonsey, 1979a, 1979b), which includes all important torso inhomogeneities, and yet is simple enough to permit analytical solutions. In the model (Fig. 1), 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 located eccentrically within a spherical torso, where the latter consists of a concentric lung region bounded by muscle and fat layers. The source of the field is chosen to be a double layer spherical cap which lies within the myocardium and represents an idealized activation wave. The direction of the double layer is radial, and since the spread of activation in the left ventricular wall is mainly from

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endocardium to epicardium, this is a realistic idealization of the source during most of the QRS. This model permits altering the conductivities of the intracavitary blood, heart-muscle, lungs, surface muscle layer, subcutaneous fat, and epicardium. The thickness of each of the layers present also can be varied. The model also allows adjustment of the location of the heart and its relative size in the torso. The idealized spherical geometry is necessary to obtain an analytic solution that includes the aforementioned complexities, and permits the easy manipulation of these parameters. The fact that the model includes all important torso compartments permits a study of their integrated effects on both body surface and epicardial potentials.

The method of determining the analytic expressions for the potential fields is described in a previous paper (Rudy and Plonsey, 1979a). We reproduce here the expression for epicardial ($\Phi_e$) and body surface ($\Phi_b$) potentials, which are given by:

$$\Phi_e(r, \theta) = 2\pi a^2 \left[ \sum_{l=0}^{n} F_l \left( \frac{1}{\ell + 1} \right) \left( \frac{r_0}{r_2} \right)^{\ell+1} P_{\ell}^r (\cos \theta) \right]$$

$$+ G_l \frac{1}{\ell} \left( \frac{r_0}{r_2} \right) \left( \frac{r_0}{r_2} \right)^{\ell+1} P_{\ell}^r (\cos \theta),$$

and

$$\Phi_b(r, \theta) =$$

$$2\pi a^2 \left[ \sum_{l=0}^{n} \left( \frac{d}{r_5} \right)^{\ell+1} \sum_{\ell=1}^{n} M_{\ell} \left( \frac{r_0}{r_2} \right)^{\ell+1} P_{\ell}^r (\cos \theta) \right]$$

$$+ \frac{1}{d^{\ell+1}} \cdot \frac{s!}{(s-\ell)!} \left( \frac{r_0}{r_2} \right)^{\ell+1} \sum_{\ell=1}^{n} N_{\ell} \frac{P_{\ell}^r (\cos \theta)}{\ell r_0^{\ell+1}}$$

$$+ \frac{1}{d^{\ell+1}} \cdot \frac{d'}{s! (s-\ell)!} \left( \frac{r_0}{r_2} \right)^{\ell+1} \sum_{\ell=1}^{n} N_{\ell} \frac{P_{\ell}^r (\cos \theta)}{\ell r_0^{\ell+1}}$$

$$\cdot \frac{d'}{s! (s-\ell)!} \left( \frac{r_0}{r_2} \right)^{\ell+1} \sum_{\ell=1}^{n} N_{\ell} \frac{P_{\ell}^r (\cos \theta)}{\ell r_0^{\ell+1}}$$

$$+ \frac{1}{d^{\ell+1}} \cdot \frac{d'}{s! (s-\ell)!} \left( \frac{r_0}{r_2} \right)^{\ell+1} \sum_{\ell=1}^{n} N_{\ell} \frac{P_{\ell}^r (\cos \theta)}{\ell r_0^{\ell+1}}$$

where $P_{\ell}$ is the Legendre polynomial of degree $\ell$, $P_{\ell}^r$ is its derivative, $r_0$ is the radius of the double layer spherical cap, $2\theta_0$ is the central angle of this double layer, $d$ is the eccentricity (distance of heart center from torso center), and $a = r_0 \sin \theta$. The radii of the heart and the torso are $r_2$ and $r_5$, respectively. The origin of $\theta$ is the center of the spherical heart in Equation 1 and the center of the spherical torso in Equation 2. The polar angle $\theta$ is measured from a line which connects the centers of the spheres. The origin of $\theta$ is the center of the spherical heart in Equation 1 and the center of the spherical torso in Equation 2. The coefficients, $F_l, G_l, M_l, N_l$, are determined by the boundary conditions at the interfaces between regions of different conductivity. The following values are used in the computations to represent typical geometrical and conductivity parameters (see Fig. 1 for identification of parameters):

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

$r_1$ (radius of blood cavity) = 4 cm; $r_2$ (radius of spherical heart) = 5 cm; $r_3$ (external radius of lung region) = 11 cm; $r_4$ (external radius of muscle layer) = 12 cm; $r_5$ (radius of spherical torso) = 12.5 cm; $d$ (eccentricity; distance of heart center from torso center) = 5 cm.

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

$\sigma_1$ (blood) = 0.006 mho/cm; $\sigma_2$ (myocardium) = 0.002 mho/cm; $\sigma_3$ (lung) = 0.0005 mho/cm; $\sigma_4$ (skeletal muscle) = 0.00125 mho/cm (corrected for anisotropic effects); $\sigma_5$ (subcutaneous fat) = 0.0004 mho/cm; $\rho_P$ (pericardial resistance) = 1,000 ohm-cm$^2$.

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 extensive enough to represent a "typical" activation wave. The double layer radius was set equal to 4.5 cm, which corresponds to a position midway between the endocardium and the epicardium. The strength of the double layer source was normalized to the value of unity. For simplicity, most of the potential values presented in this paper were computed at a single point on the torso, representing an anterior chest electrode, and at a corresponding point on the epicardium ($\theta = 0$ in Equations 1 and 2). The behavior of the potential at other anteriorly located sites is qualitatively the same. 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**

A description of the results of the model simulations and, in some cases, a discussion of their im-
The "Smoothing Effect" of the Volume Conductor

The body surface and epicardial potential distributions generated by two discrete activation wavefronts are shown in Figure 2 (A and B, respectively). The wavefronts are located in the anterior part of the spherical cardiac wall, and the potential distributions over the anterior half of the spherical torso and the epicardium are shown. The two activation waves have central angles of 10° and are separated by 40°, 80°, and 120° (Fig. 2, I, II, and III, respectively). For the simulation, when two activation fronts are separated by less than 100° (i.e., 40° and 80°, in the figure), two discrete maxima arise on the epicardium, whereas a single broad maximum appears on the body surface. For a separation greater than 100° (i.e., 120° in Fig. 2), two discrete maxima are apparent on the body surface as well. The result demonstrates that activation details seen at the epicardium tend to be smoothed out at the body surface. The fact that the single surface potential maximum shown in Fig. 2, I, originated from a multiple source configuration is reflected in its specific amplitude and shape (spatial extent of the maximum and low level "tails"), in contrast with the single maximum in II, which arises from a different multiple source configuration. Note that, even when two maxima appear on the surface (Fig. 2, III), their location does not correspond to the location of the wavefronts. In contrast, the location of the epicardial maxima accurately reflects the location of the underlying activation waves so that the epicardial potential map is an accurate reflection of the myocardial sources.

The minimum angle of separation (the "critical angle") of two cardiac activation fronts (of fixed extent) at which two discrete maxima appear on the body surface depends on the distance of the sources from the surface. Figure 2 was obtained for an eccentricity of 5 cm, that is, the minimum distance from a point on the epicardium to a point on the anterior chest wall is 2.5 cm. (This represents the typical anatomical geometry.) For this case, a critical angle of 100° was determined. When the distance between the heart and the anterior chest wall is increased by 4 cm (eccentricity of 1 cm only), a minimum separation of 140° is required between two sources located in the anterior ventricular wall in order for two discrete maxima to be detected at the surface. For the case of an eccentricity of 5 cm, but with activation wavefronts located in the posterior ventricular wall, we found a critical angle of 140°. This result demonstrates that, whereas the epicardial potential map can accurately mirror electrical events anywhere in the heart wall, body surface potential patterns are less sensitive to the details of activation in regions of the heart that are remote from the body surface—such as the posterior and diaphragmatic surfaces of the heart.

The smoothing effect of the volume conductor was observed experimentally by King et al. (1972) in the intact dog and by Spach et al. (1977, 1978) in the intact chimpanzee. Simultaneous recordings of body surface and epicardial potentials in the aforementioned studies, as well as a comparison of measured torso potentials with those simulated from epicardial recordings (Ramsey et al., 1977), show that (at many instants) the body surface potential maps are simpler and less detailed than the epicardial maps. It is noted in these studies that anterior surface potential maps are capable of resolving those epicardial extrema whose separation equals or exceeds their distance to the torso. When the

**Figure 2** Comparison of body surface (A) and epicardial (B) potentials originating from two discrete activation wave fronts located in the myocardium. The central angles of the two activation waves are 10°. The separation between the wave fronts is: I-40°, II-80°, III-120°. The geometry is illustrated by the cross section of the model in the left upper corner of each graph.
separation is less than the heart-torso distance, distinct details of epicardial events disappear but are reflected in the character of the low level potentials which occupy broad areas, all of which is in agreement with the theoretical results obtained here. Such conclusions also are supported by the work of Abildskov et al. (1976), who studied surface potential distributions arising from stimulated ectopic beats at different sites in the closed-chest dog. The smoothing effect was also demonstrated by Taccardi et al. (1951, 1958, 1972) in tank studies of the potential distribution surrounding isolated turtle and dog hearts, as well as in a study of the potential field generated experimentally by two dipoles in a circular homogeneous conducting medium (DeAmbroggi and Taccardi, 1970). An isolated, perfused rabbit heart technique was used by Mirvis et al. (1977) to assess the ability to detect and localize multiple discrete epicardial events from body surface potential distributions. They conclude that surface maps accurately depict single and dual generators only if the two sources are sufficiently separated. In view of the theoretical simulation and the experimental results described above, we can conclude that details of the electrical activity of the heart that are reflected in the epicardial potential distribution may be smoothed out by the volume conductor and hence not appear in the body surface potential map. Therefore, an inverse calculation which reconstructs the epicardial potential maps from the simpler torso maps in highly desirable since it should improve our ability to interpret the underlying events within the heart.

The Dependence of Potential on the Eccentricity of the Heart

The torso potential at an anteriorly located point \(r_{s}, 0\) and the potential at the corresponding point \(r_{e}, 0\) on the epicardium are shown in Figure 3 (A and B, respectively) as a function of the eccentricity (the displacement of the "heart" center from the "torso" center). Anatomical significance can be appreciated by noting that, 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, whereas for an eccentricity of 5 cm, the distance between the heart and the anterior chest wall is 1 cm. The behavior of the potential at other sites on the torso surface and on the epicardium is qualitatively the same as that plotted.

Figure 3A demonstrates that surface potentials are greatly affected by the heart position in contrast to the epicardial potential behavior (Fig. 3B). When the eccentricity is increased from 1 to 5 cm, the torso potential is almost doubled (the increase in potential is 97%). When the eccentricity is increased by 1 cm, from 4 to 5 cm (this could represent a normal variation in the location of the heart), the potential increases by 34.2%. The epicardial potentials (Fig. 3B) are almost completely independent of the location of the heart within the torso. For an increase of the eccentricity from 1 to 5 cm, the epicardial potential increases by only 3.8%. This result implies that the entire epicardial potential distribution is not sensitive to variations in the location of the heart, as might be caused by changes in posture, and are essentially free from effects of body shape and size. This result comes about because, as shown below, of all extracardiac sources, only the secondary sources at the heart-lung interface are normally of significance in determining epicardial potentials; but this effect is independent, of course, of the heart position in the torso.

Effects of Variations in Conductivity

Combined Effects of the Torso Compartments

The total effect of all inhomogeneities on both the epicardial and body surface potential is shown in Figure 4, I and II, respectively. In each, two cases are described and compared. "A" is the homogeneous case corresponding to a uniform conductivity which is everywhere equal to that of the myocardium (which itself is an average of the range of conductivities found within the thorax). Only the body-air interface is taken into account. "B" describes the complete inhomogeneous case, where all compartments of the model are present with their typical values of conductivity. Both torso and epicardial potentials are normalized so that the peak potential in the homogeneous case is unity. It is seen that the combined effect of the inhomogeneities is to augment both torso and epicardial potentials. The peak epicardial potential in the inhomogeneous case is 2.22 times its value for a homogeneous torso. The augmentation factor for the peak torso surface potential is smaller and has the value of 1.59. Note that the effect of the inhomogeneities depends on the location of the field point. Although the inhomogeneities affect the potentials at anterior points in the same qualitative way (i.e., the potentials are augmented by the inhomogeneities), the
potentials at posteriorly located points on the "torso" (Fig. 4B) are hardly affected at all.

Internal Inhomogeneities (Blood Cavity and Pericardium)

The epicardial potentials were computed at the boundary between the heart and the lung region, the heart being covered by a pericardium which is represented in the model as an infinitely thin resistive membrane. Therefore, the pericardium, as well as the blood cavity, is located internally to both the epicardial surface and the torso surface (i.e., the surfaces on which potentials are examined in this paper).

The body surface and epicardial potential at corresponding anterior points ($\theta = 0^\circ$), as a function of the intracavitary blood conductivity, is shown in Figure 5 (A and B, respectively). When the conductivity of the blood region equals 0.002 mho/cm, it is equal in value to the surrounding myocardium. The typical value of blood conductivity is 0.006 mho/cm. Both epicardial and torso potentials are normalized so that a value of 1.0 is attained for a blood conductivity equal to that of the myocardium (0.002 mho/cm). It is seen that the effects of variations in blood conductivity on both epicardial and surface potentials are very similar; both curves A and B increase monotonically with increasing conductivity. The effect of the intracavitary blood mass can be appreciated by comparing the potentials obtained for a typical conductivity (0.006 mho/cm) to the values obtained under "homogeneous conditions" (conductivity of 0.002 mho/cm). When the intracavitary blood is at its normal conductivity, the epicardial potential increases by 27.5%, while an increase of 26.1% is computed at the torso surface. As pointed out in a previous paper (Rudy and Plonsey, 1979b), the augmentation effect of the

![Figure 4](https://example.com/figure4.png)

**Figure 4** The combined effect of torso inhomogeneities on epicardial (I) and torso surface (II) potentials. Both epicardial and surface potentials are normalized so that the peak potential in the homogeneous case is unity.

![Figure 5](https://example.com/figure5.png)

**Figure 5** The effect of variations in intracavitary blood conductivity on torso surface (A) and epicardial (B) potentials. The potentials are adjusted to attain a value of unity for a conductivity of 0.002 mho/cm (that is, a value equal to the typical myocardial conductivity). The typical conductivity of blood is 0.006 mho/cm. The range of conductivities (0.002 to 0.01 mho/cm) corresponds to hematocrit range of 78% to 20%, respectively.
blood would be even greater were it not diminished by interactions with the other torso inhomogeneities.

Figure 6 describes the dependence of body surface (A) and epicardial (B) potentials on pericardial conductivity. The pericardium is represented in the model by an infinitely thin resistive membrane. The pericardial conductivity is shown on a logarithmic scale normalized to the typical value of 0.001 mho/cm² (marked NORMAL in the figure). The potentials are adjusted so that a value of unity is obtained for the typical conductivity. Again, as in the case of variations in blood conductivity, epicardial and torso potentials are affected similarly by variations in pericardial conductivity; they both increase monotonically when pericardial conductivity is increased. A 10-fold increase in the conductivity from the typical value results in an increase of 13% in the epicardial potential and 15% in the body surface potential. We can conclude, therefore, that the internal inhomogeneities (blood cavity and pericardium) affect both epicardial and body surface potentials in a similar way.

External Inhomogeneities (Lungs, Surface Muscle Layer, Subcutaneous Fat)

By “external inhomogeneities,” we refer to the thorax compartments that are external to the spherical surface over which the epicardial potentials are computed. These compartments constitute the volume conductor in which the heart is embedded and include the lung, surface muscle, and subcutaneous fat.

The effects of variations in lung conductivity on torso (A) and epicardial (B) potentials are compared in Figure 7. In contrast to the internal inhomogeneities, the effect of the lung inhomogeneity on body surface potentials is different from its effect on epicardial potentials. Whereas epicardial potentials decrease monotonically with increasing lung conductivity, torso potentials attain a maximum at a conductivity that is very close to typical physiological values. In Figure 7, the conductivity is normalized to 1.0 for the typical value marked NORMAL. (The relative potentials are also adjusted to unity at this conductivity.) It was shown in a previous paper (Rudy and Plonsey, 1979b) that the functional dependence of the surface potential on lung conductivity is dominated by the high conductivity surface muscle layer. In the absence of the muscle layer, the surface potential increases as the lung conductivity decreases. The maximum shown in Figure 7 is obtained only in the presence of the surface muscle layer. In contrast, the functional dependence of epicardial potentials on lung conductivity is independent of the presence of the surface muscle layer in the model, and the behavior obtained when the muscle inhomogeneity is absent is very similar to that shown in Figure 7B (in Fig. 7, all inhomogeneities, including the surface muscle layer, are taken into account). By comparing the potentials obtained for typical lung conductivity to the values obtained when the conductivity is set equal to that of the myocardium (the corresponding normalized values are 1.0 and 4.0, in Fig. 7), we see that the lung inhomogeneity augments the epicardial potentials by 85%, whereas the augmentation factor for surface potentials is only 19%. Consequently, the lung inhomogeneity is very important in determining the epicardial potential distribution but has a much smaller, though significant, effect on the body surface distribution.

Body surface and epicardial potentials are shown in Figure 8, A and B, respectively, as a function of skeletal-muscle conductivity. As described above, this high conductivity layer plays a very important role in determining the torso surface potential distribution; in particular, it controls the functional
dependence of the surface potential on the conductivity of the lungs. The muscle layer attenuates surface potentials; the decrease in potential caused by this layer is 22.5% (relative to the case in which the surface muscle layer has the same conductivity as the underlying lung region). As can be seen from Figure 8A, the surface potential decreases with increasing muscle conductivity, and a 5-fold increase in the conductivity (from 0.0005 to 0.0025 mho/cm) causes the potential to decrease by 40.5%. In contrast, the epicardial potential distribution is not strongly affected by the presence of the surface muscle layer. From Figure 8B, it can be verified that a 5-fold increase in muscle conductivity (again from 0.0005 to 0.0025 mho/cm) causes a slight increase of only 8.0% in epicardial potential. The effects of the outermost torso inhomogeneity, the subcutaneous fat, on torso and epicardial potentials are shown in Figure 9, A and B, respectively. The effect of this layer on torso potentials is small; a 10-fold increase in fat conductivity (from 0.00025 to 0.0025 mho/cm) causes the potential to decrease by only 15%. The epicardial potentials are even less affected by variations in fat conductivity and, as demonstrated by Figure 9B, the effect of the subcutaneous fat layer on these potentials is completely negligible.

In view of the results presented above, it can be concluded that the augmentation of potentials at anteriorly located points in the presence of all the inhomogeneities (Fig. 4, A and B) is caused mainly by the high conductivity intracavitary blood and by the low conductivity lung region.

Geometrical Effects

Thickness of the Ventricular Wall

The effect of hypertrophy is considered in this model by increasing the thickness of the ventricular wall, keeping all other properties of the ventricular wall constant, including the radius and area of the double layer source. This change is simulated in Figures 10 and 11. In Figure 10, the thickness of the myocardium is doubled from the typical value of 1 cm to 2 cm. The thickening is at the expense of the intracavitary blood volume. The effect on both epicardial and surface potentials (at $\theta = 0^\circ$), is seen to be very similar; both potentials decrease with increasing myocardial thickness and are reduced by about 12% when the thickness of the wall is doubled. The decrease in potential is caused by the diminished blood volume and the increase in distance from the source to the blood-myocardium interface, as a result of which the augmentation effect of the blood is reduced. In Figure 11, the thickening of the ventricular wall is at the expense of the lung region. The torso potential at $\theta = 0^\circ$ is seen to increase by 13.6% when the thickness of the myocardium is doubled, an effect caused by the replacement of lung region by the higher conductivity myocardium. The epicardial potential, on the other hand, decreases with increasing wall thickness (Fig. 11B) and is reduced by 18.1% when the myocardium is doubled. Since in this simulation the location of the double layer source relative to the blood-myocardium interface was kept constant, an important
factor which contributes to the attenuation is the increase in distance from the source to the point on the epicardium at which the potential is computed.

Variations in the Size of the Heart

The effects of dilation on epicardial and surface potentials were investigated by simulating two possible changes in the heart. In Figure 12, the size of the blood cavity was increased, keeping the area of the double layer source constant. This simulates a situation in which the source is not affected by the dilation and allows an examination of the effects caused by the changes in the volume conductor alone. Under these conditions, the effects of the increased blood chamber on both anterior torso (Fig. 12A) and epicardial (Fig. 12B) potentials are very similar—both potentials increase with increasing heart size. For an increase of 3 cm (from 5 to 8 cm) in the radius of the heart, the surface potential is increased by 35.3%; a comparable increase of 28.9% is calculated at the epicardium. A different situation is described in Figure 13; here, the radius of the heart is increased while the central angle of the double layer source is kept constant. In this case, the enlargement of the heart is accompanied by a proportionate increase in the area occupied by the activation wave front. This simulates a possible effect of dilation on the source, assuming normal action potentials in the distended myocardial fibers and a normal Purkinje system. The effect of this change is seen to be much stronger on surface potentials (Fig. 13A) than on epicardial potentials (Fig. 13B). Although both potentials increase with increasing heart size, the surface potential is augmented by 82.3% when the radius of the heart is increased from 5 to 8 cm, as compared to an increase of only 12.8% in the potential at the epicardium.

The above simulations show the difficulty of predicting the effect of hypertrophy (and dilation) on the potential distributions without considerable information on both geometrical effects, as well as possible modifications of the activation sources. Such details are, at present, poorly understood.
great biophysical and clinical significance, since it effects of the lung inhomogeneity) is therefore of the above simulations, should take into account the above, a transformation which reconstructs intracardiac events can be detected, as well as to normalize the potential maps (to free them from the effect of body shape, so that they depend on cardiac sources alone). The development of suitable inverse transformations (which, according to the above simulations, should take into account the effects of the lung inhomogeneity) is therefore of great biophysical and clinical significance, since it makes possible a direct interpretation of electrical events within the heart in a fashion that is not possible from surface distributions.

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Discussion
The results of the model simulations described above call attention to two very important properties of epicardial potential distributions. (1) Epicardial potential maps mirror details of the electrical sources within the myocardium that may not be reflected in body surface distributions and allow more detailed examination of regional electrical events within the heart. (2) Epicardial potentials (in contrast to surface potentials) are almost completely independent of the location of the heart within the torso. This implies that epicardial maps are not sensitive to variations in the location of the heart, resulting from changes in posture of the subject, and are almost free from effects of body shape and size. In addition, the effects of the surface muscle layer and subcutaneous fat on epicardial potentials were shown to be minimal so that, of all extracardiac sources, only the secondary sources at the heart-lung interface affect significantly the potential distribution at the epicardium. In view of the above, a transformation which reconstructs epicardial potential maps from body surface potential maps serves to enhance the resolution with which intracardiac events can be detected, as well as to normalize the potential maps (to free them from the effect of body shape, so that they depend on cardiac sources alone). The development of suitable inverse transformations (which, according to the above simulations, should take into account the effects of the lung inhomogeneity) is therefore of great biophysical and clinical significance, since it makes possible a direct interpretation of electrical

FIGURE 13 The effect of an increased heart size on surface (A) and epicardial (B) potentials. The central angle of the double layer source is kept constant.
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