A Simulation Study of the Effects of Torso Inhomogeneities on Electrocardiographic Potentials, using Realistic Heart and Torso Models

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SUMMARY. The effects of torso inhomogeneities on electrocardiographic potentials were investigated via computer simulation, using a 23-dipole heart model placed within a realistically shaped human torso model. The transfer coefficients relating the individual dipoles to the torso surface potentials, as well as the body surface potential maps, the vectorcardiogram, and the 12-lead electrocardiogram resulting due to normal activation of the heart model, were calculated for each of the following torso conditions: homogeneous, homogeneous + skeletal muscle layer, homogeneous + muscle layer + lungs, and homogeneous + muscle layer + lungs + intraventricular blood masses. The effects of each inhomogeneity were deduced by comparing results before and after its inclusion. For individual dipole transfer coefficients we confirm the validity of the "Brody effect," whereby the high conductivity blood masses augment radially oriented dipoles and diminish tangentially oriented ones. With regard to the vectorcardiogram, the electrocardiogram, and the body surface potential maps, the major qualitative effects were an augmentation of the head-to-foot component of the vectorcardiogram due to the lungs, and a smoothening of notches in the electrocardiogram (temporal filtering) and of isopotential contours in the body surface potential maps (spatial filtering) with a consequent loss of information, due to the blood masses, muscle layer, and, to a lesser extent, the lungs. Besides the above qualitative effects of the inhomogeneities, there were also large quantitative effects on the surface potentials, namely, magnitude increases due to the blood masses and magnitude decreases due to the muscle layer, that—if unaccounted for—could compromise the inverse solution of these potentials for the cardiac dipole sources. (Circ Res 52: 45-56, 1983)

MOST STUDIES of the effects of torso inhomogeneities on electrocardiographic potentials may be classed into one of three categories (1) theoretical, using simplified spherical and planar geometries for the heart, lungs, and thorax (Brody, 1956; McFee and Rush, 1967, 1968; Rudy and Plonsey, 1979, 1980; Rudy et al., 1979); (2) experimental, using a current dipole to represent the heart and electrolytic tank models to represent the thorax and its inhomogeneities (Burger and van Milaan, 1946, 1947; Nelson et al., 1961; Rush, 1971, 1975); and (3) experimental, using the in vivo animal heart and altering the conductivity of the thoracic inhomogeneities, usually the intracardiac blood (Nelson et al., 1972). An excellent review of most of these studies and the domain in general is to be found in Rush and Nelson (1976). The most unequivocal conclusion to result from the above body of work has been the "Brody effect." Here, theoretical studies have shown that heart dipoles oriented perpendicular to the assumed spherical intraventricular blood mass surface are effectively enhanced, resulting in larger surface potentials. On the other hand, those dipoles tangential to the high-conductivity blood mass surface are effectively diminished, resulting in lowered surface potentials.

A more precise theoretical study employing electrophysiologically and anatomically accurate heart and torso models has proved difficult on account of the computational effort involved. One noteworthy exception was the work done by Horacek (1971). His results were analyzed in terms of the changes in the so-called Burger "image-surfaces," due to the successive introduction of realistically shaped lung and intracardiac blood mass inhomogeneities in an accurate numerical torso model of an adult male. The image surface is a plot of the lead vector \( \mathbf{L} \) relating the potential \( \phi \) at a given point on the torso surface to a fixed-location current dipole source \( \mathbf{p} \) representing the heart's electrical activity. The relationship is expressed mathematically by the scalar product \( \phi = \mathbf{L} \cdot \mathbf{p} \). The three cartesian components of the lead vector \( \mathbf{L} \) are thus equivalent to the three transfer coefficients relating the corresponding components of \( \mathbf{p} \) to the surface potential at the point under consideration. As the point on the torso surface is moved, both the potential and the lead vector naturally change and the tip of the latter traces out the image surface. In addition to being calculated for different torso conditions (e.g., homogeneous, with lungs, and with lungs and blood masses), the image surface has to be recomputed each time the source dipole location changes. Horacek computed these image surfaces for 60 different source dipole locations representing the septum and left and right ventricular "free-walls."
His conclusions were that, in general, the Brody effect is valid, that the low-conductivity lungs can contribute to channeling effects resulting in larger potentials due to dipoles oriented tangential to the lung surfaces, and that often the blood masses tended to counteract the effect of the lungs. Horacek cautioned, however, that all of the above phenomena are directional, i.e., seen at certain surface points on the torso and not at others, and that because of this and the irregular geometries used, “no simple universal statement can be made to describe the influence of inhomogeneities.” One drawback of his theoretical study which he noted was the lack of validation of his computations by a comparison of calculated body surface potential maps, using simulated wavefronts to represent normal cardiac excitation, with those actually observed in man. In other words, his 60-dipole sources were not activated in concert to simulate normal excitation, but rather, the effects of the inhomogeneities were estimated on each of them individually. Thus, while the effects on individual dipole transfer coefficients or lead vectors were estimated via the changes in the image surface, the effects of the inhomogeneities on the electrocardiogram (ECG), the vectorcardiogram (VCG) and the body surface potential map (BSPM) could not be determined. This was undoubtedly due to the nonavailability of an appropriate 60-dipole heart activation model at the time his study was performed.

Recently, Miller and Geselowitz (1978) described a heart model that starts by representing the geometry of the ventricles by a three-dimensional array of approximately 4000 points, to each of which is assigned an intracellular cardiac action potential. Selection of the onset time of the action potentials and their duration is done to correspond to the known sequence of ventricular activation and action potential durations in the ventricles, respectively. The intracellular action potentials then are used to calculate the strengths and orientations, during the entire cardiac cycle, of 23 fixed-location current dipoles, each representing a heart region. This heart model, when positioned inside a homogeneous numerical torso model, generated physiologically realistic ECGs and BSPMs (Miller and Geselowitz, 1978). The 23-dipole Miller-Geselowitz heart model is thus sufficiently realistic, electrophysiologically and anatomically, and at the same time sufficiently simple, to contemplate using, in conjunction with an accurate inhomogeneous torso model, for studying the effect of torso inhomogeneities not only on individual dipole transfer coefficients, but also on the ECG, VCG, and BSPM. Thus, not only would the numerical computations associated with the inhomogeneous torso be validated and Horacek’s conclusions verified, but also the degree to which clinically important electrocardiographic potentials are affected by the inhomogeneities would be estimated.

**Methods**

The approach used here to study the effects of torso inhomogeneities is straightforward. The Miller-Geselowitz heart model is properly positioned and oriented within a finite homogeneous torso model, and the successive inhomogeneities introduced into the torso model one at a time. Transfer coefficients relating torso potentials to the individual dipoles of the heart model, as well as ECGs, VCGs, and BSPMs generated by the heart model, are compared for the different torso conditions.

**Torso Model**

The torso model used was the same one developed by Horacek (1971, 1974). As mentioned earlier, it already incorporated lungs and intraventricular blood masses of conductivity relative to the torso of 0.25 and 3.0, respectively. We, however, had to alter the geometry of the blood masses to correspond to the geometry of the ventricular cavities in the Miller-Geselowitz heart model. The heart model was positioned and oriented within the torso to enter within the left lung cavity built by Horacek (Fig. 1). Note that the right ventricle has been placed anterior to the left ventricle. It was verified that—with this heart position and orientation—the simulated ECGs and VCGs compared with one of the normal ECG and VCG patterns, as reported in an atlas of ECGs and VCGs (Reddy and Gould, 1977). The simulated BSPMs were also in good agreement with those recorded in humans (Taccardi et al., 1976).

A third inhomogeneity, besides lungs and blood masses, was added to the Horacek torso. This was the anisotropic-conductivity skeletal muscle layer covering the thorax. It was included in an approximate manner, as suggested by McFee and Rush (1968). The skeletal muscle layer runs parallel to the torso surface and immediately under the skin and subcutaneous fat tissue. The muscle fibers within the layer may be assumed to lie in sheets parallel to the surface with a preferred, but varying, orientation from sheet to sheet. Muscle conductivity is higher along fibers (σ_high) than across them (σ_low). If one further assumes the fiber directions to be distributed uniformly over all directions parallel to the surface, then, even though each of the sheets has a preferred direction of current flow, the entire muscle layer may be characterized by a common conductivity, \( \sigma_{mean} = \frac{1}{2} (\sigma_{high} + \sigma_{low}) \), in all directions tangential to the surface (Rush, 1967). The conductivity normal to the torso surface remains at \( \sigma_{low} \). Further simplification of this homogeneous but still anisotropic layer is possible by increasing its dimensions normal to the surface by the factor \( \frac{1}{2} (\sigma_{high}/\sigma_{low}) \), but replacing its conductivities tangential and normal to the surface by the isotropic conductivity \( (\sigma_{mean} \cdot \sigma_{low})^{1/2} \). For radial currents, the increased distance to the surface is balanced by the increased conductivity; for tangential currents, the increased flow cross-section is balanced by the diminished conductivity. Thus the geometrical distortion does not affect body currents. As pointed out by McFee and Rush (1968), from an electrocardiographic standpoint the muscle layer effectively moves the body surface away from the heart sources. The numerical factors may be calculated using the values of \( \sigma_{high} \) and \( \sigma_{low} \) quoted by Rush and Nelson (1976). The factor of enlargement is approximately 3, the isotropic conductivity 0.00125 mho/cm. A muscle layer assumed to be uniformly 1 cm thick all around the torso is thus increased to 3 cm, its isotropic conductivity 0.035 relative to the torso, which is assumed to have an absolute conductivity of 0.002 mho/cm.

As a result of the muscle layer enlargement, the dimensions of the torso in the radial direction were increased by 2 cm. This increase affects X and Z coordinates only, and is indicated by the outer dark outline in Figure 1A. Next, the additional torso-muscle conductivity interface (inner dark
Figure 1. Three views of the torso model depicting lungs and intraventricular blood masses. The level of the 5th intercostal space is indicated by the arrows in A. The positions of septal dipole 4, right ventricular dipole 10, and left ventricular dipole 14 are indicated by the small filled circles. For better visualization of the blood masses, the left lung is not shown in B, and neither lung shown in C. For additional details see text.

Computational Details

Individual dipole transfer coefficients are best examined by successively placing Z, X, Y-oriented unit dipoles (for coordinate axes, see Fig. 1) at each of the 23 dipole locations of the Miller-Geselowitz heart model and plotting the potential distribution on the torso surface for each of the following torso conditions: (1) homogeneous, (2) homogeneous + muscle layer, (3) homogeneous + muscle layer + lungs, (4) homogeneous + muscle layer + lungs + blood masses. The effects of the muscle layer, lungs, and blood masses can then be successively deduced by comparing the appropriate torso surface potential distribution with the preceding case, e.g., the effect of the blood masses may be deduced by comparing the potential distributions corresponding to torso conditions (4) and (3). This more direct approach of comparing surface potential distributions lends itself to easier comprehension of the changes due to the inhomogeneities than visualization of a three-dimensional image surface.

Torso surface potential distributions were computed via the techniques of Barnard et al. (1967) and Lynn and Timlake (1968). In brief, the integral equation for the surface potential is discretized, resulting in a system of 3022 equations with 3022 unknowns. These are solved by the Jacobi iteration approach, using the infinite medium potential as an initial approximation. Prior to the solution, it is necessary to “deflate” the $3022 \times 3022$ coefficient matrix so as to speed convergence (Lynn and Timlake, 1968). To solve for...

* The computed transfer coefficients may be obtained on magnetic tape by writing to the authors.
the potentials due to each triad of unit dipoles took approximately 30 minutes of CPU time on the CYBER 173. Also, because with the CYBER 173 we were able to store our deflated coefficient matrix on disk memory as opposed to magnetic tape, the total computer time (CPU plus input-output) per triad was only 2 hours.

The accuracy of the discretized integral equation approach was checked against the known analytical solution for an eccentric current dipole in a homogeneous sphere (Frank, 1952). The surface of a sphere of radius 15 cm and conductivity 0.002 mho/cm was divided into 528 triangles, and the surface potential distributions due to separate radially and tangentially oriented unit current dipoles situated 6.75 cm off-center computed with the integral equation approach. These distributions were almost identical to those calculated using the analytical solution, with coefficients of correlation in excess of 0.99. An even more stringent test involved placing the unit dipoles between two concentric spheres of radii 4.5 and 15 cm, with the conductivity of the inner sphere 0.006 mho/cm and that of the intervening space 0.002 mho/cm. The outer and inner sphere dimensions were selected to be of the same order as those of the torso model and the blood masses, respectively. The dipoles were placed at radii ranging from 4.65 to 6.75 cm, approximately the same distances from the inner sphere as the Miller-Geselowitz heart model dipoles are from the blood masses. Correlation coefficients between the surface potential distributions on the outer sphere, as computed from the discretized integral equation and the analytical solution (Geselowitz and Ishiwatari, 1966; Arthur and Geselowitz, 1970), were once again greater than 0.99 for all dipole positions. The largest absolute difference between computed and analytic potentials on any surface triangle was 0.173 mV, and, not surprisingly, occurred where the absolute value of the surface potentials in all our test simulations was the largest. This was at the site of the potential maximum due to a radially oriented unit dipole at a radius of 4.65 cm (just 1.5 mm outside the inner sphere), where the computed and analytic potentials were 1.665 and 1.492 mV, respectively. This represents a percentage error of 11.62%. However, larger percentage errors than this were encountered in regions of low-level potentials, where a smaller absolute error is larger, percentage-wise. The percent error in potential for a given triangle is therefore not a very satisfactory index with which to gauge the accuracy of the surface distributions, and we accordingly relied on the correlation coefficient, together with the magnitude of the largest absolute difference.

Another relevant point that merits discussion here is the effect of closely spaced conductivity interfaces on the accuracy of the torso surface potential computations, e.g., at one point the left and right ventricular blood masses were only 3.82 mm apart. This was the closest distance between area elements belonging to different interfaces. As long as the different interfaces are closed surfaces that do not touch, computation of the surface potential integrals via the approach of Barnard et al. (1967) remains valid. Even with area elements 3.82 mm apart, the algorithms of Barnard et al. used to compute the deflated coefficient matrix presented no obvious problems. Since the coefficient matrix depends on the geometry of the conductivity interfaces, this geometry taken by itself posed no difficulty. However, of necessity, a few of the 23-dipole sources are very close to one or the other of the blood masses, e.g., the closest distance between a dipole and a blood mass area element was 1.94 mm. In this situation, the potential gradient near the blood masses is large, leading to large changes in potential between adjacent triangles of the blood mass interface. In other words the triangles used to construct the blood mass interface, being of the order of 1 cm to a side, are too large for accurate determination of the potential distribution at the blood mass interface. This lack of fineness of the triangulation is less evident the more distant the conductivity interface from the dipole source, with no difficulty in smooth reconstruction of the potential distribution being experienced for either the skeletal muscle interface or the outer torso surface. Thus, the computed potential distributions increase in accuracy and resolution as one passes from the blood mass interfaces, to the lungs, the muscle interface, and, finally, the torso surface. The most straightforward way to show that potentials at the torso surface are accurate, and not affected by the relatively coarse triangulation used for the blood masses, is to recompute these potentials with an increased number of triangles representing the blood masses. This was done by us, by subdividing each blood mass area element into four, thereby quadrupling the number of triangles used without altering the blood mass geometry. The torso surface potential distributions due to a triad of unit dipoles at each of the four dipole locations closest to the blood masses were recomputed, and found to be almost identical to those computed with the coarser triangulation scheme for the blood mass interfaces. The latter scheme was thus retained. A similar quadrupling of area elements used for the lungs and re-computation for each of the four dipole locations closest to the lungs also resulted in essentially unchanged torso potential distributions. Again, the coarser lung geometry was retained to keep computer time and memory requirements to a minimum.

Once the individual dipole transfer coefficients are computed, the ECGs, VCGs, and BSPMs for all four torso conditions are easily obtained by simple matrix multiplication of these transfer coefficients by the corresponding dipole strengths as determined from the Miller-Geselowitz heart model. The effect of each inhomogeneity is deduced as before.

Results

Inhomogeneity Effects on Individual Dipole Transfer Coefficients

Although transfer coefficients were calculated for all 23 dipole locations, results for only three locations, one each in the septum (dipole 4), right ventricle (dipole 10), and left ventricle (dipole 14) are shown here (Figs. 2, 3, and 4, respectively). These are sufficient to illustrate the more important inhomogeneity effects. The numbering scheme used for the dipoles is identical to that employed by Miller (1977); their positions are indicated in Figure 1. In what follows, we have tried to be somewhat conservative in our conclusions, mentioning only those effects common to several dipole locations. This recognizes the fact that the realistic nonregular geometry used for the torso, lungs, and blood masses prevents conclusions of sweeping generality on the basis of results due to one or two dipoles. On the plus side, however, it is likely that our conclusions mirror reality more closely than those based on simplified heart, lung, and torso geometries.

As mentioned earlier, the transfer coefficients are presented as body surface potential distributions due to
to a triad of unit current dipoles (of strength 1 mA-cm) placed at the source location under consideration and oriented in the Z, X, and Y directions, respectively. Thus, Figure 2 shows the potential distributions for unit dipoles Z, X, Y at dipole location 4 in the upper septum. The rectangle corresponds to the unrolled thorax after slicing down the right side of the chest. Accordingly, the left half of the rectangle represents the anterior chest, the vertical mid-line represents the left side, and the right half of the rectangle represents the back. Potential values are given in microvolts, assuming the absolute conductivity values mentioned in the Methods section. An identical format is used for Figures 3 and 4. It is evident from Figures 2, 3, and 4 that—although torso inhomogeneities have little qualitative effects on the potential distribution—they do cause important quantitative changes in the potential values. The effects of each of the introduced inhomogeneities is considered, in turn, below, with reference to Figures 2, 3, and 4.

The Muscle Layer

The most evident changes due to the muscle layer, obvious on comparing columns (1) and (2) in Figures 2, 3, and 4, are lowered potentials everywhere and increased spatial separation of the positive and negative extrema. The latter change is of course not observed in the planar maps for Z-oriented dipoles where the minimum is on the anterior chest and the maximum on the back. The lowered potentials and increased spatial separation of the extrema are consistent with the notion that the muscle layer effectively displaces the body surface outward. The absolute values of the anterior chest potentials are largest for dipole 10 in the right ventricle (Fig. 3), intermediate for dipole 4 in the septum (Fig. 2), and smallest for dipole 14 in the left ventricle (Fig. 4). This simply reflects the relative proximities of the three dipoles to the thorax surface. The difference in potentials due to the three locations is largest in the homogeneous case, the addition of the muscle layer serving to reduce the disparity by reducing anterior torso potentials for dipole 10 by approximately 50% compared to approximately 25% for septal dipole 4, and approximately 20% for left ventricular dipole 14.

The Lungs

The lungs have very little effect, qualitative or quantitative, on the septal (Fig. 2) or right ventricular dipoles (Fig. 3), since these are relatively clear of the lungs. A slightly more significant effect is noted for the majority of left ventricular dipoles, since these are within the left lung cavity (Fig. 4). Thus, in Figures 2 and 3, the only change worth mentioning is the slight increase in potentials due to the Z-oriented dipoles; this may be attributed to channeling of the Z-oriented current flow by the low conductivity lungs and was noted for all dipole locations not enclosed within the left lung cavity. Channeling effects due to the lungs were also anticipated for Y-oriented dipoles at these same nonenclosed locations, but were not always seen. Thus the Y-oriented dipole in Figure 2 is enhanced, whereas that in Figure 3 is enhanced only as far as potentials on the upper half of the torso is concerned. This serves as a reminder of the directional

![Figure 2](http://circres.ahajournals.org/)

**Figure 2.** Transfer coefficients for dipole location 4 in the septum plotted as the torso surface potential distributions due to unit dipoles in the Z, X, and Y directions (top, middle and bottom rows respectively) for the four torso conditions considered. The contour interval is constant at 250 μV for all distributions. The heavier contour line identifies the zero isopotential. The locations of the maximum and minimum for each distribution are identified by the plus and minus signs, respectively, and their values are indicated immediately below each distribution. For additional details, see text.
effect of the inhomogeneities on a given dipole. As a matter of fact, channeling effects for Y-oriented dipoles are most evident at locations along the Y axis, namely, the neck and lower extremity regions. Due to truncation, these regions are excluded from the surface potential distributions of Figures 2, 3, and 4, which represent body surface locations on the chest, sides, and back that are largely perpendicular to Y-oriented dipoles. A better vehicle for the manifestation of Y-directed channeling, as we shall see later, is the Y lead of the VCG.

As mentioned above, the effect of the lungs is more pronounced on the left ventricular dipole transfer coefficients. Thus, in Figure 4, on comparing columns 2 and 3, it is evident that the lungs have resulted in a rotational shift of extrema locations. Also for the Z oriented dipole, instead of a slight increase in surface potentials due to channeling, there is actually a decrease in potentials.

The Blood Masses

Qualitatively, the most important effect of the blood masses on the potential distribution is a slight rotation of the extrema positions, observed for sev-
eral, but not all, dipole locations. For some dipoles, e.g., 14-X and 14-Y, sandwiched between the lungs and the blood masses, the rotational effect of the latter is in a direction that counter-balances that due to the former (Fig. 4). Quantitative changes due to the blood masses are in general agreement with the Brody effect (see Fig. 1 for dipole positions). Dipole 4 in the septum offers a good illustration (Fig. 2). Thus, upon introduction of the blood masses, dipole 4-Z, being approximately perpendicular to the masses, is enhanced, resulting in larger surface potentials, whereas dipole 4-Y, being tangential, results in smaller potentials and is effectively diminished. No such clear-cut rationalization is possible for dipole 4-X, owing to the complex geometry of the blood masses. Again, in Figure 3, the augmentation of dipole 10-Z and the diminution of 10-X are due to these dipoles being mainly perpendicular and mainly tangential to the blood masses. Dipole 10-Y illustrates again the differing directional nature of the inhomogeneity effects, this time due to the blood masses, on the upper and lower torso halves. Finally, in Figure 4, the potentials due to dipoles 14-Z and 14-Y are diminished upon introduction of the blood masses, but the irregular geometry of the left ventricle makes it difficult to say whether this is in accordance with the Brody effect.

Inhomogeneity Effects on the BSPM

Many of the inhomogeneity effects noted for the individual dipoles are mirrored to a greater or lesser extent in the BSPMs generated upon varying the strength and orientation of the 23 Miller-Geselowitz dipoles so as to simulate normal cardiac activation (Fig. 5). As before, each column in Figure 5 represents one of the four different torso conditions. The four BSPMs for each torso condition correspond to four successive instants during QRS activation. The contour interval is kept constant throughout at 250 μV.

The most important effect of the muscle layer is the reduction of torso potentials everywhere (column 2, Fig. 5). Accompanying this reduction is a loss of certain secondary details in the BSPM, e.g., the secondary maximum at 30 msec into activation. Note, however, that neither the slight "niche" nor bend in positive isocontours at 30 msec indicative of the earliest onset of right ventricular breakthrough (Miller and Geselowitz, 1978; Liebman et al., 1981; Toyama and Tabata, 1981) nor the more important anterior pseudopod at 40 msec, characteristic of full right ventricular breakthrough (Taccardi et al., 1976), is completely lost. Also, the spatial separation of the extrema due to the muscle layer, noted with the

**Figure 5.** The simulated body surface potential maps generated by the Miller-Geselowitz heart model during four successive time instants during QRS (30, 40, 50, 60 msec after onset of activation). As before, the maps are shown for the four torso conditions considered, and the left and right halves of each map correspond to the chest and back, respectively. Potential values are plotted using Wilson's central terminal as a reference. The contour interval is constant at 250 μV. The heavier contour line identifies the zero isopotential; the plus and minus signs indicate the locations of the extrema, as in Figure 2.
individual dipoles, is less evident here. The introduction of the lungs, as might have been predicted, resulted in little qualitative change in the BSPMs. Even the rotational shift of the extrema noted for left ventricular dipoles (Fig. 4), did not, in the final analysis, manifest itself in the activation BSPMs. Only slight magnitude increases in the anterior chest potentials were noted, most probably due to channeling effects on the Z components of the right ventricular and septal dipoles. The situation was quite different, however, for the blood masses (column 4, Fig. 5). Appreciable increases in the magnitudes of the potential were often noted. This stems from the mainly radial nature of normal activation from endocardium to epicardium. Accordingly, most of the dipoles are oriented perpendicular to the blood masses and are enhanced due to the Brody effect. The second major effect of the blood masses was a considerable loss of detail in the BSPMs. Thus the niche at 30 msec is completely smoothed out and the pseudopod at 40 msec transformed into a niche. In effect, the detection of right ventricular breakthrough in the maps has been delayed. Interestingly, the “washing out” of the pseudopod by the blood masses is accompanied by a disappearance of the sternal minimum. This could explain why, in about 40% of normals, the sternal minimum is not seen (Taccardi et al., 1976). Thus—in general—the important inhomogeneity effects evident in the BSPMs are magnitude decreases and some loss of detail due to the muscle layer, very slight magnitude increases due to the lungs, large magnitude increases and important loss of detail due to the blood masses. Maps during the T wave, although not shown here, also manifested the above effects but to a lesser extent.

**Inhomogeneity Effects on the VCG**

The three cartesian plane projections of the simulated McFee VCG corresponding to normal activation of the Miller-Geselowitz heart model are shown in Figure 6 for the four torso conditions. The crosses indicate times \( t = 0, 10, 20, 30, 40 \) msec into activation and thus mark the direction of tracing of the QRS loop. The notched nature of the VCG loops occurs simply because the voltages corresponding to the different discrete activation times were joined by straight line segments. The smaller T wave loops have also been plotted.

**Figure 6.** The simulated McFee vectorcardiograms generated by the Miller-Geselowitz heart model for the four torso conditions considered. The crosses indicate times \( t = 0, 10, 20, 30, 40 \) msec into activation. The arrows mark the direction of tracing of the QRS loops. The smaller T wave loops are also shown.
Inhomogeneity Effects on the 12-Lead ECG

Six leads of the usual 12-lead ECG are depicted in Figure 7 for each of the torso conditions studied. The waveforms correspond to normal activation of the Miller-Geselowitz heart model. This figure reaffirms the observation that the muscle layer results in diminished potentials everywhere. The lungs, as may have been expected, increase the voltages in leads II and III due to Y-directed channeling. The blood masses increase lead voltages in all derivations. Earlier, we mentioned the smoothening or spatial filtering of the isopotential lines in the BSPM due to the blood masses and, to a lesser extent, the muscle layer. Figure 7 shows the temporal filtering of notches in leads II and III, as well as a slur in lead VI, due to the successive introduction of the muscle layer, lungs, and blood masses. Both types of filtering are really manifestations of the same phenomenon, namely, the blurring of individual dipole contributions at the surface due to the inhomogeneities, principally the blood masses. In one case the spatial separation of the dipoles is lost, in the other the temporal difference in their activation.

Discussion

The good agreement of the simulated BSPMs, VCGs, and ECGs with those reported for normal subjects in the literature serves as a validation of our computations with the inhomogeneous torso model. Since many of our observations are in accordance with those of Horacek, in spite of a different geometry for the blood masses and a different orientation for the heart, this also serves as an indirect check on the validity of his conclusions. Thus, as far as individual dipole transfer coefficients are concerned, we find that in general the Brody effect is valid, that the channeling due to the lungs results in effective increases of Y- and Z-oriented dipoles not enclosed within the left lung cavity, and that the blood masses tend to counteract lung effects for those left ventricular dipoles sandwiched between the lungs and blood masses. The effect of the muscle layer, not considered by Horacek, is mainly to diminish potentials everywhere.

From a clinical standpoint, with regard to the BSPM, VCG, and ECG, our simulations reveal that the major qualitative effects of torso inhomogeneities are an augmentation due to the lungs of Y-directed forces that is most evident in the VCGs; a smoothening of notches in the ECG (temporal filtering) and of isopotential contours in the BSPM (spatial filtering) due to the blood masses, muscle layer and lungs; and, occasionally, a slight pattern rotation and/or separation of the extrema in the BSPM due to the blood masses and, to a lesser extent, the lungs. Slight pattern changes are difficult to recognize in the 12-lead ECG. Usually, the ECG reveals no changes. On the other hand, it is also conceivable that a very slight pattern change can result in a significant change in one or two of the spatially fixed precordial leads, leading to an erroneous impression of the effects of the inhomogeneity responsible.

The spatial and temporal filtering due to the inhomogeneities may be explained, somewhat descriptively, as follows. In the homogeneous torso, each dipole has a particular region of the torso surface, usually that immediately overlying it, where its influence is largest. Activation changes in each dipole are thereby more directly reflected in the surface potential distribution, leading to secondary extrema and pseudopeaks. Notches in the ECG derivations appear due to two sequentially activated dipoles sharing the same region of influence on the torso surface. The inhomogeneities widen these regions of influence, the muscle layer by effectively displacing the torso surface outward, the blood masses by effectively augmenting the predominantly radially oriented cardiac dipoles. The widening inevitably leads to a much greater mixing of the regions of influence of the individual dipoles, resulting in the observed spatial and temporal filtering. Since the lungs mainly augment Y-directed dipoles, their filtering effects are usually limited to leads II, III, and aVF of the ECG and the Y lead of the VCG.
Apart from the filtering, which does lead to a loss of significant information, the qualitative effects of the inhomogeneities taken as a whole may be considered to be relatively slight. Their quantitative effects, however, can be quite large. This is especially true of the increase in potentials due to the blood masses and the decrease in potentials due to the muscle layer. These quantitative changes have important implications for the inverse problem of electrocardiography, where measured surface potentials are used to infer either epicardial potentials or equivalent heart dipoles. The effects of the muscle layer can probably be compensated for, perhaps by an appropriate scaling up of the measured potentials. Additional simulations by us, using simply the enlarged torso with no muscle layer conductivity interface, have revealed little difference, apart from a scale factor, from simulations with the conductivity interface present. Thus, another way to take the muscle layer into account, not only for the inverse problem, but also for forward problem simulations, is simply to use an appropriately enlarged torso model with no muscle layer conductivity interface. It must be cautioned that these approaches are valid only insofar as our original approximate approach for treating the anisotropic muscle layer is valid. A similar theoretical compensation for the quantitative and qualitative effects of the blood masses in the inverse problem is difficult, if not impossible. This is due mainly to the irregular and highly variable geometry of the blood masses and their close proximity to the cardiac sources. Thus, if an inverse solution in terms of equivalent heart dipoles is desired, no fixed stereotyped torso model inclusive of the blood masses can be constructed. Besides, the multipole expansion utilized as an intermediate step in one popular approach to these inverse computations is valid only outside a sphere enclosing all assumed cardiac dipoles, and hence cannot estimate the potential on the intracardiac blood masses (Cuffin and Geselowitz, 1977). This multipole expansion approach to the inverse problem therefore cannot be used to compensate for the blood masses, although it may be used to compensate for the lungs, since these are more stereotyped and lie outside the cardiac sources (Savard et al., 1982). The most promising inverse problem approach at the present time, par-
entially validated in dogs, is to calculate the epicardial potentials, using a Bayes estimator to obtain the transfer coefficients between epicardial and torso potentials (Hersh et al., 1978). This demands a large database relating epicardial and torso potentials, making it difficult to apply in humans. The estimated transfer coefficients would, however, incorporate the effects of all torso inhomogeneities.

In our simulations, the inhomogeneities were inserted in order of increasing importance. We clearly could not have inserted the blood masses first, as this would have completely masked the relatively slight effect of the lungs. One other order of insertion was tried, however, namely, first the lungs, next the blood masses, and finally the muscle layer. The effects of each inhomogeneity on the dipole transfer coefficients, BSPM, VCG, and ECG, remained essentially unchanged, the exception being that the loss of secondary details in the BSPM due to the muscle layer was less evident now that many of these details had already been obliterated by the blood masses.

Besides the order of insertion of the inhomogeneities, the adequacy of the Miller-Geselowitz heart model for a study such as ours may be questioned. A more complex heart model, with a greater number of constituent dipoles, would result in a few of these dipoles being closer to the lungs and blood masses than is the case at present. As a result, the effects of these inhomogeneities on the transfer coefficients of such dipoles will be more pronounced. However, it is unlikely that any new inhomogeneity effects will be unmasked, given the relatively minor effect of the lungs and the fact that many of our dipoles are already quite close to the blood masses. An increased number of dipoles should not greatly affect the homogeneous torso BSPMs, VCGs, and ECGs, since each heart model point is already within 19 mm of the dipole that represents it, and lumping errors are therefore unlikely (Miller and Geselowitz, 1978). However, even if it does, the widening and mixing of the surface zones of influence of each dipole due to the inhomogeneities, presumed to underly the phenomenon of spatial and temporal filtering, would still be present.

It is also useful to compare our theoretical study with that of Rudy and Plonsey (1979, 1980), who used an eccentric spheres model to simulate the heart and torso inhomogeneities such as the blood masses, the pericardium, the lungs, and the skeletal muscle and subcutaneous fat layers. Clearly, the two approaches are different, ours being a numerical finite-element simulation with realistic geometries, and theirs an analytic solution with idealized geometries. Each of the two approaches has its advantages and disadvantages. The analytical solution is simpler, its geometry and conductivity parameters easily adjustable, and the results may have general applicability. Its major drawback is that, in certain situations, it may not be sufficiently realistic. Thus, for example, in the Rudy and Plonsey model, the consequences of the spherical low-conductivity lung region completely enclosing the heart needs to be investigated carefully. On the other hand, the major advantage of the numerical finite-element approach is its greater realism and, consequently, a more true-to-life solution. Its drawbacks are that the solution is complex and needs careful validation, its geometry and conductivity parameters are fixed, and it may be difficult to extrapolate wide-ranging conclusions from the results. We have attempted to overcome some of these shortcomings, by careful validation of our solution based on the verification procedures discussed in Methods, and on the similarity of our ECGs, VCGs, and BSPMs with the literature, and also by being conservative in our conclusions. The complementary nature of the analytical and numerical approaches is nowhere more evident than in the thrust of the two investigations. Rudy et al. (1979) have concentrated on the effects of geometry and conductivity variations on body surface potentials in general, whereas we have attempted to show the inhomogeneity effects of a fixed-geometry and fixed-conductivity model on a particular example of a simulated ECG, VCG, and BSPM.

To the extent that the results of the two approaches can be compared, many of our observations are in accordance with those of Rudy and Plonsey (1979, 1980). Thus, we too confirm that inhomogeneities do not change the general pattern of the potential distribution, that it is unlikely that inhomogeneities will result in extra peaks on the surface potential distribution but, rather, will tend to smooth out activation details, that for normal endocardium-to-epicardium excitation, the blood masses override the effects of the muscle layer and make the source appear greater in strength than if the torso were homogeneous (Figs. 5–7). Note that the last is usually not true for the case of the individual dipole components (Figs. 2–4). Finally, the enlargement of the torso to simulate the muscle layer, coupled with the loss of activation details at the surface, demonstrates the effective shift of the heart sources toward the center, as noted by Rudy and Plonsey (1979).

Finally, an apparent discrepancy between Rudy and Plonsey's simulations and ours is in the importance they attribute to the skeletal muscle layer vis-à-vis our own observation that the muscle layer mainly scales down potentials everywhere on the torso surface. Rudy et al. (1979) found that the effects of varying lung conductivity can be markedly non-physiological if the skeletal muscle layer is not present, and therefore stressed that it must be included in any torso model. The nonphysiological behavior is attributable to their simulation conditions prior to introduction of the muscle layer. In their study, the skeletal muscle and subcutaneous fat layers were initially homogeneous with the underlying lung region. In other words, the low conductivity associated with the lungs extended all the way to the surface. Furthermore, the spherical low-conductivity lungs completely surrounded the heart. This type of situation does not arise in our simulations, where a high conductivity surface layer is always present, even prior
to inclusion of the muscle layer when a homogeneous high-conductivity torso is used. Thus we, in a sense, already have a sort of skeletal muscle layer at the start, avoid any nonphysiological simulations, and consequently, when a more appropriate muscle layer is introduced, see only a scaling effect.

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