Propagation Velocities and Voltage Magnitudes in Local Segments of Dog Myocardium

By R. H. Selvester, M.D., W. L. Kirk, Jr., Ph.D., and R. B. Pearson, M.D.

ABSTRACT

Propagation velocities and differential voltages of the electromotive surface in the left ventricle were measured in dogs by multipoint bipolar intramyocardial electrodes. The average velocity of propagation in the inner 4 mm of the ventricular wall of dogs was 2.0 mm/msec, which is considerably greater than that in the middle (0.70 mm/msec) or the outer 4 mm (0.40 mm/msec). The average voltage was less in the inner levels of the ventricular wall than in the outer, but significantly different from zero. This fact indicates a definite but lesser contribution per unit volume than from the outer levels. The propagation of the electromotive surface through the ventricular wall was consistently from endocardium to epicardium with no significant areas propagated in the reverse direction. These data disagree with the hypothesis that certain levels in the wall of the heart and certain regions of the heart are electrocardiographically silent. In dogs, and to a greater degree in humans, small infarcts anywhere in the heart can be expected to produce significant and discernible changes in surface ECGs. The task remains to refine and validate criteria for the diagnosis of infarct in these areas. When viewed from within the myocardium, the waveforms observed in bipolar intramyocardial electrodes are inconsistent with the dipole layer hypothesis as a model of the electromotive surface of ventricular depolarization in the heart.

ADDITIONAL KEY WORDS

ventricular depolarization
dipole layer hypothesis

Computer simulations of the vectorcardiogram (1, 2) and the total body surface electrocardiogram (3) have been reported. Good agreement was noted between simulated and clinically recorded vectorcardiograms of normal hearts, ventricular hypertrophies, and various large infarcts of the heart. The simulated total body surface ECG agreed well with Taccardi’s (4) human maps. The model upon which the simulations were based embodied the following assumptions: (a) the heart was a distributed set of fixed locus dipoles; (b) the volume conductor was resistive, isotropic, and contained internal inhomogeneities (lungs) and was limited by a normal male adult torso external boundary; (c) the ventricular activation sequence in humans was that reported by Durrer (5) and similar to that recorded in dogs by Scher (6); (d) the wave of depolarization through the ventricles was a current dipole layer; (e) the variation in the velocity of propagation of the wave of depolarization from endocardium to epicardium in the ventricular wall was so minimal that it could be ignored; (f) the electromotive source distribution and strength were always uniform over the entire propagation front.
The initial success with these simulations encouraged us to attempt a more precise simulation of the cardiac generator. The purpose of this study is to report results of these more precise experiments, which indicate that assumptions d, e, and f may produce significant error. It is our observation that variations in propagation velocity and source strength of the electromotive surface (wave of ventricular depolarization) must be taken into account when a more precise simulation of the dog heart is desired. Since there is no previous study of the magnitude of source strength from endocardium to epicardium and in various areas of the heart wall, this report is aimed at providing that information. Scattered velocity profiles are available in the literature but have not been systematically examined for various levels from endocardium to epicardium and for various regions of the heart. This study reports velocity profiles for a number of regions in several dog hearts.

Previous workers (6-12) reported extremely rapid propagation in endocardial regions as compared to the subepicardium. Prinzmetal et al. (13) and Sodi-Pallares et al. (9) particularly, argue from this point and from the morphology of intramyocardial waveforms that these regions do not contribute to a surface ECG. Additional evidence obtained from induced infarct data (13) indicates that infarcts in these inner areas do not produce discernible changes in epicardial and body surface electrical potential measurements. Durrer and co-workers (14, 15) took a contradictory position and argued that the inner portions of the heart are not electrically silent. Work in our laboratory on clinical and simulated vectorcardiograms in myocardial infarction suggests that the inner region of the ventricular wall also is not electrically silent (2). Myocardial infarcts observed at autopsy have been limited to the inner one-third of the left ventricle in patients whose VCGs clearly showed discernible changes in the QRS loop.

The dipole layer hypothesis (assumption d) has been used for years as a model of the electromotive surface of the heart. Our interest is to develop a simulation of the propagation of this surface through the heart that can be formulated from specific physiological parameters (charge density and dipole layer separation, etc.) which could be measured in the heart wall. The simulation then could generate the time histories for any given fixed locus dipole representing a segment of the heart in the model. To achieve our goal, it was necessary (1) to examine the average waveform seen on an intramyocardial bipolar electrode to determine if it was consistent with the dipole layer hypothesis and (2) to measure the parameters that define this electromotive surface.

FIGURE 1

Intramyocardial needle showing bundle wire construction with electrodes indicated by the small circles along the needle.
The small multipoint intramyocardial electrode developed by Pearson, and similar to those used by Scher (6), offers an excellent way to investigate these questions. These electrodes permit a detailed examination of the ventricular depolarization because a large number of electrodes can be placed in the ventricular wall without changing the dynamics of the heart or its activation sequence. Our assumption is that examining a large family of waveforms recorded close to each other in the ventricular wall will yield quantitative information about the form of the wave, the propagation velocities, and the voltage magnitudes. We are particularly concerned with the distribution of values of the variables at various levels in the heart wall and for various regions of the heart.

**Methods and Materials**

**Subjects.**—Seven dogs, each weighing approximately 40 pounds, were used in the experiment. Two animals were used in pilot studies to establish recording procedures and the experimental design. All the dogs were anesthetized with pentobarbital, 35 mg/kg. The results reported in this paper comprise data obtained from the remaining five dogs.

**Apparatus.**—Pickup from the myocardium was via electrodes constructed in the laboratory by Pearson, composed of a bundle of 15 stainless steel wires 0.005 inches in diameter (Fig. 1). Each wire terminated at a point 1 mm from adjacent terminations. The voltage differences between the 14 adjacent pairs were fed into 14 differential, solid state, portable preamplifiers designed in this laboratory. The differential impedance at the inputs was approximately 5 megohms. The common mode rejection was better than 80,000:1. The frequency response was 6 db.

**FIGURE 2**

Depolarization map of a vertical section of the left ventricular wall showing approximate intramyocardial needle placement. Numbers along each needle are the arrival times of the peak voltage between the electrode pair at that level. They are all referenced in milliseconds from an electrode placed in the outflow tract of the right ventricle.
down at 0.2 Hz and at 3,000 Hz. Most recordings were made with a preamplifier gain of 100. Each preamplified signal was fed “single ended” into an input of a 14-channel FM carrier tape recorder (Amplex FR 1300).

In all of these experiments, Channel 1 (nearest the needle tip) was omitted and replaced at the preamplifier input with a reference electrode held constant during the entire recording sequence. This reference electrode consisted of a pair of small stainless steel needle electrodes inserted into the right ventricle near the base and sutured in place. Any change in the waveform from this electrode was reason to terminate the experiment.

Analysis of both waveform and timing was accomplished by playing back from each channel a single action potential (one cardiac cycle) and the reference signal and displaying them simultaneously on a Tektronics Type 564 storage oscilloscope.

Experimental Design—Data were taken from each animal and are the basis for the activation map detailed in Figure 2. The voltage differences then were recorded for each adjacent pair of electrodes through the differential amplifiers described above. Isochronous time surfaces were plotted for each animal and are the basis for the activation map detailed in Figure 2.

Voltage measurements were made at 1-msec intervals. Input polarity was chosen so that propagation from within outward resulted in positive deflections when the outer electrode was more positive than the inner of any given pair. The voltages were measured in millivolts from the baseline or quiescent level.

Propagation velocity was calculated from the time difference of voltage maxima at adjacent electrode pairs. Individual data channels were related in time to each other by means of the reference channel. Velocity in meters per second was expressed as the distance between adjacent channels in millimeters divided by the difference in the times of voltage maxima in milliseconds (t). Since the spacing was 1 mm, the equation becomes \( V = 1/t \).

The myocardial wall was considered in three levels from endocardium to epicardium, each level being approximately 4 mm thick. These boundaries were arbitrary. The voltages and propagation velocities within each of these levels were grouped and compared with the other levels.

For each level (inner, middle, or outer) in each region studied (apex, midregion, or base), the average was somewhat more than three electrode pairs on each of the 25 needles, giving a total of about 85 possible measurements of voltage and about 75 of rate in each region in each animal. In some channels current of injury did not clear up, and in others there were prolonged voltage disturbances suggesting loss of electrode contact during contraction. All such records were excluded from this study. Only 46 such aberrant waveforms were discarded out of the total of 748 measurements. Other records were discarded at random to arrive at an equal number of measurements per cell for the analysis-of-variance tables. The difference in number of data values between the two measurement types occurred because the voltage magnitude required only one electrode pair, whereas propagation rate requires two.

To define the average waveform as seen by a bipolar pair of intramyocardial electrodes normal or nearly normal to the electromotive surface, each waveform was moved in time so that the voltage peaks corresponded. Since the waveforms were predominantly monophasic and positive, the major positive peak on each waveform was superimposed on the others. In those with a major negative deflection, this peak was superimposed for the purpose of averaging. Voltages at 1-msec intervals were averaged for 10 msec on either side of the peak.

Statistical Analysis.—The experiment embodied two 3 x 3 x 5 replicated, repeated-measure designs. This design requires more than one error term for the tests of significance, and the analysis-of-variance tables were constructed in this way (16). For the analysis of propagation rate, there were 12 values. The T-method of contrasts was used to test the cell means for a significant difference from zero (17).

Results

Figure 3 shows three averaged waveforms from each of three different levels in the wall of the myocardium (inner, middle, and outer 4 mm) taken from each of three areas (apex, midregion, base). These waveforms were obtained by averaging the voltages from the three levels. The point of maximum deflection...
(positive or negative) was used as the common point for averaging. The forms thus represent the average of 65 waves each. The deflection even in the inner 4 mm is positive and not zero or negative, which indicates an outward propagation on the average.

Figure 4 shows the results of analysis of the maximum voltage values. In the top graph, the means in millivolts are plotted for the inner, the middle, and the outer, 4 mm of the three left ventricular areas (apex, midregion, and base). There are clear differences between the inner, middle, and outer levels. There also appears to be an interaction among the various levels and areas because of the nonparallelism of the lines. The interaction appears to be due primarily to the change through the midregion.

The bottom graph in Figure 4 shows the mean maximum voltage plotted at each millimeter from endocardium to epicardium. The data from apex, midregion, and base were combined. Note the reduction in voltage as the wave front approaches the epicardial boundary.

Tables 1 and 2 show the results of the statistical evaluation of voltage magnitude. Table 1 is the analysis of variance. Only the sources of level (inner, middle, and outer) and the interaction of level with the area are significant \( P < .05 \). Table 2 shows the mean voltage magnitudes for levels and areas. The marginal means are the row and column averages. The means which are significantly different from zero \( P < .05 \) by the T-method are designated by an asterisk. None of the marginal means in the bottom row and right hand columns are zero and all are positive, which indicates that when the areas are combined, all levels of the myocardium have potentials significantly different from zero.

Since no significant differences were found between regions, the averaged magnitude results for apex, midregion, and base for 222 observations were combined, and the 95% confidence intervals for the means were calculated at each millisecond interval. Figure 5 shows this averaged result for the outer myocardial level.

**FIGURE 3**

Averaged waveforms for levels and regions of the left ventricle. The differences in peak voltages of the three levels can be seen.

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Figure 6 shows the propagation rate results. In the top graph the reciprocal of rate, labeled “time differences,” is plotted for the three levels in the three areas. Differences between the levels and the areas are obvious.

The bottom graph of Figure 6 shows the mean “time differences” plotted at millimeter intervals from endocardium to epicardium. Note that the change in “time differences” is almost linear. The mean propagation velocity for the inner 4 mm is 2.0 mm/msec, for the middle, 0.70 mm/msec, and for the outer, 0.40 mm/msec. Tables 3 and 4 summarize the results for the rate analysis. Table 3 shows that only the sources of level and area are significant (P < .05). Table 4 shows the mean time of occurrence between electrode pairs for levels and areas with marginal means. The asterisks indicate means which significantly differ from zero.

In theoretical simulations of a dipole layer and in tank experiments (18) in which all combinations of dipole layer and electrode separation was examined, triphasic or multiple peaked waveforms were routinely observed, with initial and terminal deflections of approximately 30% of the maximum deflection when the dipole layer separation approximated the electrode separation (Fig. 7). In no case was a monophasic waveform seen. These findings lead us to postulate that the dipole layer hypothesis as a model of the electromotive surface of the heart is inconsistent with waveforms observed in bipolar intramyocardial electrodes.

**Discussion**

This experiment yields significant conclusions about three of the assumptions previously mentioned: (1) the constancy of the electromotive source distribution and strength; (2) the uniformity of the propagation velocity of the advancing wave front from endocardium to epicardium; and (3) the

**TABLE 1**

Analysis of Variance for Voltage Magnitude

<table>
<thead>
<tr>
<th>Source of variation</th>
<th>Degrees of freedom</th>
<th>Sums of squares</th>
<th>Mean squares</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dogs (D)</td>
<td>4</td>
<td>820.9</td>
<td>205.2*</td>
<td></td>
</tr>
<tr>
<td>Error1</td>
<td>60</td>
<td>5544.3</td>
<td>92.4</td>
<td></td>
</tr>
<tr>
<td>Area: apex, midregion, base (A)</td>
<td>2</td>
<td>260.5</td>
<td>130.3</td>
<td></td>
</tr>
<tr>
<td>D x A</td>
<td>8</td>
<td>523.7</td>
<td>65.5</td>
<td></td>
</tr>
<tr>
<td>Error2</td>
<td>120</td>
<td>8487.6</td>
<td>70.7</td>
<td></td>
</tr>
<tr>
<td>Level: inner, middle, outer (L)</td>
<td>2</td>
<td>10712.1</td>
<td>5356.0</td>
<td>52.0†</td>
</tr>
<tr>
<td>D x L</td>
<td>8</td>
<td>823.8</td>
<td>103.0</td>
<td></td>
</tr>
<tr>
<td>Error3</td>
<td>120</td>
<td>11014.8</td>
<td>91.8</td>
<td></td>
</tr>
<tr>
<td>L x A</td>
<td>4</td>
<td>1488.0</td>
<td>372.0</td>
<td>5.5†</td>
</tr>
<tr>
<td>L x A x D</td>
<td>16</td>
<td>1696.0</td>
<td>106.0</td>
<td></td>
</tr>
<tr>
<td>Error4</td>
<td>240</td>
<td>16243.2</td>
<td>67.7</td>
<td></td>
</tr>
<tr>
<td>TOTAL</td>
<td>584</td>
<td>57614.9</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Error1 is the between-dogs error variance. Error2, Error3, and Error4 are the within-dogs error variance for effects listed above them. The interactions between dogs (D), areas (A), and levels (L) are indicated by initial letter and the x between \( \star \)P < .05.

**TABLE 2**

Mean Voltage Magnitudes for Levels and Areas with Marginal Means

<table>
<thead>
<tr>
<th>Area</th>
<th>Inner</th>
<th>Middle</th>
<th>Outer</th>
<th>Marginal means</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apex</td>
<td>3.1*</td>
<td>7.8*</td>
<td>14.0*</td>
<td>9.0*</td>
</tr>
<tr>
<td>Mid</td>
<td>2.2</td>
<td>11.5*</td>
<td>15.4*</td>
<td>8.7*</td>
</tr>
<tr>
<td>Base</td>
<td>4.6*</td>
<td>14.0*</td>
<td>13.1*</td>
<td>10.8*</td>
</tr>
<tr>
<td>Marginal means</td>
<td>3.9*</td>
<td>11.1*</td>
<td>14.2*</td>
<td></td>
</tr>
</tbody>
</table>

*P < .05
correspondence of the average waveform to the dipole layer hypothesis.

Although we attempted to place all needles normal to the isosynchronous electromotive surface, this was seldom accomplished precisely. This error always tends to produce shorter time differences and lower magnitudes, which partly explain our lower magnitudes and shorter time differences in the inner regions. At most, it accounts for only a small percentage of the lower magnitudes and shorter time differences, because all needles were within 30\degree of normal to the isosynchronous surface. The major difference in magnitudes is accounted for by the presence of many more inverted waveforms in the subendocardial regions than in the subepicardial ones. This finding, and the finding of a rapid propagation of the inner one-third, is consistent with the observation made by many investigators of Purkinje penetration and potentials in these areas.

The results for propagation rates (2.0 mm/msec) for the inner 4 mm of myocardium is in accord with the results of others (6-11) in that the rate was higher than in outer levels (0.4 mm/msec). However, the argument of some investigators that this high rate is sufficient evidence for the lack of contribution by the inner regions of myocardium to torso surface voltage is untenable. The measurements of voltage magnitudes discussed below clearly are inconsistent with such a conclusion.

The results presented herein indicate that all levels of the myocardial wall contribute to the electric field of the heart. All major areas (apex, midregion, or base) and all levels in the wall (inner, middle, and outer) are electrically active. Statistical analysis of these data shows clearly that the mean voltage

![Diagram](image)

**Figure 4**
Top: Comparison in mean millivolt peaks for the areas and levels of the left ventricle. The differences among levels can be seen. Bottom: Change in mean peak voltage, averaged over all areas, as the propagation front proceeds from endocardium to epicardium.

**Figure 5**
Plot of waveform which is the average of 222 bipolar electrode observations from the outer 4 mm of the left ventricular wall of the five dogs. Vertical lines are the 95% confidence limits of the mean values at each millisecond. The second curve superimposed is the waveform produced by a dipole layer that most resembled the observed average waveform. It can be seen that the large initial and terminal negative deflections extend several standard deviations outside the 95% confidence limits of the observed data.
TABLE 3

Analysis of Variance for Propagation Time Differences

<table>
<thead>
<tr>
<th>Source of variation*</th>
<th>Degrees of freedom</th>
<th>Sums of squares</th>
<th>Mean squares</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dogs (D)</td>
<td>4</td>
<td>15.2</td>
<td>3.8</td>
<td></td>
</tr>
<tr>
<td>Error1</td>
<td>56</td>
<td>173.6</td>
<td>3.1</td>
<td></td>
</tr>
<tr>
<td>Area: apex, midregion, base (A)</td>
<td>2</td>
<td>58.7</td>
<td>28.4</td>
<td>9.2</td>
</tr>
<tr>
<td>Error2</td>
<td></td>
<td>21.0</td>
<td>2.8</td>
<td></td>
</tr>
<tr>
<td>D x A</td>
<td>8</td>
<td>341.0</td>
<td>4.2</td>
<td></td>
</tr>
<tr>
<td>Level: inner, middle, outer (L)</td>
<td>2</td>
<td>389.5</td>
<td>194.7</td>
<td>42.3</td>
</tr>
<tr>
<td>Error3</td>
<td></td>
<td>22.7</td>
<td>2.8</td>
<td></td>
</tr>
<tr>
<td>D x L</td>
<td>8</td>
<td>506.0</td>
<td>6.3</td>
<td></td>
</tr>
<tr>
<td>L x A</td>
<td>4</td>
<td>17.5</td>
<td>4.3</td>
<td></td>
</tr>
<tr>
<td>L x A x D</td>
<td>16</td>
<td>52.2</td>
<td>3.2</td>
<td></td>
</tr>
<tr>
<td>Error4</td>
<td>220</td>
<td>836.0</td>
<td>3.8</td>
<td></td>
</tr>
<tr>
<td>TOTAL</td>
<td>540</td>
<td>2431.1</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

See footnote to Table 1.

*P < .05.

Top: Comparison in mean time differences (reciprocal of velocity) for the areas and levels of the left ventricle. The differences among levels and regions can be seen. Bottom: Change in mean time differences, averaged over all areas, as the propagation front proceeds from endocardium to epicardium. Note the apparent linear trend in the data.

Magnitude of the inner 4 mm of the left ventricle is not zero and has a positive value. Therefore, this level must contribute to the surface potentials, both of the heart and the torso, and cannot be considered "silent." This finding is contrary to the conclusions of other workers (11-13). The contribution of the inner levels, particularly in dogs, would be expected to be less than the outer levels,

TABLE 4

Mean Time of Occurrence between Electrode Pairs for Levels and Areas with Marginal Means

<table>
<thead>
<tr>
<th>Area</th>
<th>Level</th>
<th>Inner</th>
<th>Middle</th>
<th>Outer</th>
<th>Marginal means</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apex</td>
<td>.3</td>
<td>.8*</td>
<td>2.0*</td>
<td>1.0*</td>
<td></td>
</tr>
<tr>
<td>Mid</td>
<td>.4</td>
<td>1.3*</td>
<td>2.8*</td>
<td>1.5*</td>
<td></td>
</tr>
<tr>
<td>Base</td>
<td>.6</td>
<td>2.0*</td>
<td>2.8*</td>
<td>1.8*</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Marginal means</td>
<td>.5</td>
<td>1.4*</td>
<td>2.5*</td>
<td></td>
</tr>
</tbody>
</table>

P < .05.

TABLE 5

Number and Percent of Negative Voltage Maxima for Myocardial Level and Area

<table>
<thead>
<tr>
<th>Area</th>
<th>Level</th>
<th>Outer</th>
<th>Middle</th>
<th>Inner</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apex</td>
<td>1 (2%)</td>
<td>15 (20%)</td>
<td>20 (26%)</td>
<td></td>
</tr>
<tr>
<td>Mid</td>
<td>2 (2%)</td>
<td>8 (8%)</td>
<td>24 (32%)</td>
<td></td>
</tr>
<tr>
<td>Base</td>
<td>2 (2%)</td>
<td>3 (3%)</td>
<td>22 (29%)</td>
<td></td>
</tr>
</tbody>
</table>

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however, since they exhibited significantly smaller voltages. In no region of the left ventricular wall (exclusive of the papillary muscles) does the electromotive surface move predominantly inward. In no region is the surface predominantly negative or predominantly neutral as it would be if there were significant "islands of negativity" as postulated by Sodi-Pallares and Calder (12). The surface is, on the average, positive, directed outward, and moving outward in all parts of the ventricular wall.

The abrupt decrease in voltage as the epicardium is approached (Fig. 4) can be explained as an effect of the termination of the electric field lines at the epicardial boundary. This effect would be expected to be less in an intact animal because the normal fluid and tissue interface would have a higher conductivity than air.

Durrer detailed the activation sequence in a revitalized, perfused human heart that was activated through the atrium (5). In living intact humans and in the revitalized heart, he observed that the activation sequence was similar to that in dogs. The most important exceptions were a slower activation of the inner levels in humans and many fewer reversals (negative voltage maxima) in the bipolar records from the inner levels. These effects are probably due to less Purkinje penetration in the inner levels in man than in dogs. Table 5 tabulates the percent of reversed waveforms (negative maxima) from each of the levels examined, and shows that the inner levels in dogs have about 30% of their bipolar waveforms inverted. When the waveforms were averaged without including these inverted complexes, the peak magnitude for the inner 4 mm increased considerably to levels only slightly less than the outer regions. This finding indicates that in humans the inner levels of the myocardium, where reversals are rare, may contribute nearly as much to the surface ECG potentials as do the outer levels. The human bipolar waveforms published by Durrer (5) are consistent with this conclusion. It appears, therefore, that when the human heart is simulated, the assumptions of uniform generator strength over the electromotive surface and the assumption of a uniform velocity of propagation of that surface do not introduce significant errors, Vectorcardiograms which show definite local loss of forces frequently have been observed by us in patients where the fibrosis or infarct observed at autopsy is limited to the inner one-third of the ventricular wall. The simulated VCGs reported from this laboratory (2), which are partly based on the data reported in this paper, indicate that lesions less than 1 cm in diameter anywhere in the heart can be expected to produce definite changes in body
surface ECGs and VCGs. When the dog heart is simulated, however, the model must be modified to account for a significantly greater propagation velocity and a significantly decreased current source strength in the inner one-third to one-half of the ventricular wall.

In another study (18) we noted that a dipole layer immersed in a conductive medium and allowed to pass a bipolar electrode produced triphasic waveforms in general, with initial and terminal deflections of approximately 30% of the maximum deflection when the dipole layer separation approximated the electrode separation (Fig. 7). No combination of electrode separation or dipole layer separation produced monophasic waveforms. If a dipole layer were indeed the appropriate model of the front, then averaging around the peak value should produce a triphasic waveform for a dipole layer separation approximating 1 to 1.5 mm (as Durrer postulates) and an electrode separation of 1 mm. If a triphasic waveform were actually present as the wave front passed a large number of bipolar electrodes, then averaging about the peak would reduce the noise (both biological and electronic), and the more waveforms averaged, the more obvious the triphasic waveform would become. This statement would be true even if the width of the waveforms varied or if initial and terminal deflections were much smaller than would be predicted by a dipole layer separation of 1 to 1.5 mm. No triphasic waveforms emerged from averaging in this study. Figure 5 includes a 95% confidence limit for the data for the outer 4 mm from 222 locations and clearly shows that the waveform is monophasic; a significant triphasic wave could not be fitted within these confidence bounds. Therefore, we conclude that the dipole layer hypothesis as a model of the electromotive surface in the heart, when viewed from within the myocardium, is inconsistent with the predominantly monophasic waveforms observed on the bipolar intramyocardial electrodes in this study. These data do not bear directly on the question of whether the dipole layer idealization is adequate to explain potential distribution at a distance from the electromotive surface. Our previously reported simulations with multiple dipoles, as well as the work of investigators referred to earlier, indicate that when viewed at a reasonable distance, the dipole layer hypothesis may be a useful model to represent that surface. The important fact is that when one wishes to find the parameters by which to estimate the field strength from any myocardial segment based on intramyocardial measurements, as we wish to do, a model more appropriate than the dipole layer must be used.

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