A Study of the Human Heart as a Multiple Dipole Electrical Source

I. Normal Adult Male Subjects

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SUMMARY

A model for the electrical activity of the heart during depolarization, in which the ventricles were represented by 12 dipoles and the thorax by an inhomogeneous conductor, was evaluated. Potentials measured at 126 locations on the thoracic surface were used to determine the dipole strengths as a function of time for 58 normal sedentary subjects. Most of the dipole strengths had the form of a single pulse in time, the dipole onset sequence being consistent with the depolarization sequence found by others. Dipole activities (DA), obtained for each dipole by time integration of its strength, varied considerably between subjects. However, much of this variation arose from a systematic decrease of DAs with age. The separate DAs were summed to obtain the DAs of the septum and left and right ventricular free walls. The proportions of the summed DAs agreed with the cardiac component weights found by others. When the variation with the subject's age was removed, the total DA fluctuated by ±13% (standard deviation of the mean) in this series.

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I N T H E C O N C E P T U A L model underlying nearly all clinical electrocardiography (ECG) and vectorcardiography (VCG), the heart is represented as a single dipole generator (the "equivalent dipole"), and the thorax is treated as having uniform conductivity.1-3 This highly simplified model, although useful, is only the first step in understanding

the relationship between ECG potentials and myocardial changes. To obtain a more complete understanding of this relationship, with the associated possibility of more detailed diagnoses, more accurate models are needed.

Numerous papers have discussed more accurate models of the electrical source and the medium surrounding it,4-17 but little effort has been directed toward testing some of these ideas in clinical practice. The present study is a step in this direction. Our intention is to evaluate, for subjects without cardiac abnormality, the method we proposed earlier.17 This method involves multiple surface recordings, electrically complex torso configuration, and a multiple dipole representation of the ventricular myocardium as an electrical source. In the present paper we shall establish a normal range for the various parameters of the model.
The procedure involved in our method is, of course, more complicated than the conventional ECG. The key question is whether extra information is forthcoming to make the extra trouble worthwhile. In the following papers in this issue,18,19 we report studies of cardiac patients in which we found that information not obtainable by other atraumatic technics can be obtained from our method. If this experience is confirmed by others, multiple dipole ECG may be a useful complement to conventional ECG.

Models of the Heart as an Electrical Source

Experiments with intramyocardial electrodes20-23 have shown that at any instant of the QRS interval the depolarizing cells lie on imaginary surfaces. The location of the surfaces approximately 60 msec after the onset of QRS is indicated in figure 1A. As time progresses, the surfaces propagate through the myocardium (the “depolarization wave”) and may be represented electrically by moving dipole layers. Such a representation of the intramyocardial electrical events is probably fairly complete but is too complicated to be practical for clinical interpretation. It is necessary to use a less complete but more convenient characterization of the source.

The simplest characterization is the single dipole1-3 (fig. 1B). The dipole surfaces at any instant are approximated by the resultant of all the elemental dipoles on the surfaces, so that a single dipole represents the entire heart electrically. However, it can be seen that much more information is contained in the dipole surface than in the resultant dipole (it would clearly be impossible to reconstruct the dipole surfaces if only the resultant dipole were known) so that this characterization discards information which might be valuable.

Several proposals have been made for characterizations which are more complete than the single dipole but still simple enough to be practical. One approach is to recognize that the reason why the single dipole is incomplete is that the dipole layer shown in figure 1 has higher multipole moments.2,4-9 Thus, the resultant dipole is merely the first term in a series representing the source, and the inclusion of higher moments gives a more complete account of the source. Because the first term in this series is the most important, the single dipole approximation gives useful results. The obstacle to the clinical usefulness of the multipolar approach is the problem of physiologic interpretation of the higher multipole moments.

Another approach is to represent the dipole surface by a number of discrete dipoles,16-17 as illustrated in figure 1C. The advantage of characterizing the source by multiple dipoles lies in the possibility of associating each dipole with a particular segment of the myocardium.
A cardiogram might then be obtained for individual segments of the heart, rather than for the entire heart.

In this paper we use a multiple dipole characterization of the cardiac electrical generator. The ventricular myocardium is divided into 12 segments: four in the septum, five in the left ventricle, and three in the right ventricle. The direction of the dipole representing each segment is fixed in advance to be the average direction of propagation of the depolarization wave front in that segment of myocardium (in the absence of conduction defects). The dipole locations and directions are listed in table 1. The strength of the dipole is expected to vary with time, rising from zero as the depolarization wave front enters the segment and returning to zero when the wave front leaves the segment.

The strength of any given dipole at any instant should be approximately proportional to the instantaneous area of the depolarization wave in the segment represented by the dipole. A priori, the time integral of the dipole strength, should be approximately proportional to the amount of viable myocardium in the segment. Thus, in the case of hypertrophy we expect the integral to be increased, and in the case of infarction we expect it to be reduced. These assumptions provide a convenient means for analyzing the significance of the results obtained later.

### Table 1

<table>
<thead>
<tr>
<th>No.</th>
<th>Name</th>
<th>Abbreviation</th>
<th>x*</th>
<th>y*</th>
<th>z*</th>
<th>l*</th>
<th>m*</th>
<th>n*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Anterior basal septum</td>
<td>ANBS</td>
<td>0.075</td>
<td>0.035</td>
<td>0.253</td>
<td>0.805</td>
<td>0.563</td>
<td>0.187</td>
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<td>2</td>
<td>Low anterior septum</td>
<td>LANS</td>
<td>0.085</td>
<td>0.035</td>
<td>0.212</td>
<td>0.920</td>
<td>0.310</td>
<td>-0.200</td>
</tr>
<tr>
<td>3</td>
<td>Mid-anterior left ventricle</td>
<td>MALV</td>
<td>0.067</td>
<td>0.060</td>
<td>0.243</td>
<td>0.112</td>
<td>0.954</td>
<td>0.278</td>
</tr>
<tr>
<td>4</td>
<td>Lateral left ventricle</td>
<td>LALV</td>
<td>0.070</td>
<td>0.065</td>
<td>0.218</td>
<td>-0.097</td>
<td>0.621</td>
<td>-0.777</td>
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<tr>
<td>5</td>
<td>Diaphragmatic left ventricle</td>
<td>DIAP</td>
<td>0.060</td>
<td>0.020</td>
<td>0.198</td>
<td>0.000</td>
<td>0.000</td>
<td>-1.000</td>
</tr>
<tr>
<td>6</td>
<td>Posterior lateral left ventricle</td>
<td>PLLV</td>
<td>0.045</td>
<td>0.055</td>
<td>0.222</td>
<td>-0.707</td>
<td>0.707</td>
<td>0.000</td>
</tr>
<tr>
<td>7</td>
<td>Apex</td>
<td>APEX</td>
<td>0.095</td>
<td>0.060</td>
<td>0.222</td>
<td>0.070</td>
<td>0.707</td>
<td>0.000</td>
</tr>
<tr>
<td>8</td>
<td>Posterior basal septum</td>
<td>POBS</td>
<td>0.045</td>
<td>0.005</td>
<td>0.222</td>
<td>-0.829</td>
<td>-0.290</td>
<td>-0.479</td>
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<tr>
<td>9</td>
<td>Posterior left ventricle</td>
<td>PBLV</td>
<td>0.033</td>
<td>0.026</td>
<td>0.217</td>
<td>-0.707</td>
<td>0.014</td>
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<td>10</td>
<td>Anterior basal right ventricle</td>
<td>ABRV</td>
<td>0.097</td>
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<td>0.888</td>
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<td>0.222</td>
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<tr>
<td>11</td>
<td>Anterior apical right ventricle</td>
<td>AARV</td>
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<td>0.949</td>
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<tr>
<td>12</td>
<td>Posterior basal right ventricle</td>
<td>PBRV</td>
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<td>0.206</td>
<td>-0.053</td>
<td>-0.997</td>
<td>-0.053</td>
</tr>
</tbody>
</table>

* x, y, and z give the coordinates in meters of the dipole location. The x axis points forward, the y axis to the left, and the z axis upward. The origin is at the level of the umbilicus. l, m, and n are the corresponding direction cosines.

### The Thoracic Model

We consider the thorax as composed of three different materials: blood, lung, and the remaining "body tissue." It is necessary to specify the surfaces delineating these three different tissues, and this information was obtained from a cross-sectional anatomy text. From these cross sections, x, y, and z coordinates of many points on each of the surfaces of interest (endocardium, pleura, and skin) are obtained. These points are connected so that each surface is divided into triangles, making it possible to represent the surfaces in the computer. Thus, we have six regions (two lungs, two intracardiac blood masses, the remainder of the body, and the surrounding air). The electrical properties vary from region to region, but each region is treated as electrically homogeneous. The conductivities relative to body tissue were assumed to be 2.5 for blood and % for lung.

### Determining the Source Parameters from Surface Potential Measurements

Let us consider the preliminary hypothetical problem where the strengths of an N-dipole source are known and it is desired to determine the potentials produced on the thoracic surface. First consider only one dipole, with unit strength, and let the potential it produces on the surface of the...
Thorax at a given location, say the $i^{th}$ location, be $T_{ii}$. This quantity will be referred to as the transfer coefficient. In earlier papers we showed how to compute transfer coefficients for different geometrical and electrical assumptions. The particular geometric and electrical assumptions underlying the results of the present work were described above.

If the first dipole had strength $d_1$ instead of unit strength, the potential would be $T_{ii}d_1$. If we now activate all $N$ dipoles with strengths $d_1, d_2, d_3 \ldots d_N$, the resulting potential at the $i^{th}$ surface location denoted by $\phi_i$, is the superposition of the potentials due to each dipole separately:

$$\phi_i = T_{ii}d_1 + T_{i2}d_2 + \ldots + T_{iN}d_N$$

Similarly for the $j^{th}$ surface locations:

$$\phi_j = T_{i1}d_1 + T_{i2}d_2 + \ldots + T_{iN}d_N$$

and so on for any number of surface locations.

If the dipole strengths $d_1, d_2, d_3 \ldots d_N$ are known at some instant, the potential at any location on the thoracic surface can then be calculated from equations like those above. However, this hypothetical problem is not the one met in practice: here the surface potentials are known and the source strengths are to be determined.

If potentials ($\phi_1, \phi_2 \ldots$) at some instant are known at $N$ surface points, we can find the dipole strengths as follows: We write $N$ equations like those above in which the left-hand sides are now known, but the right-hand sides contain $N$ unknowns ($d_1, d_2, d_3 \ldots d_N$). The equations can then be solved simultaneously for the $d$'s, which are the dipole strengths at that instant. The solution is unique. Unfortunately this is an impractical procedure, since any measurement of the potentials is subject to noise and the transfer coefficients are not known with absolute accuracy. The solution to this difficulty is well known: surface potentials at more than $N$ points are measured and the $N$ unknowns are inferred using the "least squares fitting" (or "multiple regression") procedure.

In our case this procedure is modified further to ensure that the resulting solution represents a depolarization wave traveling from endocardium to epicardium, rather than in the reverse direction, which in normal subjects and many patients would be physiologically incorrect. To effect this, we require the constraint that the $d$'s all be nonnegative numbers. The mathematical technics for imposing this constraint were discussed in an earlier paper. The solution is still unique.

We have so far discussed obtaining the $d$'s at a given point in time. Clearly the same procedure can be followed for any instant in time, resulting in a profile for each dipole of its strength versus time. These results can be discussed in terms of the times of onset and offset of the dipole and the time-integrals of the dipole strength. As mentioned above, the latter quantity should be proportional to the amount of electrically active myocardium in the segment.

**Methods**

**Subjects**

This paper reports studies on 58 male subjects without cardiac abnormalities, whose chest configuration approximated the geometry of the model. The anthropometric data are presented in table 2. All subjects were judged to be physically sedentary. Since cardiac patients are usually sedentary individuals, normal values should be established in sedentary subjects.

| Table 2 |
|---|---|---|---|---|
| **Anthropometric Data on 58 Male Subjects** | | | | |
| Age (yr) | Height (inches) | Weight (pounds) | BSA ($m^2$) | Chest circumference (inches) |
| Mean | 39.0 | 69.6 | 166.4 | 1.92 | 37.8 |
| SD | 12.1 | 2.5 | 21 | 0.15 | 2.5 |
| Range | 16–72 | 63–76 | 120–230 | 1.56–2.30 | 32–45 |
Data Acquisition and Processing

A schematic representation of the data acquisition and processing system is shown in figure 2.

Electrodes are placed at 126 specified locations on the chest of the subjects (fig. 2A). The locations form 18 vertical columns, each containing seven electrodes. The columns are located round the circumference of the torso so that column 1 is in the V1 position, column 2 in the V1A position, and so on through column 18, which is in the V3R position. Within each column the lowest electrode is at the level of the mid-epigastrium and the highest electrode at the level of the manubrium sterni. There are exceptions to this under the arms, when the highest electrode is removed from the column and placed at the base of the neck. The objective of the placement of these electrodes is to obtain good coverage of the thoracic surface. An electrode is fixed at any convenient location and used to obtain a synchronizing signal (see below), and a potential reference electrode is placed in the right lower quadrant of the abdomen.

The thoracic electrodes are of a cup design with a silver conductor, 19 mm in overall diameter. The cup is filled with electrode paste, no paste being allowed to escape beyond the electrode border. The reference electrode is 22.5 cm² in size.

The electrodes are connected to AC high-gain amplifiers each with an input impedance of 5 megohms. Because of equipment limitations, only eight electrodes can be recorded at a time. The seven electrodes constituting a column are recorded together with a fixed synchronizing electrode. The column of electrodes is connected to the amplifiers through a rotary selector switch so that different columns can be selected successively until a complete set of data is built up.

The output of the eight amplifiers is recorded on eight channels on analog magnetic tape, using an Ampex DAS 100 Acquisition System.

To calibrate the system, square waves of known amplitude are the input to the eight amplifiers and recorded immediately before and after the ECG data. The recording conditions for each set of data (for example the anatomic location of the column) are carefully described using a voice track on the analog tape.

All electrodes are positioned, and all recordings are made, with the subjects in the supine position. Several cardiac cycles are recorded at each of the 18 columns, about 10 minutes being required to obtain a complete set of data. The subjects breathe quietly during each recording period.

Further processing of the data is done entirely on an IBM 1800 computer system. Interaction between an operator and the system is via a typewriter and a cathode-ray oscilloscope.

Two-second segments of the beginning calibration recording of each of the 18 sets of ECG recordings, and of the final calibration recording are digitized (fig. 2B). Each of the eight channels is sampled 1000 times per second, using a multiplexer. The data are stored in multiplexed (channels intermixed) form on digital magnetic tape until digitization for this subject is complete.

The data are then demultiplexed (fig. 2C) to separate the channels. The calibration signals are measured for each of the eight channels. This is done automatically, except that the operator indicates the edge of the square wave to the system. Subsequently, all ECG signals are

Figure 2

Schematic representation of the data acquisition and processing. Recording of surface potentials is shown in A. The switch is for selection of one of the 18 columns of seven electrodes each. The synchronizing signal is unswitched. In B 2 sec of each signal are digitized at a frequency of 1000 times per second using a multiplexer. In C the leads are separated (demultiplexed), adjustments are made for differences in amplifier gain and for drift of the zero level of potential, and the leads are synchronized. Finally, in D the refined and edited digital surface potential data are combined with the precomputed transfer coefficients. Output is in the form of multipole or multiple dipole strengths and can be plotted, tabulated, and stored on digital magnetic tape.
automatically scaled to their absolute values in microvolts. (The calibrations are later checked using the final calibration signal.)

The synchronizing channel associated with the first set of ECG data (the recordings for the seven electrodes of the first column) is then displayed to the operator. An easily identified point, for example the positive peak of the QRS complex, is selected and indicated to the system for use as a synchronizing point. The beginning and end of the time interval of interest is indicated to the system (usually a 100-msec period covering the QRS complex). In order to correct for offset and drift, the average of 10 msec of data on each channel is computed around a time point at which the synchronizing channel wave form suggests that there is negligible cardiac electrical activity (this is usually taken about 75 msec before the peak of the QRS complex). All the data for the selected time interval for the seven channels (that is, the QRS complexes at the seven electrodes in the column) are then displayed sequentially to the operator. For each electrode the operator has the choice of accepting the data if it looks satisfactory or rejecting it if it appears unsatisfactory. Data might be rejected because of noise or inconsistency with wave forms at neighboring points (a situation which arises if that electrode is making poor contact with the skin). Usually very few pieces of data are rejected. The data accepted are stored on digital magnetic tape.

The system then displays the synchronizing channel associated with the recordings for the second column of electrodes, and the operator again indicates the synchronizing point. It is assumed that the subject's heart activity is repetitive. Then, by indicating one corresponding time point (the synchronizing point) on different heart cycles, a set of data can be built up in 18 groups of seven recordings. The assumption is that this set of data is identical to that which would be obtained if 126 channels could be recorded at the same heart cycle. More sophisticated automated editing procedures have proved no more satisfactory than the technics described here. One reason is that we are determining 12 quantities (dipole strengths) from 126 quantities (recordings), and this large overdetermination factor leaves considerable room for errors such as misplacements of leads and recording-editing artifacts.

The unknown dipole strengths at each instant of time are obtained by fitting the data from all electrodes at that instant of time, following the procedure outlined in the description above of determining source parameters from surface potential measurements (fig. 2D). The transfer coefficients, with which the surface potential data are combined, are precomputed. At present we apply one set of transfer coefficients to all subjects. The constraint of nonnegative dipole strengths is imposed. However, no time constraints are imposed, that is, the solution at every time point is computed independently of the solution at any other time point. This is in contrast to the models of Selvester and associates and Horan and Flowers.

Routinely calculated from these data are the dipole strengths integrated with respect to time, which will be referred to as dipole activities (DAs) and the root-mean square (RMS) error between the potentials recalculated from the dipole strengths (that is, the potentials corresponding to the best available fit) and the experimentally measured potentials. The RMS error is used as a check on the consistency of the edited data, rather than as an indicator of the validity of the model. Another check on the consistency of the edited data is obtained by computing the fit for the first 15 terms (dipole, quadrupole, and octopole) of the multipole expansion and by examining the RMS error.

Results

Figure 3 shows results for a representative normal subject. The dipole strengths (constrained nonnegative) are plotted as a function of time over a 100-msec interval embracing the QRS complex. The curves for the most part have the form of a single pulse in time. No time constraints were imposed upon the model, so that the single pulses obtained are a significant result of the calculations.

The reliability of the dipole strength results has been tested in various ways. Recordings have been repeated at approximately 1-week intervals for two subjects. The relative times of initial activity and of peak amplitude of the dipoles were reproduced to within a few milliseconds, and the spread in total DAs was less than 10%. Similar results were obtained in repeated recordings from a subject who was asked to maintain various phases of respiration during the 10-sec recording period. Another test was to make a recording from a subject in the usual way, with all 127 electrodes in place at one time, and then to repeat the recording with only eight electrodes in place at a time. The results were essentially identical, showing that the presence of the electrodes does not significantly
disturb the potential distribution being measured. Finally, it was checked that the results for one patient did not change if different reference electrode locations were used (the standard location in the right lower quadrant of the abdomen, the right leg, and a remote water pipe).

One aspect of the dipole strengths is the time during which a particular dipole is "on." Figure 4 illustrates this for the nine left ventricular and septal dipoles averaged for 32 subjects.

The DAs were computed for the 12 individual dipoles and also for anatomic groups of dipoles such as those in the left ventricle. The means and ranges of the DAs for the normal subjects are given in Table 3. For comparison, the results for three representative cardiac patients, one with left ventricular hypertrophy, one with right ventricular hypertrophy, and one with myocardial infarction, are also shown.

The DAs tend to decline as the age of the subject increases. In figure 5 this is illustrated for the total DA (summed over all regions of the heart) of the 58 sedentary subjects. The same tendency was exhibited by the activities of all 12 dipoles separately.

Table 3

<table>
<thead>
<tr>
<th>Dipole</th>
<th>58 normal males</th>
<th>LVH: case 75</th>
<th>RVH: case 167</th>
<th>Myocardial infarction: case 164</th>
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<tr>
<td></td>
<td>Mean</td>
<td>sd</td>
<td>Range</td>
<td></td>
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<tr>
<td>ANBS</td>
<td>137</td>
<td>47</td>
<td>43-273</td>
<td>321</td>
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<tr>
<td>LANS</td>
<td>127</td>
<td>52</td>
<td>28-250</td>
<td>171</td>
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<tr>
<td>MALV</td>
<td>92</td>
<td>47</td>
<td>11-212</td>
<td>285</td>
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<tr>
<td>LALV</td>
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<td>56</td>
<td>27-279</td>
<td>554</td>
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<td>DIAP</td>
<td>225</td>
<td>145</td>
<td>26-663</td>
<td>729</td>
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<td>PLLV</td>
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<td>100-585</td>
<td>1458</td>
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<td>PBLV</td>
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<td>99</td>
<td>1-474</td>
<td>467</td>
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<tr>
<td>ABRV</td>
<td>109</td>
<td>45</td>
<td>15-334</td>
<td>152</td>
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<tr>
<td>AARV</td>
<td>46</td>
<td>28</td>
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<td>67</td>
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<tr>
<td>PBRV</td>
<td>103</td>
<td>53</td>
<td>4-242</td>
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<td>Septal total</td>
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<td>152</td>
<td>335-1010</td>
<td>2105</td>
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<tr>
<td>LV total</td>
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<td>238</td>
<td>469-1685</td>
<td>2774</td>
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<tr>
<td>RV total</td>
<td>256</td>
<td>91</td>
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<tr>
<td>Grand total</td>
<td>1761</td>
<td>401</td>
<td>1073-2971</td>
<td>5231</td>
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</table>

* Units are milliampere-meter-seconds. Abbreviations: Same as in table 1.

Discussion

From figure 3 it can be seen that the septal and left ventricular dipole strengths have the form of a single pulse in time. This is the way we expected the model to reflect the electrophysiologic situation, where the depolarization wave front moves through the myocardial segment.

According to Durrer, the depolarization wave front in the right ventricular wall moves mainly tangentially. This contrasts with the situation in the left ventricular wall, where

Table 4

| Comparison of Component Weights with Dipole Activities* |
|-------------|-----------------|-----|-----------|
|             | Dipole activities (58 sedentary normal subjects) | Weights† |
| LV          | Mean            | 46  | 49        |
| Total       | Range           | (34-57) | (37-60) |
| IVS         | Mean            | 31  | 36        |
| Total       | Range           | (25-43) | (24-50) |
| RV          | Mean            | 23  | 15        |
| Total       | Range           | (17-33) | (7-23)   |

*All quantities expressed as per cent
†Reiner and associates, 1959

Abbreviations: LV and RV = left and right ventricles; IVS = interventricular septum.
with the depolarization sequence established by work with intramural electrodes\textsuperscript{20-23} in which the anterior regions of the heart are activated first and the posterior regions last. The late onset of the dipole at the apex may be anomalous in this respect.

The activities are fairly uniformly distributed among the segments of the myocardium. In table 4 the summed activities of the dipoles located in the principal regions of the heart are compared to the weights of myocardium in those regions.\textsuperscript{28} Both quantities are expressed as a percentage of total. The agreement is good although the percentage of the DAs of the left ventricle and interventricular septum are slightly higher than the corresponding weights, and the right ventricular percentage DA is correspondingly lower than

Excitation moves continuously outward. This may explain why the activity of our right ventricular dipoles is drawn out in time (fig. 3) in contrast to the short pulse form of the left ventricular dipole strengths.

Figure 4 shows that there exists a characteristic dipole onset sequence for normal subjects. The sequence is fairly consistent

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure3}
\caption{Dipole strengths (constrained nonnegative) as a function of the time for a representative normal subject (case 256). The strength scale is the same for each dipole. The dipole sequence from top to bottom is determined by onset of activity in each, the earliest onset at the top, except that the right ventricular dipoles are placed at the bottom. Same abbreviations as in table 1.}
\end{figure}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure4}
\caption{Onset and offset of dipole activity, averaged for 32 subjects. The left end of the dotted bar represents the average onset and the right end the average offset for each dipole. The clear extensions represent 1 standard deviation. The time markers are 20 msec apart, time zero being the average dipole onset time (not QRS onset). Same abbreviations as in table 1.}
\end{figure}
the right ventricular percentage weight. This agreement is evidence that favors the idea that the DAs are proportional to the amount of muscle in the particular segment of the heart.

Figure 5 shows a definite negative correlation between total DA and the age of the subject. This corresponds to the age dependence of the conventional ECG. It would be interesting to know if this effect is caused by a change in the source or in the medium surrounding it (for example, by the development of a superficial fat layer). Since our calculations assume the same thoracic model for all subjects, we cannot distinguish between these possibilities.

Within the group studied there is considerable scatter of the separate DAs for the individual normal subjects (table 3). Part of the scatter is caused by the systematic variation of the activities with age. If the results for each subject are corrected to age 35 years, the scatter in the series is, of course, reduced. The standard deviation of the total activity from the mean is ±13% as compared to ±23% before age correction. No significant correlations were found between DA and other variables. Therefore, ±13% can be regarded as an intrinsic variation (that is, apart from age) among the normal subjects. This is comparable to the variation in heart weights found by Zeck for normal male subjects, the standard deviation in that study being ±17.5%. The DAs summed over regions of the heart showed less scatter than the separate DAs.

Even though the DAs of normal subjects scatter considerably, the patients with left and right ventricular hypertrophy have activities for the appropriate dipoles which are clearly above the normal range. The results for the patient with myocardial infarction show the DAs of the left ventricle, and specifically the posterior lateral left ventricle, to be below the normal range, as anticipated. These observations further support the conclusion that the method is successfully measuring the activity of segments of the heart.

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