Vector mapping of myocardial activation

ALAN H. KADISH, M.D., JOSEPH F. SPEAR, PH.D., JOSEPH H. LEVINE, M.D., ROBERT F. HANICH, M.D., CHARLES PROOD, M.S., AND E. NEIL MOORE, D.V.M., PH.D.

ABSTRACT A custom-made probe, consisting of four electrodes arranged so that two orthogonal bipolar electrograms could be recorded from a single site, was used to record epicardial activity during atrial and ventricular pacing in five normal and five anesthetized open-chest mongrel dogs with myocardial infarction. Unfiltered bipolar electrograms recorded with a 2 mm interelectrode distance averaged 36 ± 15 mV in amplitude and 16 ± 5 msec in duration in normal areas and 14 ± 11 mV and 23 ± 12 msec in infarcted areas (p < .01 infarct vs normal). The bipolar electrograms were vector summed so that a vector loop could be generated at each site. The direction of epicardial impulse propagation as determined by multipoint isochronal activation mapping was compared with that indicated by maximum x,y deflection of the vector loop. At 203 sites (141 normal and 62 infarcted) here was a median error of only 13 degrees and an excellent correlation by linear regression (r² = .95). In normal myocardium vector loops were straight (60%), open (21%), or hooked (19%). In infarcted myocardium, notched and irregular loops were occasionally seen. However, a clear maximum x,y deflection was still obtained from 98% of infarcted sites. During ventricular pacing in normal dogs, uniform epicardial conduction was observed for up to 4 cm longitudinal to fiber orientation but only 1 cm transverse to it. At selected sites longitudinal to fiber orientation conduction velocity was 0.618 n/sec, electrogram duration 12 msec, and vector amplitude 76 mV compared with 0.304 m/sec, 18 msec, and 38 mV during conduction transverse to fiber orientation (p < .05 for all comparisons).

Vector mapping of epicardial activation was performed during ventricular tachycardia induced by programmed stimulation in two of five 2-week-old canine myocardial infarcts. Aside from minor irregularities caused by impulse spread around areas of block, vector loops indicated when impulses were spreading away from the area of early epicardial activity and thus directed mapping to the region of earliest activation. We conclude that vector loops generated by summing orthogonal local bipolar electrograms accurately represent the direction of epicardial activation in both normal and infarcted myocardium. Such loops may prove useful in mapping tachycardias and in clarifying details about cardiac activation processes.

vector loops generated by such a method accurately reflect the direction of epicardial activation, to examine the morphology of such loops under different circumstances, to apply this information to study activation patterns in normal and infarcted myocardium, and to use the technique to map experimental VT.

Methods

Experimental protocol. Studies were performed in five normal mongrel dogs and five dogs with 2-week-old anterior myocardial infarction created by an occlusion-reperfusion protocol previously reported. Each dog was anesthetized with pentobarbital sodium (30 mg/kg) and ventilated via a tracheostomy. The heart was exposed via a left thoracotomy and a pericardial cradle was created. A clear, soft plastic 6 x 4 cm guiding plaque was sutured to the exposed cardiac surface over areas of the right ventricle and interventricular septum and extending to the left ventricular apex. The guiding plaque contained 40 equally spaced sites separated by 0.75 cm and arranged in an 8 x 5 array. To evaluate various activation patterns, bipolar plunge stimulating electrodes were placed just beneath the ventricular surface at one to five sites in and around the plaque. A bipolar plunge electrode was also placed in the left atrial appendage. Atrial and ventricular stimulation was performed with rectangular pulses of 2 msec in duration at two times diastolic threshold delivered by a standard stimulator (Bloom Associates). Pacing was performed at cycle lengths just below the sinus cycle length. In the dogs with infarction standard programmed electrical stimulation techniques were used to induce VT. Rectangular pulses of 2 msec duration were delivered at twice diastolic threshold. A basic drive at a cycle length of 300 msec was delivered for 8 beats, followed by an extrastimulus introduced up to the shortest possible coupling interval. Single, double, and triple extrastimuli were introduced to initiate VT. Up to five sites around the recording plaque were evaluated in an attempt to initiate VT. A custom-made, hand-held bipolar recording electrode (figure 1, A) containing four 0.4 mm diameter electrodes arranged in two orthogonal pairs was used to record electrical activity. The center-to-center interelectrode distance was 2 mm along each axis and electrograms were filtered at 1 Hz to 1 kHz (see below: Justification of the vector method). The body surface electrocardiogram and two bipolar recordings were displaced simultaneously on an oscilloscope and recorded on a 16-channel electrostatic strip-chart recorder (Gould ES1000). In addition, the x and y bipolar recording electrodes were placed into x and y axes of an oscilloscope, where they could be observed and photographed on 35 mm film. An example of the vector loop produced in such a fashion is shown in figure 1, B. Amplitudes and durations of electrograms were manually digitized and measured with a Hewlett-Packard 9836 computer and digitizing system. Vector loops were analyzed in several ways. In all cases, an x axis was defined according to the orientation of the probe relative to the long axis of the mapping template (figure 2) and the angle between this reference and a line from the origin to the point of maximum x,y deflection of the vector loop was determined. In addition, in a large number of representative electrograms, the maximum amplitude of the vector as well as the direction and speed of vector motion throughout its cycle were determined by manually digitizing the x and y electrograms and using computer generated vectors.

In each dog, 40-point maps of ventricular activation were derived during atrial pacing and ventricular pacing at one to six different sites. In three dogs, collisions were set up by pacing simultaneously from two different ventricular electrodes. The

FIGURE 1. A. Photograph and schematic diagram of the electrode array of the hand-held epicardial probe. Electrode tips were 0.4 mm in diameter and interelectrode distance along both the x and y axes was 2 mm. One of the electrodes along the x and y axes was arbitrarily designated as positive and a marker on the probe allowed it to be positioned similarly at each mapping site. A metal ring 5 mm in diameter was placed 0.5 mm from the tip of the electrode. This stabilized the probe on the epicardial surface and decreased the current of injury. B. Example of a vector loop generated on a standard oscilloscope during propagation almost parallel to the x axis electrode. The amplified x and y electrogram outputs were fed into the respective axes inputs of a standard oscilloscope and the beam was intensity modulated by a 4 V sawtooth at 3 kHz. The arrow indicates the direction of the beam around the loop. In this and future examples, the narrow point of the loop points in the direction the loop is moving, and the length of individual dots identifies the speed of vector motion with each dot representing 0.33 msec in time. Both direction and time information can be gained from inspecting such a loop.

The effect of propagation both longitudinal and transverse to fiber orientation on the vector loop was determined at selected sites by using areas in which uniform fiber orientation was present. In the two dogs in which VT was induced, tachycardia mapping including additional sites around the periphery of the mapping template was performed.

To compare vector mapping with activation mapping, 40-point isochronal activation maps were obtained by digitizing intrinsic activation times from the bipolar x and y coordinates for each of the sites. In the dogs that had undergone occlusion of the left anterior descending coronary artery, approximately 25 of the 40 sites in the map were over infarcted areas. Although there was some variability in infarct location among the dogs, in general the anterior left ventricle was involved. In each case, activation time was determined at the point at which the most rapid deflection crossed the isoelectric baseline, with two elec-
trograms being available for each site. If no isoelectric crossings occurred within the electrogram, the activation time was taken as the point of maximum rate change in the most rapid deflection. The x-y electrogram with the earlier activation time was used as the activation time for that site. Isochronal activation maps were generated with a Hewlett-Packard 9836 computer plotting system using the CONDOT algorithm of Simons as previously described and were analyzed by one of us (J. S.) who had not examined the vector data. Activation directions were blindly assigned to each map site based on a line perpendicular to the isochrone tangent. To validate the vector technique, we defined criteria that would ensure that the activation direction from the isochronal activation map was clear and accurate. They included uniform parallel isochrones (5 msec apart) on each side of the activation site and a conduction velocity greater than 0.1 and less than 1 mm/sec. Velocities faster than this would likely involve activation of the His-Purkinje system, resulting in complicated activation patterns on the surface; slower velocities could represent areas of discontinuous conduction or block. One hundred forty-eight of 520 sites (28%) in the normal dogs and 64 of 300 (21%) infarcted sites fulfilled these criteria, and an "isochronal angle" of propagation was obtained for these sites. Similar angular data were calculated by computer from the digitized vector loops and plotted on a separate isochronal map of the same activation. In 141 normal sites and 62 infarcted sites, both a vector and isochronal angle was available and these were used for comparison. In this way, propagation direction from vector and isochronal data were compared for each site.

FIGURE 2. Schematic diagram of the approximate position of the guiding plaque on the epicardial surface in each of the dogs. Forty-point maps were obtained from each of the numbered sites. For the purpose of orienting the epicardial probe to the heart, the positive x axis was arbitrarily designated as pointing along the plaque toward the right ventricular free wall and a positive y axis toward the left ventricular apex as shown by the arrows.

Statistics. Correlation coefficients between the vector- and isochrone-derived activation angles were determined by linear regression. An angular representation system spanning from 0 to 180 and 0 to -180 was used. In 13 of 203 angle pairs, one of the angles was in the positive domain and the other in the negative domain, complicating the performance of linear regression. In these cases the positive angle of propagation was converted to a negative angle by the Von Miller Fisher procedure. Electrogram and vector characteristics were compared by paired or unpaired t test when appropriate.

Justification of the vector method. Durrer et al. proposed in 1961 that the morphology of extracellular electrograms or electrocardiograms can be explained by the assumption that the current generators that produce recorded voltage can be represented as a uniform double layer source, occurring at the interface between resting and depolarized fibers. An assumption inherent in this hypothesis is that points on the same side of this boundary are isopotential. Experiments performed by varying the interelectrode distance of a recording bipolar suggest that the width of this arbitrary boundary separating two different isopotential areas was approximately 0.9 ± 0.1 mm. Recently, it has become clear that a consideration of the anisotropic properties of cardiac muscle is necessary to fully describe the potential fields that exist on the epicardial surface and predict the morphology of extracellular electrograms and that the boundary over which the largest potential drop occurs may be up to 3 to 4 mm. Although anisotropy introduces complexities into the extracellular potential distributions, the largest potential differences still occur at the interface between resting and depolarized tissue.

Voltage recorded across a bipolar extracellular electrode will depend on the difference in the potential field present between the two electrodes in any given instant. If a wavefront is traveling toward and parallel to the axis of a bipolar of some given interelectrode distance, the edge of activation will first approach the proximal pole and produce a small positive deflection. After the area under this electrode has been depolarized, a negative extracellular potential will be recorded at this electrode that is larger in magnitude than the initial positive deflection. Just after the impulses pass the first pole of the bipolar electrode, the distal electrode will still be recording a zero or slightly positive potential, resulting in a large negative bipolar recording. After the impulse passes the second electrode, a terminal positive deflection may be produced. If an impulse were traveling exactly perpendicular to such a bipolar electrode and the field were uniform, no deflection would be recorded on this electrode at all because both poles would always be isopotential. Obviously intermediate amplitudes would be recorded depending on the angle between the propagating impulse and the electrode. If two such bipolar electrodes were arranged in an orthogonal array, the vector sum of these two electrograms would be determined by the relative angle of propagation to the two bipolar pairs. In the simplest instance in which an impulse is propagated parallel to one of the pairs, a large either positive or negative deflection will be recorded by that pair depending on the direction of propagation relative to the positive pole. The other electrode pair would record no electrical activity. The resultant vector would be a large deflection in the direction of impulse propagation. In situations in which conduction direction is changing beneath the surface of the electrode and thus the potential field is more complex, more complicated patterns will be produced.

We selected a 2 mm interelectrode distance for each of the orthogonal poles for our experiments for a variety of reasons. Vander Ark and Reynolds demonstrated that for bipolar electrodes, the maximum recorded amplitude is obtained when the interelectrode distance is significantly larger than the boundary
surface between depolarized and resting tissue. In their experiments, the surface was 0.9 ± 0.1 mm wide. Because of slower conduction transverse to fiber orientation, it would be expected on theoretical grounds and has been shown experimentally that the interelectrode distance to record maximum amplitude during transverse conduction would be somewhat larger. Thus we selected a 2 mm interelectrode distance to obtain close to the maximum extracellular electrogram amplitude while still recording relative local activity. We obtained lower amplitude vectors with a similar maximum direction by means of a probe with a 1 mm interelectrode distance. However, in addition to decreases in amplitude, this small probe tended to produce injury potentials complicating the vector loops. Larger interelectrode distances would not have allowed the definition of conduction direction in relatively small areas. Also, we found empirically that use of high-pass filters with frequencies greater than 1 Hz produced truncation of the vectors and loss of information.

Results

Electrogram characteristics. We analyzed electrogram characteristics obtained from 200 normal and 125 infarcted sites with two electrograms being recorded from each site. Only one electrogram pair was analyzed from each site even if multiple maps were obtained in a particular area of myocardium. The infarcted sites were defined by their location in a characteristic area of the left ventricle rather than by physical or electrophysiologic criteria. Electrogram characteristics comparing normal and infarcted regions are shown in table 1. We defined criteria for abnormal electrograms based on being more than 2 SD from the mean for normal sites in amplitude, duration, or ratio. As a group, electrograms recorded in infarcted sites had significantly lower amplitudes and longer durations. Ninety-four of 250 electrograms recorded from infarcted areas were abnormal and in 36 of 125 sites both electrograms were abnormal.

Isochronal activation mapping. An example of a 40-point isochronal activation map obtained during pacing from near the left anterior descending artery in a normal dog is shown in figure 3. In all the dogs we used a similar plaque orientation (see figure 2). The upper border of the plaque was approximately the territory of the lower mid-portion of the distal left anterior descending artery, the left side of the plaque along the anterior left ventricle, and the right side of the plaque along the anterior right ventricle. In the experiment shown in figure 3, there was a relatively uniform epicardial impulse spread for approximately 1 to 3 cm from the pacing site. The large dark arrows note the positive x and y directions. In the upper left-hand corner of the plaque area (left ventricular apex), there was evidence of early breakthrough, presumably via His-Purkinje activation. In panel A, the narrow arrows indicate the measured directions of the vector loops. In panel B, propagation directions are indicated by the open arrows (see Methods) based on the isochrones for sites where we could be certain of the direction of impulse spread.

In most experiments fiber orientation was relatively uniform parallel to the long axis of the plaque over the left ventricle. In dogs without myocardial infarction, we were able to observe uniform conduction across almost the entire mapping area when we paced from the middle or from either side of the plaque. Conduction velocity longitudinal to fiber orientation appeared to be rapid enough to allow most epicardial activation in the measured area to occur via muscle-to-muscle epicardial spread without involvement of or breakthrough via the His-Purkinje system. When pacing from above or below the plaque near the proximal or distal left anterior descending artery, conduction near the pacing site tended to be perpendicular to fiber orientation and extremely slow (figure 4). In this case uniform epicardial conduction extended less than 1 cm, after which the remainder of the mapping area was activation in an irregular complicated fashion by other routes. Isochronal mapping during atrial pacing or sinus rhythm also demonstrated irregular epicardial breakthrough at multiple sites with only a few sites being found where an unequivocal direction of epicardial spread was evident. Since we could rarely be certain about the local direction of impulse propagation, we did not use atrial pacing maps for validation of vector directions. Of 820 sites obtained in 24 separate

<table>
<thead>
<tr>
<th>TABLE 1</th>
<th>Electrogram characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal (n = 400)</td>
<td>Criteria for abnormality</td>
</tr>
<tr>
<td>Amplitude</td>
<td>36 ± 15 mV</td>
</tr>
<tr>
<td>Duration</td>
<td>16 ± 5 msec</td>
</tr>
<tr>
<td>Amplitude duration ratio</td>
<td>2.48 ± 1.13</td>
</tr>
</tbody>
</table>

\(^a\) p < .001 infarct vs normal.
ventricularly paced maps, we were able to obtain definitive directions of isochronal activation at 212 sites. Conduction velocity in these sites ranged from 0.101 to 0.985 m/sec. In some instances, isochrones in infarcted areas were irregular or electrograms so abnormal that a clear activation time could not be defined nor could a clear direction of epicardial activation be determined from the isochronal maps. In other instances, despite markedly abnormal electrograms, activation times could be identified.

**Validation of vector directions.** Vector loops were created for each of 520 epicardial sites during 13 separate epicardial maps in normal dogs and for each of 300 sites during 12 maps of dogs with infarction using the x and y inputs of an oscilloscope to instantaneously vector sum the orthogonal bipolar electrograms recorded from the probe electrode. An example of the most typical kind of vector loop appears in figure 1. At the first approximation, we chose the angle from the origin to the maximum vector amplitude to indicate the propagation direction. Other portions of the vector loop may provide different and additional information as discussed below. We were able to obtain a clear maximum vector amplitude in 805 of 820 sites evaluated. In 203 of these sites, both the isochronal activation maps and vector loops demonstrated a clear direction of activation. Thus these 203 sites were available for the correlation between isochronal- and vector-derived directions. Figure 5, A, demonstrates that there was an excellent correlation between the isochrone direction and the vector direction. In only six of 203 sites (3%) was there more than a 60 degree difference between the two methods. The median angle error was 13 degrees (figure 5, B), and vector loops were equally accurate in areas of slow or fast conduction (figure 6). Although the median angle of error in infarcted tissue was slightly larger than that in normal tissue (14 vs 12 degrees), the correlation coefficient for infarcted sites was still excellent ($r^2 = .95$).

**Vector loop morphology in normal myocardium.** Vector loop morphology created by continuously summing the x and y coordinate inputs at a given site was consistent on a beat to beat basis. We observed three major types of vector loop morphologies in normal dogs in our recordings: (1) Narrow, defined as a loop with a less than a 30 degree divergence among instantaneous vector angles at more than 75% maximum amplitude. An example of this is shown in figure 7, A (60% of vectors). (2) Open, in which a more than 30 degree angle of divergence was present between instantaneous vectors at more than 75% maximum vector amplitude (figure 7, B) (21% of vectors). (3) Hooked, an example of which is shown in figure 7, C (19% of vectors). For open vectors, the vector direction was taken as a point bisecting the open portion of the loop. In evaluating hooked vectors the “hook” was ignored.

We observed open vectors in three different circumstances. In areas where isochrone direction changed near the recording site, vectors were open. An example
of such a site and the vector produced are shown in figure 8. Before the local activation at the site, impulse propagation was spreading in an angular direction toward the left lower portion of the plaque. The initial part of the vector loop produced was thus in that direction (1 in figure 8, B). Around the activation site, isochrone lines curved to a more vertical direction and the vector was more horizontal at this point (2 in figure 8, B). Open vectors were also observed in areas of collision (figure 9), presumably because of a similar time-dependent phenomenon involving local changes in propagation direction. Finally, marked ST segment elevation or depression caused by current of injury produced a displacement of the terminal baseline and an opening of the vector. Hooked vectors were observed during propagation parallel to one of the electrode pairs and thus one electrode pair was recording little activity. These hooked vectors could have been caused by a brief period of time in which electrode pairs that were essentially recording no activity (because the impulse was almost exactly perpendicular to them) were briefly in areas of slightly differing potential as the wavefront passed. Hooked vectors may also have been produced by local discontinuities in conduction.

Vector morphology in areas of differing fiber orientation. Although there were relatively few sites available in which epicardial conduction occurred transverse to fiber orientation, we were able to identify 15 separate sites in which uniform fiber orientation could be determined by visual inspection of the epicardial surface and in which isochronal activation maps confirmed that propagation direction was transverse. We compared these with a similar number of sites during which longitudinal conduction occurred. Mean conduction velocity for sites of longitudinal conduction was 0.618 m/sec and that for sites of transverse conduction was 0.304 m/sec (p < .05). Mean amplitude of loops was higher for sites of longitudinal (76 ± 14) than transverse (38 ± 24) conduction (p < .005). Mean electrogram duration was 12 ± 4 msec for longitudinal sites and 18 ± 6 msec for transverse sites (p < .05). Examples of sites that were considered to represent uniform longitudinal and transverse conduction can be seen in the starred areas of figures 3, A, and 4.

Spatial area and time dependence of vectors. The duration of electrograms in areas of uniform longitudinal and transverse propagation were measured to gain insight into the spatial area in which electrical activity affects the resultant vector loops. We measured the time from the earliest onset of a rapid deflection to the latest rapid deflection in either bipolar electrogram at a single site. By calculating conduction velocity at that site we were able to estimate over what distance (velocity × time) rapid deflections were being produced in the local electrogram. In areas of longitudinal conduction, we estimated that an average distance of 3.7 ± 1.2 mm on either side of an arbitrary central point influenced the vector loop produced at a site. For areas of transverse conduction, we estimated a 2.7 ± 0.9 mm "field of view." During the 10 to 20 msec over which impulse propagation affected the vector recorded at a given site, the direction of epicardial activation could potentially undergo major changes. Vector directions before the intrinsic activation time should represent the direction of impulse spread approaching the recording site and vector directions after the activation time should represent the direction of impulse spread.
as the impulse is receding from the recording site. Examples of open vectors demonstrating this phenomenon have already been discussed and are depicted in figures 8 and 9.

Vector morphology in infarcted myocardium. Figures 10 and 11 present an isochronal map and selected vectors and their component electrograms from an animal with infarction. As mentioned previously, despite markedly abnormal electrograms activation times could usually be identified in infarcted regions. In such cases the direction of maximum vector amplitude showed a remarkable correlation with the isochronal maps. It can be seen in figure 11 that although the electrograms and resultant vectors were of lower amplitude and more complex in the infarcted region (B through F) than in the normal area (A), a direction of maximum vector amplitude could still be determined. In 294 of the 300 infarcted sites (98%) a predominant activation direction was apparent.

In addition, the irregular vector loops with multiple notches may reflect underlying complex conduction in the infarcted region. Although it is difficult to be certain, we believe that each of these notches and time periods of different directions represents wavefronts of activation occurring within the field of view of the vector probe. This can be seen especially for vectors E and F of figure 11, which were recorded in areas showing complex local activation (figure 10).

Mapping of VT. We were able to induce and completely map VT in two of five dogs with infarction. An example of one such 63-point map is shown in figure 12. The tachycardia in this dog had earliest sites of epicardial activation along the anterior left ventricle. A portion of a reentrant circuit may have been identified, although the large interpoint distance and the lack of

FIGURE 5. A, Correlation between the direction of impulse propagation determined by the vector and isochronal techniques. The positive x direction shown in figures 2 and 3 was arbitrarily designated as 0 degrees in all experiments, and angles counter-clockwise positive to 180 and clockwise negative to 180 were assigned. The crosses represent sites in normal areas and the dots sites in infarcted areas. An excellent agreement was noted between the two methods. There were fewer points in the +90 and -90 degree regions because fiber orientation tended to be parallel to the x axis and uniform epicardial spread perpendicular to fiber orientation occurred over only small areas as noted above. However, in sites in the +90 and -90 degree range that were adequate for comparison, the agreement between the two methods was just as good as in other areas. B, Histogram showing the angle of error between the two methods in each of the 203 sites. Median angle of error was only 13 degrees, and in only six of 203 sites was an error greater than 60 degrees present.

FIGURE 6. Differences in propagation angles between vector and isochrone indicated propagation directions at each of the 203 sites (ordinate) and conduction velocities at those sites (abscissa). Vector directions were equally accurate in the areas of slow or rapid conduction. The crosses represent sites in normal areas and the dots sites in infarcted areas.
plunge electrode recording did not allow certain identification of a reentrant circuit. In figure 12, A, an area of early epicardial activity during the tachycardia (in the area of the circle) could be readily identified by examining vector directions that pointed away from this site throughout the entire mapping area (anterior right and left ventricular including left ventricular apex). In addition, a complex pattern of impulse propagation spreading in both directions around an area of block on the anterior left ventricle (solid curved line) could be seen. We also examined sites on the posterior left and right ventricles and found vector directions pointed away from the area of early epicardial activity. The isochrone map as shown in figure 12, B, verified these observations. In figure 13, representative examples of vectors and their component electrograms from sites identified by the lettered circles in figure 12, B, are shown together with the electrocardiogram and 

FIGURE 7. Three examples of typical normal vector morphologies and their component electrograms. The open arrows indicate activation times. A. The most commonly observed vector morphology (60% of vectors) was straight and narrow. B. An open vector (21% of vectors) defined as a more than 45 degree difference between two different instantaneous vectors (at a time of greater than 75% of maximum vector amplitude). C. A typical hooked vector (19% of vectors). These vectors generally had a morphology similar to that of straight vectors except sharp narrow hooks appeared at the end. See text for details.

FIGURE 8. A. Isochronal activation map during pacing from the right ventricular free wall. In the lower right-hand corner of the diagram, isochrones are angled so that propagation was occurring at approximately 45 degrees to the longitudinal axis of the guiding plaque. As propagation proceeded from right to left over the plaque, activation direction changed somewhat so that the isochrone directions become more vertical. B. Example of a vector loop recorded at the site indicated by the circle in A. The vector loop is shown in the upper portion of the panel and the component x and y electrograms in the lower portion. The open arrow indicates the activation time. Instantaneous vectors can be examined at any time along the vector loop and the component electrograms. At 1, which occurred at a time when the wavefront was approaching the recording site and the isochrones were angled, the instantaneous vector was pointing at an angle that would be designated approximately -110 degrees, the angle indicated by the arrow labeled 1. Later in the course of local activation, the instantaneous vector direction, as indicated by the arrow labeled 2, shifted to a more superior orientation. Thus instantaneous vectors at different portions of the vector loop represent the direction of activation at different times during propagation.
initially proposed that the moving cardiac impulse could be approximated by a dipole and analyzed the resultant changes in the extracellular electrogram on this basis. Spach et al. analyzed extracellular potentials in the canine Purkinje system and demonstrated that while the waveforms were not simply proportional to the second derivative of the intracellular potential, they could be accurately predicted by means of the spatial distribution of the intracellular potentials. The theoretical analysis of extracellular potentials in anisotropic cardiac muscle is more complex, but recent studies have been able to also accurately predict extracellular potentials in anisotropic muscle by means of intracellular potentials and a consideration of both extracellular and intracellular anisotropy. Although complexities in the extracellular potential distribution generated by the moving wavefront do occur, the largest component appears as a simple dipole at the interface between active and inactive tissue. By summing vectors from two orthogonally placed bipolar electrodes, the direction of this traveling dipole should theoretically be measurable. However, when an impulse is distant from a recording electrode, a potential distribution opposite of that which occurs when the impulse is in the region of the recording dipole may be present at the recording site. Thus potential distributions that occur early or late in time in the vector loop may be less accurate in determining directions of activation.

**Electrogram characteristics.** Component bipolar electrograms recorded using the vector probe differed from those we and others have previously reported in epicardial mapping using conventional electrodes. Electrograms were of far greater amplitude (36 mV) and were less often clearly biphasic. Electrode size, interelectrode distance, position, and filtering all will affect the characteristics of recorded electrograms.

Using a wide bipolar probe with unfiltered recordings, Van der Ark and Reynolds recorded electrograms with similar morphology and even higher amplitude than ours and demonstrated how decreasing interelectrode distance decreases electrogram amplitude.

**Validation of the method.** We have found that a vector loop generated by a pair of orthogonally placed bipolar electrodes accurately represents direction of propagation in epicardial tissue. The median difference between the direction of activation obtained from isochronal maps and that identified by the vector technique was 13 degrees and in only 3% of sites was there a greater than 60 degree divergence between the two methods. Vectors were obtainable even from areas containing epicardial fat or blood vessels.

Discussion

**Electrophysiologic basis of vector loops.** Potentials recorded from the extracellular space in cardiac tissue should in theory be related to and predicted by a knowledge of the changes in intracellular potentials and the geometry of cardiac muscle. Durrer et al. propose that the moving cardiac impulse could be approximated by a dipole and analyzed the resultant changes in the extracellular electrogram on this basis. Spach et al. analyzed extracellular potentials in the canine Purkinje system and demonstrated that while the waveforms were not simply proportional to the second derivative of the intracellular potential, they could be accurately predicted by means of the spatial distribution of the intracellular potentials. The theoretical analysis of extracellular potentials in anisotropic cardiac muscle is more complex, but recent studies have been able to also accurately predict extracellular potentials in anisotropic muscle by means of intracellular potentials and a consideration of both extracellular and intracellular anisotropy. Although complexities in the extracellular potential distribution generated by the moving wavefront do occur, the largest component appears as a simple dipole at the interface between active and inactive tissue. By summing vectors from two orthogonally placed bipolar electrodes, the direction of this traveling dipole should theoretically be measurable. However, when an impulse is distant from a recording electrode, a potential distribution opposite of that which occurs when the impulse is in the region of the recording dipole may be present at the recording site. Thus potential distributions that occur early or late in time in the vector loop may be less accurate in determining directions of activation.

**Electrogram characteristics.** Component bipolar electrograms recorded using the vector probe differed from those we and others have previously reported in epicardial mapping using conventional electrodes. Electrograms were of far greater amplitude (36 mV) and were less often clearly biphasic. Electrode size, interelectrode distance, position, and filtering all will affect the characteristics of recorded electrograms.

Using a wide bipolar probe with unfiltered recordings, Van der Ark and Reynolds recorded electrograms with similar morphology and even higher amplitude than ours and demonstrated how decreasing interelectrode distance decreases electrogram amplitude.

**Validation of the method.** We have found that a vector loop generated by a pair of orthogonally placed bipolar electrodes accurately represents direction of propagation in epicardial tissue. The median difference between the direction of activation obtained from isochronal maps and that identified by the vector technique was 13 degrees and in only 3% of sites was there a greater than 60 degree divergence between the two methods. Vectors were obtainable even from areas containing epicardial fat or blood vessels.
In infarcted areas where isochronal mapping suggested a clear uniform direction of impulse spread, vector loops accurately reflect the direction of such spread. A clear maximum \(x,y\) deflection of the loop and thus a representation of the “major” direction of impulse spread in a given region was obtainable from almost all infarcted sides (98%) even when electrical activity was markedly abnormal and fragmented (figures 10 through 13).

**Patterns of ventricular activation during ventricular pacing in normal myocardium.** During ventricular pacing, a variable area of epicardial tissue was activated in a homogeneous way, suggesting muscle-to-muscle spread. Homogeneous activation depended on the direction of impulse propagation relative to fiber orientation and to a lesser extent on whether propagation was taking place in the left vs the right ventricle. When impulses were recorded during propagation longitudinal to fiber orientation, large areas of homogeneous activation with conduction velocities ranging from 0.5 to 0.8 m/sec was noted. These results are similar to those obtained in vitro in two-dimensional sheets. Activation tended to be more uniform and homogeneous over the surface of the left ventricle than the right ventricle, potentially because of either a more uniform fiber orientation or a thicker myocardial wall and thus less His-Purkinje activation. When propagation occurred transverse to fiber orientation, homogeneous conduction occurred in only relatively small areas with multiple sites of nearly simultaneous epicardial breakthrough occurring in other regions of the mapping area and again resulting in multiple collisions. Vector amplitude was higher during longitudinal than during transverse conduction. Spach et al.\(^{17}\) also have demonstrated differences in extracellular potentials recorded during longitudinal and transverse conduction, with the amplitude of extracellular potentials tending to be higher during longitudinal conduction and the morphology of the electrograms more biphasic. Thus our finding of higher vector amplitudes during longitudinal conduction is easily explicable on the basis of two unipolar electrograms making up a bipolar component that contributes to the vector loop.

**Characteristics of vector loops.** The vector loop represents a sum of instantaneous \(x\) and \(y\) vectors and thus additional information is contained in the morphology and time distribution of points in the loop. As already noted, deflections occurring very early or very late in time in a loop represent patterns of extracellular current flow generated by the active wavefront while it is distant from the recording site, and thus may not contain information about the direction of impulse propagation. Major deflections in the vector loop reflect activity between 2 and 4 mm on each side of the center of the probe. Thus instantaneous vectors generated from the loop when the wavefront is in the area of the recording electrodes should represent the direction of propagation at each instant. We have been able to show that when the direction of excitation is changing in the area around the recording electrode site, different portions of the vector loop recorded at different times accurately represent the direction of activation at these times. This time-dependent change in the instantaneous vector may generate an open loop mor-
FIGURE 11. Representative component electrograms and vector loops from the isochronal activation maps shown in figure 10. In each panel, x and y electrograms and the resultant vector loop for the lettered sites in figure 10 are shown. The open arrow indicates the activation time chosen for each site in these unfiltered wide unipolar recordings. Although multiple areas of slow activity were present, relatively reproducible rapid deflections were obtainable from these sites and this was taken as the activation time. Time and amplitude calibrations for both the vector loops and component electrograms are shown in each panel. The length of the thick line at the right side of the figure represents the amplitude calibration, thus the electrograms in panels B through E are far smaller in amplitude than the electrograms in panel A. The electrogram in panel A represents a recording from the normal right ventricle. Panels B through F are recordings from areas of myocardial infarction. Note that although electrograms have markedly reduced amplitude and prolonged duration, a clear direction of maximum x,y vector impulse is present.

Examples of the criteria used for determining the maximum x,y deflection can be seen by examining panels E and F. In panel F the vector loop appears as a two-component open vector loop. Since the amplitude of both areas of this loop are approximately equal as measured from the origin, the vector direction was taken as bisecting these two areas. In panel E, although two directions differing by about 30 to 45 degrees are also present, the amplitude of one of the directions is less than 75% of the amplitude of the maximum x,y deflection, and thus the maximum x,y deflection was taken as the vector direction. In panel F, the different portions of the vector loop represent different directions of epicardial impulse propagation at different times. Early in the vector loop corresponding approximately to the 35 to 40 msec isochrone in figure 10, the vector direction corresponds to the superior portion of the depicted vector loop and is approximately perpendicular to the 35 and 40 msec isochrones in figure 10. Later in the vector loop, activation direction is proceeding almost directly downward.

Electrograms and vectors in infarcted areas were of lower amplitude and longer duration. Many vectors from infarcted areas were far more complex, including bends and areas of widely disparate directions, suggesting that localized areas had impulse propagation occurring in a direction other than the major wavefront motion. Previous studies of electrograms recorded from canine myocardial infarction have suggested the possibility that different electrogram components represent groups of cells that are being activated at different times. Thus the finding of changing instantaneous vector directions at different times in areas of abnormal electrograms suggests that in some infarcted regions wavefronts are spreading in several directions at different times in a localized area. These may represent dead-end pathways or a manifestation of an extremely irregular pathway of primary impulse conduction. The relatively large distance between mapping sites in the present studies precludes definitive resolution of these
activation directions but demonstrates the potential utility of high-density vector mapping to clarify new details about conduction in abnormal areas. Vector loops therefore may provide far more information than

![Diagram](image)

**FIGURE 12.** Vector and isochronal activation maps of induced VT with a cycle length of 165 msec from a dog with a 2-week-old myocardial infarction. In panel A, the vector map from this tachycardia is shown alone. If sites on the right and left superior-inferior borders of the guiding plaque are examined, the origin of impulse propagation is clearly directed toward the interior of the guiding plaque. The arrows pointing toward each other along the dashed line in the center of the diagram represent a collision as previously shown in figure 9. By tracing back these arrows, one can clearly see an area of epicardial origin or breakthrough of the tachycardia that is located at the circled site. An area of block, indicated by the solid line, was present. It is impossible with the resolution of mapping utilized in this experiment to be certain whether a small area in which an impulse reenters through this area of block indicates a macroreentrant circuit on the epicardial surface. In any case, vector directions not only point away from the area of initial epicardial activity but also provide information about the details of areas of block and collision. In panel B, an isochronal activation map from the same tachycardia is shown. In general, there is good agreement between the directions of epicardial activation as determined by isochronal activation mapping and those determined by vector mapping. In some areas, where isochrones are widely spaced, a clear isochronal activation direction cannot be generated and here the vectors as shown in A indicate areas of collision.

is available from a simple consideration of activation times.

Although in the present series of experiments we were able to record characterizeable vector loops from 98% of infarcted sites, certain areas of dense infarction may be associated with no local electrical activity and thus may be unsuitable for analysis with any mapping technique.

**Mapping of VT and further applications.** Isochronal mapping of ventricular arrhythmias has provided a great deal of information about the pathophysiology and anatomic substrate of arrhythmias in both experi-

![Diagram](image)

**FIGURE 13.** Examples of representative electrograms from lettered sites in figure 12. Tracings of electrograms in this figure were obtained from recordings on a Gould electrostatic recorder, whereas vector loops were obtained from 35 mm film. Thus the organization of this figure is slightly different than that of previous figures. In each panel, y and x component bipolar electrograms and resultant vector loops are presented for lettered sites. Also the electrocardiogram (ECG) and reference electrogram (REF) are shown. Amplitude and time calibration factors are shown for each panel. Despite the presence of low-amplitude, prolonged electrograms in the area of myocardial infarction, a clear direction of maximum x, y deflection of the vector loop is present in all cases. These correspond well with the directions of activation shown by isochronal mapping in figure 12.
mental and clinical situations. 1-6, 8, 21-23 Limitations of the techniques do exist. When standard single-electrode mapping techniques are used, a given site is classified as early or late in relation to the surface QRS. If an impulse is not “early,” it is unclear what direction the impulse is emanating from and one can only state that it is not near the site of origin. In addition, mid-diastolic potentials may be difficult to classify as either “early” or “late” in relationship to the tachycardia. Such limitations have led to some difficulties in intraoperative mapping of clinical tachycardias. For example, it may be occasionally difficult to distinguish microentrant from macroentrant circuits with a limited number of recording sites. 21, 22

Vector mapping has a potential for obviating some of these disadvantages of isochronal mapping. We have found in experimental VT that a vector direction showing the general area of the myocardium from which an impulse is originating may speed single-point mapping by directing further recordings toward the tachycardia origin. Vector mapping also may help to distinguish “early” from “late” sites by showing that impulses in other nearby regions do point away from a mid-diastolic site. This allows classifying the site as being unrelated to the tachycardia origin. In addition, by determining whether an impulse is moving toward or away from a site where a tachycardia may originate, it may be possible to more clearly identify whether such areas are part of a macroentrant loop or simply being activated from a small focus. Higher density vector mapping may allow the details of even small circuits to be elucidated.

In addition, vector mapping may provide information not available even with multiple-site simultaneous isochronal mapping. For example, vector mapping may allow the differentiation between slow conduction and unidirectional block and may allow analysis of complicated activation patterns in abnormal areas as previously described. In any case, these techniques may provide complementary information in a variety of experimental and clinical situations.

Computerized multielectrode systems now in use in both clinical and experimental laboratories have addressed some of the problems with conventional mapping cited above. 1, 2, 6, 23 However, such systems do require substantial investments of time and equipment and thus may not be as widely applicable as the combination of vector mapping with standard single-point activation mapping techniques.

We thank Ralph Iannuzzi and William Moore for their expert technical assistance and Bejay Moore for typing the manuscript.

References

Vector mapping of myocardial activation.
A H Kadish, J F Spear, J H Levine, R F Hanich, C Prood and E N Moore

Circulation. 1986;74:603-615
doi: 10.1161/01.CIR.74.3.603

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/74/3/603