Potential Fields on the Ventricular Surface of the Exposed Dog Heart during Normal Excitation

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SUMMARY. We studied the normal spread of excitation on the anterior and posterior ventricular surface of open-chest dogs by recording unipolar electrograms from an array of 1124 electrodes spaced 2 mm apart. The array had the shape of the ventricular surface of the heart. The electrograms were processed by a computer and displayed as epicardial equipotential maps at 1-msec intervals. Isochrone maps also were drawn. Several new features of epicardial potential fields were identified: (1) a high number of breakthrough points; (2) the topography, apparent widths, velocities of the wavefronts and the related potential drop; (3) the topography of positive potential peaks in relation to the wavefronts. Fifteen to 24 breakthrough points were located on the anterior, and 10 to 13 on the posterior ventricular surface. Some were in previously described locations and many others in new locations. Specifically, 3 to 5 breakthrough points appeared close to the atrioventricular groove on the anterior right ventricle and 2 to 4 on the posterior heart aspect; these basal breakthrough points appeared when a large portion of ventricular surface was still unexcited. Due to the presence of numerous breakthrough points on the anterior and posterior aspect of the heart which had not previously been described, the spread of excitation on the ventricular surface was “mosaic-like,” with activation wavefronts spreading in all directions, rather than radially from the two breakthrough points, as traditionally described. The positive potential peaks which lay ahead of the expanding wavefronts moved along preferential directions which were probably related to the myocardial fiber direction. (Circ Res 52: 706–715, 1983)

The spread of excitation on the surface of the canine heart has been studied extensively in past years by means of multiple unipolar or bipolar epicardial leads. According to the earlier descriptions (Lewis and Rothschild, 1915; Harris, 1941; Schaefer and Trautwein, 1949; Sodi-Pallares and Calder, 1956), excitation at the dog’s ventricular surface spread from a right anterior ventricular breakthrough point (BKTP) in the mid-paraseptal area, which appeared 5–10 msec after the beginning of ventricular excitation. From this region, a single wavefront spread over the right and left anterior ventricular surface. A second breakthrough occurred about 10 msec later, close to the left ventricular apex. Epicardial activation spread then radially from two main ventricular breakthroughs. Some have reported a few additional accessory BKTPs on the right and left ventricle (Durrer and Vander Tweel, 1957; Taccardi, 1958, 1960; Kawasuji and Tagashi, 1978). The above-mentioned studies used isochrone maps, but a more recent approach has used isopotential contour maps. This approach has certain advantages that have been discussed at length by Spach et al. (1969) and Spach and Barr (1975). The method is more satisfactory from a biophysical point of view, since it provides a representation of the actual electrical events associated with excitation, i.e., the distribution of heart potential on the epicardial surface.

In all the studies published so far, the distance between points explored on the ventricular surface was about 1 cm or more, considerably larger than the wavefront width, which varies from 1 to 3 mm (Vander Ark and Reynolds, 1970; Van Oosterom and Van Dam, 1976). Therefore, the representation of isochrones and equipotential lines required extensive interpolation to cover the comparatively large unexplored areas, with the risk of joining epicardial points, excited at the same time, by isochrones even if they were not activated by the same wavefront. Similarly, interpolated equipotential lines may connect points that have the same instantaneous potential value, but may, in fact, belong to different families of “real” equipotential lines (Fig. 1).

In the present study we used a large electrode array with 1124 electrodes placed at 2-mm intervals to define ventricular activation sequence in greater detail than previously reported.

Methods

Twelve mongrel dogs weighing 14–24 kg were anesthetized with sodium pentobarbital (30 mg/kg, iv). Under artificial respiration, the heart was exposed by means of a mid-ster nal thoracotomy and cradled in the pericardium.

Electrodes

An array of electrodes was constructed by securing 1124 silver wires, 0.05 mm in diameter, to a thin nylon cloth. The electrodes were arranged in a matrix with the shape of the ventricular surface of the heart, with an interelectrode distance of 2 mm along rows and columns. Previous studies
**FIGURE 1.** Potential distribution on a limited portion (about 6 cm²) of the anterior aspect of the right ventricle, 11 msec after the beginning of ventricular activation. Stippled areas indicate regions of negative potentials. In part A, cardiac potentials were measured at epicardial points spaced 2 mm apart along rows and columns. Potential distribution in part B was obtained by considering only the potential values at epicardial points spaced 6 mm apart. The potential values are in millivolts; the equipotential lines are drawn every 5 mV. Although two breakthrough points are still present, other details are missing: (1) the densely packed equipotential lines associated with the epicardial wavefronts; (2) the three positive potential peaks located close to two excited areas (15, 10, and 15 mV in part A); (3) the depression (region completely surrounded by higher potentials) indicated by the arrow ([ ]) in A (5 mV).}

from our laboratory showed that potential patterns determined from electrodes spaced at 1-mm intervals had the same features (Stilli et al., 1978; Arisi et al., 1979). The electrode array was first placed on the anterior ventricular surface and, in four experiments, was also used to explore the posterior surface. The electrode array was moist and adhered to the epicardium. It followed the heart movements without sutures. During the entire procedure of data acquisition, the electrode array was kept moist with Ringer’s solution so that a very thin layer of fluid covered the heart surface.

**Data Acquisition and Processing**

Groups of 120 epicardial electrodes were simultaneously connected to the non-inverting inputs of differential amplifiers (Cottini et al., 1972). The second input of all amplifiers was connected to a common reference point placed on the dog’s hindleg. The amplified signals were multiplexed and converted into digital form on-line. The sampling rate was 1000 Hz per channel, with an overall sampling rate of 120 kHz. The resolution of the converting procedure was 100 µV for signals lower than 3 mV, and varied progressively for higher signals. The digital data were conveyed to a PDP 11/40 minicomputer and stored on disk or tape. This procedure was repeated until all 1124 unipolar electrograms were recorded. An electrogram from one electrode was recorded throughout the entire procedure for time reference. The acquisition of 1124 electrograms required 20-30 minutes. All the signals were recorded in the expiratory pause.

The electrograms were plotted by the computer on a diagram of the heart and electrode array (Fig. 2). Then the instantaneous potential values were printed on tables which also had the format of the electrode array, and epicardial isopotential maps with 1-msec intervals were manually constructed; in addition, a map of isochrones was drawn. Epicardial wavefronts were identified in the regions of densely packed equipotential lines in instantaneous maps; the isochrones were drawn inside these regions along the zero line which separate positive from negative areas. Isochrones drawn with this procedure were similar to those obtained by computing the maximum negative derivative of the unipolar electrograms. The spread of excitation was illustrated on diagrams of the heart. The successive groups of 120 electrograms were time-aligned with parts m, the epicardial wavefronts moved 0.33 to 1.2 mm; therefore the maximum theoretical error was less than the space resolution of our electrode array (2 mm). In every experiment, some epicardial areas were explored a second time, 10 minutes or more after the first recording. Comparing the two recordings revealed a good reproducibility of the data.

**Results**

**Definition of Terms**

The following terms were used to describe some map features which can be seen in Figure 3. "Mountain"—an area where the potential values progressively increase from the outer to the inner regions. A mountain contains one or more peaks. "Peak"—a limited region or a point where the potential is higher than that measured at the surrounding points. "Saddle"—a region of lower potentials between contiguous mountains or peaks. "Niche"—an inlet or cavity on the slopes of mountains. "Depressions" and "basins"—regions completely surrounded by higher potentials. Basins generally contained one or more sites where the potential values were lower than those measured at surrounding sites. "Escarpment"—a steep slope surrounding a basin.

**Equipotential or Isochrone Patterns**

The general time-course and spatial distribution of the electrical events on the epicardium were similar in all experiments, although the details of the activa-
FIGURE 2. Electrograms of QRS recorded from the 1124 electrodes on the ventral aspect of the ventricles in one experiment. In this experiment, there were nine missing or poor electrograms (marked by •). These waveforms were replaced by the mean of the signals recorded by the surrounding 2 to 4 electrodes. In all experiments, there were 6 to 12 missing electrograms.

Anterior or Ventral Aspect of the Right Ventricle

During the initial phases of ventricular excitation (1–6 msec) a potential mountain, exhibiting 1–3 peaks, was usually observed on the anterior right ventricular surface (Fig. 3A). The highest peak ranged between 15 and 25 mV; saddles were present between the peaks. Usually, one peak was located in the central portion of the ventral aspect of the ventricle, close to the region where excitation emerged at the surface in the following instants (Fig. 3A, 15 mV). Another peak was generally located close to the right border of the area explored (Fig. 3A, 10 mV). In one case, only the right peak was observed. When present, the third peak was located between the two main peaks. Sometimes a depression with potential values of about —2 mV was present in the region close to the atrioventricular (AV) groove or the pulmonary conus (Fig. 3A, stippled area).

Between 7 and 13 msec from the beginning of ventricular activity, one or more depressions appeared above or to the right of the central portion of the anterior descending coronary artery. These depressions were located close to the central peak on the slopes of the mountain (Fig. 3B), where they excavated a niche. The potential values in the depressions reached —15 to —25 mV in 2 or 3 msec (Fig. 3, B–D). We believe this event to be the expression of an excitation wavefront emerging at the right ventricular surface. These epicardial areas, where excitation wavefronts appear earlier than at surrounding points, are commonly called breakthrough points (BKTPs). The resulting basins were contoured by more-or-less steep escarpments with a potential jump of 35–45 mV.
apparent velocities and traveled 2 mm or more before along a line which ran nearly parallel to the anterior were separated 2-8 mm and were constantly located at the right ventricular surface, 4-6 BKTPs constantly underlying excitation wavefront. Considered to be the electrical manifestation of an potential lines could be traced. The escarpment was gated, roughly elliptical shape, with the major axis generally appeared a few mm from the AV groove. The new wavefronts spread in all coming from the source region was 1 or 2 cm from the These BKTPs often appeared when the wavefronts colliding with another wavefront. The merging of the paraseptal excited areas gave rise to a large basin (Fig. 3E). The sequence of events leading to the appearance of this basin was completed in 4-7 msec. At the end of the first third of the QRS, this area had an elongated, roughly elliptical shape, with the major axis parallel to the anterior descending coronary artery. This large negative region was in the same location as the “source region” observed by Schaefer (1957). Thus, the spread of excitation on the right ventricular surface, often described as a single wavefront surfacing at one point and then spreading, actually had a much more complicated sequence. The elongated excited area was surrounded by positive regions with up to six peaks (Fig. 3D). The peaks were generally located ahead of the long side of the expanding wavefront and constantly preceded their propagation toward the base and the apex of the heart. They were never observed at the short ends of the elliptical wavefront. In the following instants, additional BKTPs appeared on the right lateral border of the heart (Fig. 3, E and F) and to a lesser degree on the left (Fig. 4, BKTP at 21 msec). These BKTPs generally were 2-10 mm away from the main wavefront. During the second half of ventricular excitation, three to five new BKTPs appeared in the basal region of the heart (Fig. 3, G-I). The earliest basal BKTP generally appeared a few mm from the AV groove near the lateral wall of the right ventricle. Later, new BKTPs occurred in the proximity of the AV groove and on the pulmonary conus. In these areas, the arrival of excitation was often preceded by one or more peaks of positive potential (not presented in the figures). All these BKTPs appeared in 5–8 msec at a distance of a few millimeters (Fig. 3, G-I, Fig. 5 at 19 msec) to 20 mm (Fig. 5 at 27 msec) from one another. These BKTPs often appeared when the wavefront coming from the source region was 1 or 2 cm from the AV groove. The new wavefronts spread in all directions on the heart surface; thus, in a few milliseconds, the excitatory process covered comparatively large areas close to the AV ring, while much of the right ventricular surface was still unexcited (Fig. 3, H, I, L). Some potential peaks developed near the wavefronts bordering the newly excited areas, as had previously occurred around the main source region (Fig. 3, G-I). The activation of the ventral aspect of the right ventricular surface was generally completed through the merging of the wavefronts coming from the basal region with the excitation wave spreading from the source region. The fusion of the two main excited areas was not instantaneous, but developed progressively along a line as long as 40 mm (Fig. 3, I and L). The potential peaks which bordered the wavefronts decreased in amplitude and disappeared before the wavefronts collided. The location and time of appearance of BKTPs on the anterior ventricular surface, as they occurred in the experiment relating to Figure 3, are presented in Figure 4. Ventral or Anterior Aspect of the Left Ventricle During the early stages of ventricular activation, a depression with potential values of about +1 or −1 mV often was observed on the apex. Generally, between 8 and 13 msec after the beginning of ventricular activation, a peak appeared on the left ventricle at the apex; in the case shown in Figure 3C, the peak appeared at 13 msec. In the following instants, the peak migrated progressively toward the right lateral border of the heart, while its potential value increased from 15 to 23 mV (Fig. 3, D, E, △). Between 16 and 23 msec, an activation wavefront reached the left anterior ventricular surface either as a BKTP or as a wavefront coming from the border of the area explored. The earliest BKTP was generally in the vicinity of the peak described above (Fig. 3F). Another early BKTP often was observed in the left part of the paraseptal region, 10 mm or less from the main trunk of the anterior descending coronary artery; it appeared later, at 30 msec, in the experiment of Figure 3H. In Figure 5, at 21 msec, two BKTPs are present in this area. The time-course of later stages of excitation on the left anterior ventricle exhibited considerable variability in different dogs. The following patterns were observed:

1. A broad wavefront, emerging from the apical region and from the paraseptal area close to the left margin of the heart, spread toward the main wavefront coming from the right ventricle. The left and right wavefronts collided along a line roughly parallel to the anterior descending coronary branch and as long as 50 mm (this pattern is not illustrated).

2. More often, a few accessory BKTPs appeared in the area between the advancing main wavefronts (Fig. 5, instants 21 and 23).

3. In some cases, the number and scattering of the accessory BKTPs was so considerable, that a "mosaic" pattern of excitation ensued. An example of this is shown in Figure 3. An epicardial wavefront spread from the apical region toward the paraseptal area, covering a distance of about 10 mm (Fig. 3, F and G). A potential peak preceded this wavefront. Meanwhile, a number of BKTPs appeared in the area located below the anterior descending coronary artery and near the left heart border; these new wavefronts...
FIGURE 3. Distribution of equipotential lines on the ventral aspect of the heart at 10 time instants (parts A to L), indicated by the vertical lines intersecting the electrograms at the lower left of each map, and by the numbers under the electrograms (in milliseconds from the onset of QRS). The dashed area indicates fatty tissue from which no data are available; negative areas are stippled. The potential values are in millivolts. The equipotential lines are drawn every 2.5 or 5 mV, dotted lines indicate the zero line. Symbols: ↓ = depression preceding the appearance of breakthrough point; ▲ and ▼ (in the positive and negative areas respectively) = points where the absolute value of measured potential is 1–4 mV greater than that of the closest equipotential line. Part A: in the initial phases of ventricular activation, the potential field was simple—two peaks in the mountain and a large depression with negative potential values (stippled area) were present on the right ventricle. Part B: the anterior ventricular surface showed positive potentials; two depressions (arrows) and two peaks of 20 and 22 (▲) mV were present. Part C: a more complex potential field was present: two breakthrough points (stippled areas) and two new depressions appeared. Moreover, four peaks were adjacent to these BKTPs and depressions; there was another peak (15 mV) at the apex and a mountain (10 mV) close to the AV groove. Part D: two epicardial activation wavefronts were revealed by the densely packed equipotential lines on the paraseptal area of the right ventricle. Three new BKTPs and six positive peaks were present in the same region. Part E: an elongated region was activated on the right paraseptal area. This area was in the same location as the "source region" described by Schaefer. Six positive peaks lay ahead of the expanding wavefronts; another peak was on the left ventricle close to the region where the activation wavefront appeared in subsequent instants. Part F: the main wavefront spread from the source region in all directions while a BKTP appeared close to the AV ring (−3 mV); in this instant, the greatest apparent wavefront width was about 1 cm (right side of the figure). A wavefront emerged on the heart apex. Part G: a more complex activation pattern was present. On the right ventricle, two BKTPs appeared close to the AV groove, and there was a depression on the pulmonary conus, 1.4 cm from the main wavefront. On the left ventricle, three new BKTPs appeared, and there was a depression in the left paraseptal region a few millimeters from the anterior descending coronary artery. Part H: in the right basal region, a new depression appeared; several peaks were present close to the separate basins of activated tissue. In the left ventricle, a depression appeared in the central portion of the paraseptal area. Part I: a long portion of wavefront traveled from the base of the heart toward the apex, another wave spread on the pulmonary infundibulum; on the left ventricle, one BKTP and three separate basins were present. Part J: on the left ventricle, two basins merged with the main basin.
spread over a distance of a few millimeters and met the excitation waves coming from the apex. The epicardial activation of this region was usually completed through collisions of wavefronts from the right ventricle with wavefronts spreading from the heart apex and from the accessory BKTPs (Fig. 3, H, I, L).

**Posterior or Dorsal Aspect of the Heart: Right and Left Ventricles**

We described here the general features of the potential patterns on the posterior ventricular surface. The equipotential contour maps are not presented, but a typical isochrone map is illustrated in Figure 6.

During the early stages of ventricular activation, we often observed a large depression with negative potential values of about -1 mV, on the left posterior epicardial surface, while positive potential values were constantly present on most of the posterior right ventricle. In the following instants, the negative area moved to the base and then disappeared. A mountain with prominent peak (up to 30 mV) developed in the apical region, where an early left ventricular BKTP soon appeared.

Between 21 and 27 msec after the beginning of ventricular activation, two excitation waves emerged in the vicinity of the apex, usually first on the left ventricle and then, a few milliseconds later, on the right. Thereafter, several new BKTPs appeared on the lateral borders and central area of the posterior ventricular surface at a distance of 2 to 6 mm from the main wavefront (Fig. 6, at 31, 33, and 35 msec). Between 37 and 41 msec after the beginning of ventricular activation, two to four BKTPs often appeared on both ventricles a few millimeters from the AV groove. A number of peaks of positive potential constantly bordered the propagating wavefronts and disappeared just before their collisions, as occurred on the ventral aspect of the heart. On the posterior heart...
surface there was a general progression of excitation from apex to base, but in many areas surrounding the BKTPs excitation, waves moved in all directions, including base to apex (Fig. 6).

Figure 7 shows the location and times of appearance of BKTPs found in the same experiment illustrated in Figure 6. At least 10 BKTPs were observed on the dorsal surface of the heart in all experiments.

**General Features of Excitation of the Ventricles**

The details of the activation patterns described above showed considerable variability in the various dogs; however, the following features were constantly observed in all the experiments: numerous BKTPs, 15 to 24 on the anterior and 10 to 13 on the posterior aspect of the heart; numerous early BKTPs in the source region on the right ventricle and a number of BKTPs in the basal region of the ventricles, prior to the excitation of a large portion of the ventricular surface; the occurrence of numerous collisions; the presence of peaks ahead of the long axis of the expanding wavefronts.

**Quantitative Aspects of the Potential Distribution**

Negative potential values ranging from $-10$ to $-62$ mV were recorded in the excited areas. The breakthrough points remained the seat of the potential minima for several msec. The potential peaks ranged between $+15$ and $+50$ mV.

The apparent width of the wavefronts was difficult to measure because it was ill-defined. The escarpments surrounding the excited areas, where the equipotential lines were most densely packed, were 2 to 4 mm wide. In some cases, on limited portions of the wavefront, the equipotential lines were less densely packed and the apparent width of the wavefront reached as much as 10 mm.

On the ventral surface of the ventricles, the potential drop across the wavefront ranged between 20 and 76 mV, and the potential gradient varied between 2 and 38 mV/mm. On the posterior heart surface, the potential drop ranged between 15 and 48 mV, and the potential gradient varied between 1.5 and 24 mV/mm.

The spread of the epicardial wavefront had an apparent velocity ranging between 33 and 120 cm/sec. These figures were obtained by measuring the distance the wavefront traveled in a direction normal to its tangent in a time unit. More than 40 such measurements were performed.

**Discussion**

Exploration of the dog's ventricular surface by means of an electrode array with 1124 closely spaced terminals and the presentation of data in the form of equipotential contour maps enabled us to describe the epicardial electric field in more detail than previously reported. In particular we collected new information regarding: (1) the number, location, and timing of "breakthrough" events, i.e., the arrival of excitation at the surface of the ventricles; (2) the topography of potential maxima and minima in relation to the topography of the wavefronts; (3) the topography and apparent width of the excitation wavefronts on the ventricular surface; (4) the approximate value of the potential drop across these wavefronts; (5) the apparent velocities and direction of propagation of these excitation waves; and (6) the collision of wavefronts and merging of excited areas.

**Breakthrough Points**

In agreement with many previous reports, our data confirmed the localization of two main breakthrough points in the right paraseptal area and near the apex on the left ventricle. However, in each experiment, 23 to 32 additional BKTPs were seen. These were scattered on both right and left ventricular surfaces. Thus, the spread of excitation on the epicardium was not a continuous propagation of wavefronts emerging at a few sites on the surface, but rather a "mosaic-like" process where wavefronts emerged at many sites on the epicardium and spread over limited areas before colliding with other wavefronts moving in different directions.

The occurrence of many breakthrough points on the right and left ventricular surfaces probably resulted from bulges in wavefronts advancing from deeper layers of the ventricular walls. The most likely reasons for these bulges are that excitation wavefronts start from the endocardial surface which is not planar, but has many hollows and grooves, and that junctions between the Purkinje fibers and heart muscle are discontinuous. Both factors would be expected to generate bulges in wavefronts as they proceed from endocardium to epicardium.

It is difficult to explain why a number of BKTPs
invariably appeared in the basal portion of the right ventricular surface, close to the AV groove. Myerburg et al. (1972) reported that there is no Purkinje tissue in this portion of the endocardium. Even if there were Purkinje fibers in this region, it would be difficult to explain why excitation reached these epicardial regions prior to activation of much of the right ventricle (Fig. 3G). It is possible that some Purkinje strands run for comparatively long distances along the endocardium without establishing junctions with the underlying contractile myocardium. Another possibility might be that activation in these areas originates from vestigial AV bypass tracts. The latter possibility seems unlikely, however, because these BKTPs were so late in the activation sequence.

Potential Maxima

The topography of potential maxima during normal ventricular activation deserves some comment. According to traditional models, the excitation wavefront is considered as a uniform dipole layer. One would therefore expect to find potential maxima where the wavefront is closest to the surface, i.e., at the breakthrough points. This, however, did not occur. The potential maxima were located not too far from the areas where the breakthrough appeared, but they were not exactly at the breakthrough points. When extensive wavefronts spread over the ventricular surface, they were often preceded by peaks of positive potentials. The peaks generally were close to the long side of the elongated, excited area that appeared on the right ventricular epicardium 12–16 msec after the onset of ventricular excitation (Fig. 3E). During subsequent instants, the peaks moved preferentially along the apex-to-base axis. The peaks generally were not observed along the narrow ends of elliptical or elongated wavefronts. We feel that such behavior may be related to the effect of myocardial anisotropy on the potential field (Corbin and Scher, 1977; Baruffi et al., 1978; Roberts et al., 1979; Colli-Franzone et al., 1982). According to recent observations, potential maxima precede a spreading wavefront only where the front is moving along the main fiber direction.

Epidermal Wavefronts

The main purpose of this study was to collect new detailed information on the spread of excitation, and the exact location and timing of breakthrough points on the canine epicardium during normal activation. In addition, however, an attempt was made to interpret other features of the potential field. Our experimental setup enabled us to determine the topography of superficial wavefronts from the presence of densely packed equipotential lines at a number of time instants during ventricular excitation. The real potential drop across a wavefront could not be determined accurately from epicardial recordings because the spatial limits of the wavefront were not clearcut. However, the highest potential drop we measured over a distance of 2 mm was 76 mV, and the highest potential jump over 4 mm was 86 mV. These high values occurred mainly when the wavefront was propagating base-to-apex or apex-to-base on the ventral aspect of the heart. Where propagation occurred in a transverse direction (right to left or left to right), the potential drop per unit distance was considerably smaller; potential gradients as low as 2 mV per mm often were observed in these areas, with total potential jumps of only 20 mV. These figures are difficult to compare with previously reported data (Vander Ark and Reynolds, 1970; Roberts et al., 1979; Roberts and Scher, 1982), since our measuring procedure was different from the ones used in those reports. Vander Ark and Reynolds (1970) defined the wavefront voltage as the highest potential difference between two epicardial points 1 mm apart, and found values of 74.1 ± 8.3 mV in open-chest dogs. Roberts et al. (1979) and Roberts and Scher (1982) measured the highest difference between two voltage samples, 2 msec apart, in a unipolar lead. They found 22 ± 11 to 43 ± 18 mV (Roberts et al., 1979) and 43 ± 6 to 74 ± 7 mV (Roberts and Scher, 1982), depending on whether they considered the wavefronts moving across fibers or along fibers. The maximum potential jump we observed over a distance of 2 mm was 76 mV. Since we never found such high potential drops with an array of electrodes spaced 1 mm apart (Stilli et al., 1978) it seems likely that the apparent wavefront dimensions are greater than 1 mm.

Measurements of the wavefronts widths had the same limitations as the measurements of potential drop across the wavefronts just described. We assessed the wavefront apparent width from the width of the escarpment between positive and negative regions. This width is known to vary considerably, depending on whether the wavefront is advancing along the direction of the fibers, or perpendicular to the main fiber direction. In the latter case the spatial potential jump has been reported to be distributed over 3–4 mm (Baruffi et al., 1978; Roberts et al., 1979; Roberts and Scher, 1982), even when the wavefront intersected the epicardial surface at right angles as a result of epicardial pacing. In the experiments described in our study, the angle between the wavefront and the cardiac surface was unknown, but could be expected to be less than 90° during normal endo-epicardial spread of excitation. Therefore, the wavefront would be expected to be generally wider than 3–4 mm. However, we often found wavefronts with an apparent average width of only 2–4 mm.

Our maps allowed us to observe the apparent direction of epicardial waves and to measure the tangential component of the velocity of the wavefronts. Epicardial measurements can be used to determine conduction velocity only when the wavefront is perpendicular to the ventricular surface. This condition was satisfied in the experiments of Baruffi et al. (1978) and Roberts et al. (1979), which yielded similar results during ventricular pacing: \( V_x = 25 \pm 3 \text{ cm/sec} \) for transverse (across fibers) velocity, and \( V_y = 58 \pm 8 \text{ cm/sec} \) for longitudinal (along fibers) velocity. In the experiments described here, apparent conduction ve-
locities are not entirely comparable to those reported previously. Nevertheless, the lowest velocity observed in our experiments (33 cm/sec) was somewhat greater than the transverse velocity previously reported, probably because the plane tangent to the epicardial wavefront was not perpendicular to the heart surface. We observed a wide range (33–120 cm/sec) of apparent conduction velocities, probably due to the variable angles between direction of propagation and fiber orientation, which resulted in various intersection angles between the excitation waves and the ventricular surface.

In conclusion, the results of our study indicate that the epicardial activation sequence has a much more complicated pattern than previously described. Such a detailed description of activation was possible because of the large number of epicardial sites that were sampled with our electrode array. From a biophysical point of view, the instantaneous equipotential contour maps are more satisfactory than isochrones; isopotential maps provide a representation of the actual electrical events associated with excitation, i.e., the potential distribution on the heart surface. Such detailed knowledge of the distribution of epicardial potential is a prerequisite for mathematical models of the heart as a bioelectric generator. Ideally, comparable information about the transmural distribution of potential and the extracardiac electric fields should also be obtained for the purposes of these models. At the present time, such data are not available.

It is probable that similar events occur in human hearts, and the method we have described could be used to define these phenomena in patients during cardiac surgery. Knowledge of such events is important for correlation between epicardial and body surface isopotential maps by both inverse and forward approach to the problem. In addition, knowledge concerning normally occurring sites of early breakthrough is important for interpretation of electrophysiological data acquired in patients during surgery for intractable arrhythmias.

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