Detection and Localization of Prolonged Epicardial Electrograms With 64-Lead Body Surface Signal-Averaged Electrocardiography

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Background. Prolonged, fractionated ventricular electrograms often are detectable after myocardial infarction and are a marker for an arrhythmia-prone state. QRS late potentials detected on the body surface with signal-averaged electrocardiography (SAECG) are thought to arise from the diseased tissue that generates prolonged ventricular electrograms and as such are also a marker for arrhythmias. A limitation of the current SAECG technique is that recordings are obtained from only three bipolar lead pairs. Because late potentials probably arise from multiple small sources in the heart, more extensive sampling of the body surface may contribute additional information to the SAECG. The present study investigates the additional sensitivity of SAECG using 64 body surface leads in detecting prolonged epicardial electrograms and examines its use in determining the epicardial location of prolonged electrograms.

Methods and Results. Dogs were studied before and 5–10 days after either lateral left ventricular (n=13) or right ventricular (n=8) myocardial infarction. Greater prolongation of signal-averaged QRS duration was detected with 64-lead SAECG (postinfarction QRS duration, 100.3±16.3 msec) than with three-lead SAECG (postinfarction QRS duration, 89.4±10.1, p=0.0005). Nineteen of the 21 dogs (90%) had prolonged epicardial electrograms detected over the infarct. The correlation between epicardial electrogram duration and signal-averaged QRS duration calculated from individual leads was much better for 64-lead SAECG (r=0.88, p<0.0001) than for three-lead SAECG (r=0.53, p=0.01), and the difference was most marked in cases with longer electrogram durations (more than 100 msec). Local late potential maxima on the thorax after lateral left ventricular infarction were located to the left and inferior compared with those after right ventricular infarction (p=0.006).

Conclusions. SAECG with more extensive recording from the body surface using 64 leads detects greater QRS prolongation than three-lead SAECG, and the longer QRS durations detected correspond to the duration of prolonged epicardial electrograms. Body surface location of late potentials corresponds to the epicardial location of the prolonged electrograms. This application of body surface mapping techniques to SAECG may permit more sensitive detection of arrhythmia-prone states and may aid in identifying arrhythmia sources. (Circulation 1991;84:871–883)

Prolongation of electrograms recorded directly from ventricular myocardium can frequently be detected after experimental and clinical myocardial infarctions.1–10 Prolonged, fractionated electrograms are thought to be generated by slow and inhomogeneous activation of adjacent muscle fibers, resulting from impairment of cell-to-cell coupling by excess connective tissue.6 These prolonged electrograms are associated with ventricular tachycardia and ventricular fibrillation in both experimental and clinical studies.3–5,7–10 It is possible that tissue generating the prolonged electrograms participates as the slowly conducting limb of a reentrant circuit.11

On the body surface, signal-averaging recording techniques can detect low-amplitude electrocardiograms of Health. R.A.F. is an Established Investigator of the American Heart Association.

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graphic potentials at the end of the QRS complex that are not detectable by standard electrocardiography (ECG).12–16 These electrocardiographic “late potentials” are commonly seen in patients with sustained ventricular tachycardia16–18; in canine infarct models, they are associated with inducible ventricular arrhythmias.19–22

Late potentials are thought to arise from tissue that generates prolonged, fractionated electrograms. Studies in humans and canine infarct models have shown an association between the presence of late potentials and the presence of prolonged electrograms.12,15,19,21,23,24 For example, Spear et al19 showed in a canine infarct model that dogs with inducible ventricular tachycardia generally had both electrocardiographic QRS late potentials and prolonged ventricular epicardial electrograms and that dogs without inducible arrhythmias generally had neither. However, in this and other studies, there were examples of marked discrepancy between signal-averaged QRS duration and the duration of epicardial electrograms; in particular, the signal-averaged QRS frequently underestimated the duration of epicardial electrograms.

A limitation of the current technique of signal-averaged ECG (SAECG) is that recordings are obtained from only three bipolar leads.16 These three leads, approximately orthogonal, would fully characterize a late potential only if the latter were an isolated, single dipolar signal (and the torso a homogeneous conductor). Furthermore, the late potentials’ low amplitude might limit their detectability to circumscribed areas of the precordium. Experience in body surface potential mapping has shown that use of multiple direct precordial leads adds additional diagnostic information concerning regionally selective electrical properties of the myocardium, suggesting that the QRS complex cannot be modeled as a single electrical dipole.25–28

In addition to enhancing the detection of late potential signals, application of body surface mapping techniques to SAECG may provide information about the cardiac location of abnormal ventricular electrograms. Not only is conventional SAECG limited to three leads, but the information from these three leads is usually combined into a single “vector magnitude,”16 and as a result any information localizing the late potential is lost. Localization of the late potential on the body surface could theoretically aid in identifying arrhythmia sources.

The purpose of the present study was to compare the accuracy of a 64-lead body surface SAECG with that of a standard three-lead SAECG in the detection of prolonged ventricular electrograms and to investigate the usefulness of body surface late potential mapping in identifying the cardiac location of abnormal ventricular electrograms.

Methods

Mongrel dogs weighing 20–25 kg were used under a protocol approved by the Institutional Animal Care and Use Committee of the University of Utah. After baseline SAECG, dogs underwent coronary ligation. Five to 10 days later, dogs underwent repeat SAECG and epicardial recordings.

Animal Preparation

All recordings and surgery were performed with the animals under general anesthesia, which was induced with morphine (approximately 0.5 mg/kg) and pentobarbital (approximately 20 mg/kg) administered intravenously and maintained with fluothane (Halothane) administered by inhalation. The depth of anesthesia was maintained to suppress spontaneous movement and the corneal reflex. Dogs were tracheally intubated and ventilated with a Harvard respirator (Harvard Apparatus, South Natick, Mass.). Catheters were inserted percutaneously into the femoral artery and vein. Arterial blood was sampled approximately every 30 minutes, and minute ventilation was adjusted to maintain pH at 7.35–7.45. A bipolar 5F pacing electrode (USCI, Billerica, Mass.) was introduced into a femoral vein and positioned in the right atrial appendage, and the atrium was paced with rectangular stimuli of 2-msec duration at twice the diastolic threshold current.

Body Surface Recordings

All body surface recordings were obtained during atrial pacing at cycle length of 310 msec. This cycle length was chosen because it was uniformly shorter than the spontaneous sinus cycle length but long enough to permit 1:1 conduction to the ventricles. Furthermore, 310 msec is not a multiple of the cycle length corresponding to 60-Hz electrical interference (16.67 msec); this minimizes the effect of 60-Hz interference on the averaged signal. The recording period was 240 seconds, which allowed for acquisition of approximately 770 beats.

Dogs were placed in the supine position, and 64 stainless-steel needle electrodes were inserted subcutaneously in an 8x8 grid over the thorax that extended from right to left posterior axillary lines and from suprasternal notch to costal margins. (See Figure 4 for schematic representation of electrode positions.) Each of the 64 thoracic leads was referenced to a Wilson central terminal. Signals were processed through 64 separate custom-built amplifiers with 800–2,500 gain and band-pass frequencies of 0.03–500 Hz.29 All 64 amplified signals were simultaneously sampled at 1 kHz and digitized to 12 bits. Digitized signals were recorded onto digital magnetic tape (Kennedy, Monrovia, Calif.).

Coronary Ligation

After baseline SAECG, dogs were subjected to a lateral left ventricular (n=17) or right ventricular myocardial infarction (n=14). Lateral left ventricular infarction was performed via a left thoracotomy in the fifth intercostal space. All diagonal branches of the left anterior descending coronary artery that supplied the lateral left ventricular free wall were ligated. Similarly, all
obtuse marginal branches of the circumflex coronary artery that supplied the lateral left ventricular free wall were ligated. In general, three or four diagonal branches and three or four marginal branches were ligated. The left anterior descending and circumflex arteries were not ligated.

Right ventricular infarction was performed via a right thoracotomy in the fifth intercostal space. The right coronary artery was ligated proximally and distally, the latter to prevent collateral blood flow from the distal circumflex artery. Collateral blood flow from small right ventricular branches of the left

FIGURE 1. Tracings showing detection of longer QRS duration by 64-lead signal-averaged electrocardiography (SAECG) than by three-lead SAECG after right ventricular infarction. Top tracing: One of the recordings from the 64-lead SAECG; it was selected as the one that showed the longest filtered, averaged QRS duration (115 msec). This lead was located in the right midprecordium at the sixth intercostal space; several neighboring leads showed comparable QRS durations. Next three tracings are the bipolar X, Y, and Z complexes, respectively, derived from the same recording data. Note that the longest QRS duration among these is only 87 msec. As discussed in “Methods,” QRS onset of the 64-lead SAECG is defined as the earliest onset among all 64 leads—in this case, in a lead other than lead 20 depicted here. QRS onset of the three-lead SAECG is defined as the earliest onset among the three derived bipolar leads—in this case, in lead Y. In this dog, onsets of the 64-lead and the three-lead SAECGs were identical, which was a typical result. Thus, the difference in QRS duration between the 64-lead SAECG and the three-lead SAECG is entirely accounted for by differences in the times of QRS offset.
anterior descending and posterior descending arteries was interrupted with a fine running suture in the epicardium 2–3 mm to the right of these arteries.

Dogs received 2 mg/kg lidocaine hydrochloride just before coronary ligation and again after a 20-minute interval. Ventricular fibrillation and sustained ventricular tachycardia were treated with immediate defibrillation or cardioversion. Blood pressure was supported as necessary during infarction with intravenous fluids, epinephrine, and calcium chloride.

After coronary artery ligation, the pericardium was sutured, and the chest was closed in layers. A thoracostomy tube was placed and attached to suction for 60 minutes. Dogs were allowed to recover from anesthesia and were returned to the vivarium fully conscious. Dogs received parenteral antibiotics for 3 days after surgery and parenteral analgesics as necessary. Dogs that appeared to be in distress after appropriate analgesia were euthanatized.

Thirteen of the 17 dogs that underwent lateral left ventricular infarction and eight of the 14 dogs that underwent right ventricular infarction survived until follow-up study 5–10 days later. These 21 surviving dogs are the subject of the present study. Two of the dogs that had undergone right ventricular infarction were found to be in stable monomorphic ventricular tachycardia at the time of follow-up study; in these two dogs, ventricular tachycardia was terminated with ventricular pacing using a transvenous catheter. However, no systematic attempt was made to induce ventricular tachycardia in the study dogs.

**Epicardial Recordings**

Epicardial recordings were performed at follow-up study after repeat body surface recordings. The chest was opened with a median sternotomy, and the heart was suspended in a pericardial cradle. Recordings were made from the epicardium using a previously described nylon sock electrode array.**30** Sixty-two fine silver-wire electrodes were embedded in the sock with approximately equal spacing, and the sock was stretched over the surface of the ventricles. Signals from each of the 62 sock electrodes were referenced to the Wilson central terminal. Like the body surface recordings, epicardial recordings were performed during atrial pacing at a cycle length of 310 msec. Recordings were filtered, and onsets and offsets were determined using the method described below for body surface recordings. The sock lead showing the longest ventricular electrogram was identified. Then the sock was removed, and a plaque with 64 closely spaced electrodes was sewn to the epicardium, centered at the point of the longest electrogram as determined by the sock recordings. The plaque has been described previously**31; it consists of 64 flat silver electrodes (0.6-mm-diameter) in an 8×8 array with 2-mm interelectrode distances. Signals from each of the plaque electrodes were referenced to the Wilson central terminal, and they were amplified (gain, 50–150), digitized, and recorded in a manner similar to that described above for the body surface recordings.

**Body Surface Signal Analysis**

Analysis was performed with a MicroVax II computer (Digital Equipment Corp., Maynard, Mass.). For signal-averaging, body surface QRS complexes were aligned to a manually selected template QRS complex using an algorithm that maximized the cross correlation of a 50-point (50-msec) portion of the QRS complex in a single lead. Only beats with a cross correlation with the template that exceeded 0.9 were included in the average.

Three bipolar recordings, constructed to resemble conventional SAECG recordings, were derived from data obtained from the 64-lead SAECG using the 8×8 electrode grid. An X bipolar lead was derived as the difference between the unipolar lead at the left midaxillary line, fifth intercostal space (positive), and that at the right midaxillary line, fifth intercostal space (negative). A Y bipolar lead was derived as the difference between the unipolar lead at the inferior, lateral, left costal margin (positive) and that just to the right of the sternal notch (negative). A Z bipolar lead was derived as the difference between the unipolar lead in the conventional V2 position (positive) and that in the right posterior axillary line in the same horizontal plane (negative).

The 64 averaged body surface unipolar recordings and the derived X, Y, and Z averaged recordings were each processed with a high-pass digital frequency filter. At each data point *x* (*n*), a second-order polynomial curve was fit using the least-squares technique to the values at *x* (*n*) and the 10 immediately preceding data points (*x* (*n*−10), ... , *x* (*n*−1)) and 10 immediately following data points (*x* (*n*+1), ... , *x* (*n*+10)). The value of the high-pass filtered curve at *x* (*n*) is then taken as the difference between the original value at *x* (*n*) and the value of the smoothed polynomial curve at *x* (*n*). This process was repeated at each point along all 64 original averaged recordings and the derived X, Y, and Z averaged recordings.

This least-squares filter has a corner frequency (half-power point) of 70 Hz, and its slope is 80 dB/decade, which is similar to that of a four-pole filter. However, the least-squares filter does not create the “ringing” artifacts seen with Butterworth filters.**16** (The maximum duration of signal spreading at the beginning and end of the signal resulting from the least-squares filter is 10 msec.) Furthermore, unlike the “bidirectional” Butterworth filters widely used for SAECG,**16** the least-squares filter does not distort the center of the signal.

The onset of each filtered QRS complex was determined automatically by a computer algorithm as follows: The root-mean-square value of a 17-point (17-msec) isoelectric portion of the PR segment, RMS*<sub>noise</sub>*, was calculated. The curve was then scanned forward in time until the mean value of three consecutive points exceeded ±1.6×RMS*<sub>noise</sub>*. If the mean value of the next three consecutive points also met...
this criterion, then the first of these six points was determined to be the QRS onset; if not, the algorithm continued with the next point to the right. The offset of each filtered QRS complex was determined in a similar fashion, except that a 33-point window of the TP segment was used to calculate $RMS_{\text{noise}}$.

The offset algorithm was devised to detect very-low-amplitude signals at the terminus of the QRS complex while minimizing the likelihood that isolated aberrant points would be taken as QRS activity. The sensitivity of the offset detection algorithm was tested using a computer-simulated signal of a sinusoidal wave of 80 Hz. The RMS amplitude of the smallest detectable sine wave equaled $2.0 \times RMS_{\text{noise}}$. (By contrast, the sensitivity of an algorithm commonly used clinically for the same test signal is $2.9 \times RMS_{\text{noise}}$.) For all 64 leads in all dogs studied after infarction, the $RMS_{\text{noise}}$ value used for offset detection was $0.28 \pm 0.09 \mu V$ (range, 0.13–0.64 $\mu V$). Therefore, in these experiments, signals with RMS amplitude as low as approximately 0.26 $\mu V$ could be detected.

Local QRS duration at each of the 64 surface leads was defined as the difference between QRS onset and QRS offset in that lead. The total QRS duration of the 64-lead SAECG was defined as the interval from the earliest QRS onset among all 64 leads to the latest QRS offset among all 64 leads. The total QRS duration of the three-lead SAECG was defined as the interval from the earliest QRS onset among the X, Y, and Z leads to the latest QRS offset among the X, Y, and Z leads.

Dogs in which the total QRS duration by 64-lead SAECG increased by 10 msec or more between baseline and postinfarction studies were defined as having a late potential. In these dogs, the local late potential amplitude was defined at each of the 64 body surface recording sites as the RMS value of the filtered, averaged recording at that site, taken over the terminal 25 msec of the total 64-lead QRS complex. Similarly, late potential amplitudes were defined for the derived X, Y, and Z leads as the RMS values of the filtered, averaged X, Y, and Z leads, taken over the terminal 25 msec of the total three-lead QRS complex.

**Epicardial Data Analysis**

Analysis was performed on the 64 electrograms recorded by the plaque from a single beat (without averaging). Signals were processed by the least-squares filter described above, and onset and offset were determined as described above for body surface recordings. The range of normal epicardial electrogram duration for our recording system was determined from a sample of 200 normal electrograms (100 recordings from the right ventricle in dogs who had undergone left ventricular infarction and 100 recordings from the left ventricle in dogs who had undergone right ventricular infarction). Mean normal epicardial electrogram duration was $55.9 \pm 7.1$ msec (range, 40–70 msec). The upper limit of normal was taken as mean+2 SD (70 msec). Epicardial electrograms were classified as abnormal solely on the basis of duration (longer than 70 msec); although abnormalities of shape also were noted, no attempt was made to quantitate these. Total epicardial activation time was defined as the interval from the earliest onset among the 64 plaque electrograms to the latest offset.

**Statistical Analysis**

Values of continuous variables are expressed as mean±SD. Paired and unpaired Student’s $t$ statistic was used to test for differences between continuous variables; the probability value cited is for the two-tailed hypothesis. A probability value of 0.05 or less was considered significant. Pearson’s $r$ statistic was used to test correlations between continuous variables.

To test differences in location of local late potential amplitude or duration maxima between right ventricular and left ventricular infarcts, the location of each amplitude maximum was coded as a $1 \times 2$ matrix $(j,k)$, where $j$ is the column number (1, . . . , 8) and $k$ is the row number (1, . . . , 8) of the maximum. Hotelling’s $t^2$ statistic in multivariate analysis of variance was used to test for differences in $(j,k)$ between the two infarct groups.

**Results**

**Effects of Myocardial Infarction on SAECG Variables**

At baseline, total QRS duration measured using all 64 body surface leads ($82.5 \pm 6.6$ msec) was similar to total QRS duration measured using three leads ($81.0 \pm 7.2$ msec, $p=\text{NS}$). However, the 64-lead SAECG detected more than twice as great an increment in total QRS duration resulting from infarction ($17.8 \pm 17.9$ msec) than did the three-lead SAECG ($8.4 \pm 11.2$ msec) ($p=0.002$) (Table 1). An increment of 10 msec or more in total QRS duration between baseline and postinfarction recordings was detected in 13 of 21 dogs (62%) with the 64-lead SAECG but only seven of 21 dogs (33%) with the three-lead SAECG. Similar increases in total QRS duration were detected by 64-lead SAECG in dogs undergoing right ventricular infarction ($15.4 \pm 17.0$ msec) compared with dogs undergoing left ventricular infarction ($19.4 \pm 18.9$ msec, $p=\text{NS}$). Figure 1 is from a dog that underwent right ventricular infarction in which postinfarction signal-averaged QRS duration detected using all 64 leads exceeded by 28 msec that detected using only three leads. In this case, greatest

<table>
<thead>
<tr>
<th>QRS Duration</th>
<th>Three-lead SAECG</th>
<th>64-lead SAECG</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>81.0±7.2</td>
<td>82.5±6.6</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>After infarction</td>
<td>89.4±10.1</td>
<td>100.3±16.3</td>
<td>0.0005</td>
</tr>
<tr>
<td>Increment</td>
<td>8.4±11.2</td>
<td>17.8±17.9</td>
<td>0.002</td>
</tr>
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SAECG, signal-averaged electrocardiography.
prolongation of the QRS complex was detected on the right midprecordium, which is an area not sampled by the three-lead SAECG.

QRS onsets determined by the 64-lead and three-lead SAECGs were usually simultaneous and never differed by more than 2 msec; therefore, the differences in total QRS duration between the two methods were almost entirely accounted for by differences in QRS offset (Figure 1).

In the 13 dogs with late potentials, the maximum local late potential amplitude detected among all 64 leads was 2.0±2.0 μV (range, 0.4–7.5 μV). Maximum local late potential amplitude did not differ significantly between dogs with right ventricular infarctions (1.7±1.0 μV) and those with left ventricular infarctions (2.2±2.5 μV).

**Relation to Epicardial Electrograms**

Prolonged (more than 70 msec) epicardial ventricular electrograms were recorded from the plaque electrodes in 19 of the 21 dogs (90%). Maximum electrogram duration in each dog was 99.4±18.7 msec. Abnormal electrograms were found only over the visible area of infarction.

Correlations between total epicardial activation time and total body surface signal-averaged QRS duration are shown in Figure 2 for both three-channel \((r=0.53, p=0.01)\) and 64-channel SAECGs \((r=0.88, p<0.0001)\). It is apparent that the 64-lead SAECG provides a much better estimate of ventricular activation time than does the three-lead SAECG; the mean absolute difference between epicardial activation time and QRS duration by 64-lead SAECG was 6.9±5.8 msec versus 14.7±11.3 msec for the three-lead SAECG \((p=0.007)\). Total epicardial activation times that were 100 msec or longer were particularly poorly estimated by three-lead SAECG QRS duration; the mean difference in 12 such dogs was 21.0±10.5 msec. An example of a dog in which epicardial activation time was better estimated by the 64-lead SAECG than by the three-lead SAECG is given in Figure 3.

**Localization of Late Potentials on the Body Surface**

Localization of body surface late potentials was performed in the 13 dogs in which late potentials
lateral left ventricular infarction. Leads showing a right-sided late potential in a dog with a right ventricular infarction are depicted in Figure 5, and leads showing a left-sided late potential in a dog with a left-sided ventricular infarction are depicted in Figure 6.

It is possible that the distribution and localization of detected late potential amplitude were influenced by differences in baseline noise levels among leads. However, the distribution of noise within a given dog was relatively even; the average SD of the noise in the 64 leads of each dog was only 0.04 μV. Furthermore, there was no correlation between local late potential amplitude and RMSnoise in the dogs with late potentials (r = −0.06; p = NS). The RMSnoise of the lead in each dog showing the maximum late potential amplitude (0.33 ± 0.11 μV) was not significantly different from that of the remainder of the leads (0.28 ± 0.09 μV). Therefore, it is unlikely that the distribution of local late potential amplitude was significantly influenced by uneven distributions of noise.

Local late potential amplitudes in the X, Y, and Z leads of the three-channel SAECG were examined in the 13 dogs with late potentials. Among the eight dogs with late potentials after left ventricular infarction, maximum local potential amplitude was in the X lead in three, the Y lead in four, and the Z lead in one. This distribution did not differ significantly from that in the dogs with late potentials after right ventricular infarction, for which the maximum was in the Y lead in two and the Z lead in three.

**Comparison of Local Late Potential Amplitude and Local QRS Duration in Localization of Late Potentials**

Local QRS duration measured at each of the 64 surface leads in the 13 dogs with late potentials was 82.7 ± 10.6 msec. In 10 of these dogs, there was a significant correlation between local QRS duration and local late potential amplitude. However, local QRS duration showed less dispersion among the leads compared with local late potential amplitude. For instance, the coefficient of variation (SD divided by mean value) for local QRS duration (11 ± 3%) among the 13 dogs was significantly less than the coefficient of variation for local late potential amplitude (34 ± 13%, p < 0.0001). Similarly, the ratio of maximum to mean local QRS duration (1.3 ± 0.1) was significantly less than that of local late potential amplitude (2.0 ± 0.4, p < 0.0001). A discrete, single maximum of local QRS duration was present in only nine of the 13 dogs with late potentials (three with right ventricular infarctions and six with left ventricular infarctions). In these nine dogs, the duration maxima in the dogs with right ventricular infarctions was to the right and above the duration maxima in the dogs with left ventricular infarctions, similar to the pattern seen with local late potential amplitudes. However, the distinction between infarct locations using local QRS duration (p = 0.04) was not as statistically significant as the distinction using local late potential amplitude (p = 0.006). Figure 7 shows data from a dog with a left ventricular infarction.

![Figure 3](http://circ.ahajournals.org/content/87/6/877/F3.large.jpg)
ventricular infarct in which local late potential amplitude showed a more discrete and localizing maximum than did local QRS duration.

**Discussion**

The principal findings of the present study were that 1) more extensive recording from the body surface with 64-lead SAECG detects longer late potentials than does conventional three-lead SAECG, 2) the longer late potentials detected with 64-lead SAECG correspond temporally to prolonged ventricular epicardial electrograms recorded in the area of infarction, and 3) the body surface location of late potentials corresponds to the cardiac location of infarction and prolonged electrograms.

**Enhanced Detection of Late Potentials With 64-Lead SAECG**

Conventional SAECG" uses recordings from three bipolar lead pairs, which are approximately orthogonal in orientation. However, experience in electrocardiographic body surface potential mapping indicates that additional information concerning the QRSST complex is obtained from the addition of a number of precordial recording sites, which provide regionally selective information about cardiac electrical activity." The results of the present study confirm the enhanced detectability of ventricular late potentials with body surface mapping techniques using multiple thoracic leads.

There have been two prior preliminary reports of enhanced detection of late potentials with body surface mapping techniques; one showed additional QRS prolongations of as long as 14 msec compared with conventional SAECG, and the other showed additional QRS prolongations averaging 16 msec. The magnitude of these additional QRS prolongations is in agreement with the results of the present study (Table 1). A third study comparing signal-averaged QRS durations between conventional and body surface SAECG techniques reported only a minimal difference. However, unlike the present study and the above-mentioned reports, this latter study measured signal-averaged QRS duration from the vector combination of the leads, and this probably interfered with the improved sensitivity of the body surface technique.

The amplitude of the late potentials detected with our methods was low (mean, 2.0 μV) compared with the amplitude of late potentials detected using methods commonly used clinically; for example, Simson used 15 μV in 39 patients with inducible ventricular tachycardia. There are several explanations for the lower amplitude of the late potentials detected in the present study. First, the corner frequency of the filter used in this study (70 Hz) was higher than that generally used clinically (25–40 Hz), and the higher corner frequency will result in diminution of the absolute amplitude of the filtered signals. Second, the offset...
individually, allowed for the detection of low-amplitude late potentials that would not have been detected by the usual technique of examining only the vector magnitude of three leads.36,37 The physiological relevance of the low-amplitude late potentials

**FIGURE 5.** Top panel: Tracings from selected leads of 64-lead signal-averaged electrocardiography after lateral left ventricular infarct; tracings are arranged from right to left at the same horizontal level (sixth intercostal space, anterior). Horizontal bar under each tracing marks the terminal 25 msec of the total 64-lead QRS complex; it is simultaneous for all tracings and represents the time from which local late potential amplitudes are measured. Number under each tracing is the local late potential amplitude measured in microvolts. Note that the greatest local late potential amplitude is found in the left midprecordium; this corresponds to the larger amplitude in this lead above the horizontal bar. Bottom panel: Contour map of local late potential amplitudes from the 8x8 electrode grid. (See Figure 4 for grid landmarks.) Each contour line represents an increment of 0.2 μV. Contour map illustrates the concentration of leads with greatest local late potential amplitude over the left lower precordium.

**FIGURE 6.** Tracings from selected leads of 64-lead signal-averaged electrocardiography after right ventricular infarction. Figure is arranged similarly to Figure 5. In this case, greatest local late potential amplitude is over right parasternal and midprecordial area. Each line in contour map represents an increment of 0.1 μV in late potential amplitude.

detection algorithm used in the present study was designed to be sensitive to low-amplitude signals and is more sensitive than the algorithm commonly used clinically (see "Methods"). Last and most important, use of a larger number of leads, each examined
detected in the present study is supported by their temporal and spatial correlations with prolonged epicardial electrograms. However, it remains to be determined whether they have an association with spontaneous ventricular arrhythmias, as has been found for late potentials detected with more conventional techniques.

**Correlation With Epicardial Activation Times**

Total duration of epicardial activation in this canine infarct model was frequently underestimated by the total QRS duration measured with three-channel SAECG (Figure 2). Although not systematically described, examples of similar results have been previously depicted in other reports of subacute and chronic canine infarct models.\(^{19,21}\) However, one previous study reported a much better correlation between epicardial activation time and QRS duration by three-channel SAECG.\(^{15}\) Recordings in this study were made during acute ischemia induced by coronary artery ligation and latex embolization; it is possible that the prolonged electrograms produced by this method are more widespread than in the subacute or chronic phase of infarction and thus would be more fully detected by three-lead SAECG.

The findings of the present study indicate that after infarction, some delayed components of epicardial electrograms cannot be detected by any of the three bipolar leads used in conventional SAECG, but they can be detected by one or more of the 64 leads used in our body surface mapping technique. Even the 64-lead system failed to detect the full duration of epicardial activation in some dogs in the present study. It is possible that such activation could be detected by use of a larger number of surface leads, an enhanced signal-to-noise ratio, or a more sensitive QRS offset detection algorithm.

In some animals in the present study, the QRS duration determined by 64-lead SAECG overestimated by as much as 15 msec the total measured duration of epicardial activation. This phenomenon has been previously reported in humans studied with

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**FIGURE 7.** Comparison of contour maps of local QRS duration and local late potential amplitude in a dog with lateral left ventricular infarction. Contour maps are oriented identically to those in Figures 4–6; zones of the contour map are shaded for clarity. Note the more distinct features of the amplitude contours compared with the duration contours; there is a single amplitude maximum, whereas there are two duration maxima, one of which is quite broad. Also shown are representative filtered QRS complexes from the right and left precordium. Left precordial lead has a late potential with amplitude nearly fivefold that of the right precordial lead, whereas the QRS durations of the two leads are nearly identical.
three-lead SAECG; in these cases, recordings from the endocardium revealed electrograms temporally coinciding with the terminal signal-averaged QRS complex. The origin of the QRS activity that extended beyond the recorded epicardial electrograms in the present study is not known: it could have been endocardial, intramural, or an epicardial location not sampled by the plaque.

Body Surface Localization of QRS Late Potentials

The two infarct models used in this study—lateral left ventricular and right ventricular—were selected to result in divergent epicardial locations of prolonged electrograms. As expected, prolonged electrograms were invariably confined to the areas of infarction. The general relation between the epicardial location of prolonged electrograms and the body surface location of maximum late potential amplitude was straightforward; just as the right ventricular free wall is located to the right and slightly superior to the left ventricular free wall, maximum late potential amplitude for right ventricular infarctions was located to the right and slightly superior compared with that for lateral left ventricular infarctions. Such a relation would be predicted by field theory of an electrical signal and a unipolar electrode in a homogeneous volume conductor.

However, there was some variability in the location of late potential maxima within each of the two infarct groups, and there was some overlap between the late potential maxima of the two infarct groups (Figure 4). It is possible that subtle differences in infarct location or in abnormal electrogram location accounted for the variability in late potential location among similar infarcts; however, any such pattern was not apparent in the present study. As an alternative, these differences may have been caused by heterogeneity of the volume conductor between the epicardium and the body surface, variability among breeds of dogs in thoracic conformation, and variation in the position of the heart within the thorax.

Late potential maxima tended to be located centrally on the anterior thorax. All except one maximum was located horizontally between the right parasternal area and the left midprecordium, and all were located vertically between the fourth intercostal space and the xiphoid process (Figure 4). Localization of late potential maxima more laterally and posteriorly may have been limited by attenuation of the late potential signal by intervening lung. In humans (especially those with chronic heart disease), there is more extensive apposition of the anterior and lateral surfaces of the heart with the chest wall, and precordial mapping may provide more detailed localization of late potentials than in dogs.

We found that quantitation of local late potential amplitude was a more useful technique for localizing late potentials than was simple measurement of local QRS duration. Others have used amplitude techniques to quantitate high-frequency components of the QRS complex. For example, Ohe et al examined signal-averaged QRS complexes at two precordial sites in patients with late potentials and found that the relative amplitude of the two late potentials correlated with the presumed site of origin of the late potential. Ohe et al found less variation in the duration of the late potential between the two leads, and differences in duration did not correlate with the presumed site of the late potential. Although the positive results of Ohe et al are encouraging for application of body surface mapping techniques to SAECG in humans, it is likely that better results will be obtained with more than two precordial leads.

Shibata et al also quantitated high-frequency signals by calculating their amplitude. Using an 87-lead SAECG system, Shibata et al were able to distinguish patients with anterior and inferior left ventricular infarction on the basis of the body surface distribution of high-frequency signal amplitude. Unlike the present study, however, Shibata et al examined the terminal portion of the standard QRS complex (a 20-msec segment terminating at the QRS J point), and they did not analyze signals after the J point that contained late potential activity.

Faugère et al and Lacroix et al reported a different approach to body surface mapping of late potentials. They constructed body surface isopotential maps of filtered, averaged electrocardiographic signals at various instants during the period of the late potential and determined the sites of positive and negative poles on the isopotential maps. They found that late potentials arising from anterior or apical cardiac sites tended to have closely spaced positive and negative potential poles, whereas late potentials arising from inferior or left lateral sites had more widely separated potential poles. The relation between this technique of examining instantaneous isopotential distributions and our technique of summing the late potential amplitude over its duration is not straightforward. We chose not to examine isopotentials of filtered data because of concerns that the filtering process could result in time shifts and polarity reversals, which could distort isopotential maps, such as was documented by Lacroix et al using one of their filters.

Limitations

In the present study, data were collected from dogs 5–10 days after permanent occlusion of coronary arteries. This is a period of active remodeling of infarct anatomy, and there is evidence that electrophysiological properties of the ventricular myocardium, including inducibility of ventricular arrhythmias, is changing during this period. Thus, the 5–10-day infarct may not be representative of the more chronic postinfarction state. Furthermore, because this study was not designed to produce a model of inducible ventricular tachycardia but rather to produce models of prolonged electrograms, it is possible that the abnormal electrograms and the late potentials were somehow not typical of those associated with ventricular arrhythmias. However, two of
the dogs in the present study had spontaneous, sustained monomorphic ventricular tachycardia, and their electrograms and signal-averaged QRS complexes were typical of those seen among the entire study group.

Control dogs undergoing sham operation were not included in this study, so it is possible that the prolonged epicardial electrograms and late potentials detected did not result from the infarction per se but rather from nonspecific epicardial trauma, inflammation, or denervation. However, this possibility appears remote for several reasons. There is abundant clinical and experimental evidence that myocardial infarction results in prolongation of electrograms and late potentials, whereas there is no evidence to suggest that these are likely to result from other processes related to thoracotomy and pericardiectomy. Furthermore, prolonged electrograms were detected only over the area of visible infarction, which generally was distal to the points of coronary ligation where the greatest nonspecific trauma would be expected. In any case, the temporal and spatial correlations between the abnormal electrograms and the late potentials are likely to be valid regardless of the precise mechanism by which the abnormal electrograms were produced.

Our method of determining the duration of cardiac activation was limited to recordings of epicardial sites, and we did not record from intramural or endocardial sites. As noted above, intramural or endocardial activation may account for some portions of late potentials that persisted beyond the termination of epicardial activation. However, previous study of canine infarct models has shown that abnormal electrograms and reentrant tachycardia circuits are usually confined to the subepicardium. This is consistent with the overall excellent correlation we found between epicardial total activation time and signal-averaged total QRS duration.

The two infarct locations used in the present study were designed to result in abnormal electrograms arising from opposite sides of the heart. Clinically, late potentials are usually associated with left ventricular disease—most often, left ventricular infarction. Therefore, the most clinically relevant application of late potential mapping would be the distinction among late potentials arising from different left ventricular sites. As noted above, other authors, using methods different from those used in the present study, have found differences in the distribution of high-frequency signals between patients with anterior and those with inferior left ventricular infarctions. Whether the techniques described in the present study will distinguish late potentials arising from different left ventricular sites remains to be determined.

Implications

Detection of patients at risk for life-threatening ventricular arrhythmias after myocardial infarction and with other forms of heart disease is crucial for timely intervention. The SAECG, although a new technique, has already assumed an important role in detecting arrhythmia-prone patients. Remarkably, the technique of SAECG used clinically has remained nearly identical to that introduced in 1981 by Simson, with analysis limited to the vector combination of three approximately orthogonal leads. The present and other recent studies indicate that analysis of a larger lead set may increase the sensitivity of the SAECG in detecting abnormal electrograms. Furthermore, the spatial distribution of late potential amplitudes on the body surface may permit localization of abnormal electrograms within the heart, which may be sites of origin for arrhythmias.

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