Characterization of the spatial distribution of late ventricular potentials by body surface mapping in patients with ventricular tachycardia

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ABSTRACT  Low-level activity at the end of the QRS complex was analyzed from 63 thoracic leads in 15 normal subjects and in 21 patients with ventricular tachycardia (VT). The latter had old myocardial infarction and no conduction disturbances and had not been receiving antiarrhythmic drugs. In both normal subjects and patients with VT, isopotential maps of the time-averaged and filtered (25 Hz high-pass) electrocardiograms during the terminal portion of the QRS were dipolar, i.e., they showed single positive and negative regions. For patients with VT, the extrema were either distant, with one over the precordial area and the other over the back, or close together in the precordial region. In 10 patients, maps recorded after administration of antiarrhythmic drugs remained the same while QRS duration was prolonged. In six patients, maps recorded before antiarrhythmic surgery showed distant extrema for septal or posterobasal VT sites of origin and close extrema for anterior or posterocaudal sites. Generally, QRS duration was reduced and maps were modified after surgery. Late potentials can be well detected with only three orthogonal leads because their distributions are dipolar, but maps provide additional information about their distribution, which may be related to conduction delay sites and possibly to VT sites of origin. Sources near the torso surface would produce close extrema, whereas deeper sources would produce distant extrema.

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HIGH-RESOLUTION signal-averaged electrocardiograms have shown the presence of low-level, high-frequency potentials immediately after the QRS complex in patients with coronary heart disease and ventricular tachycardia (VT) during normal sinus rhythm. It has been suggested that these late ventricular potentials reflect the presence of areas of delayed conduction where reentry can easily occur and that they can be used as a sensitive marker for the identification of subjects prone to sustained VT. These features have also been previously described in cases of right ventricular arrhythmogenic dysplasia and more recently in cases of nonischemic congestive cardiomyopathy with cardiomegaly and VT.

In this study, we have characterized the spatial distribution of the late potentials by analyzing 63 simultaneously recorded leads in normal subjects and in patients with old myocardial infarction and documented VT. Previously, late potentials had been recorded with only three or four bipolar leads, which did not supply information about the location and extent of these potentials on the torso surface.

The analysis of these 63 time-averaged and filtered electrocardiograms can provide (1) information about the number and location of leads required to record late potentials, (2) a more detailed description of the late potentials and of the modifications caused by antiarrhythmic drugs or surgery than the one provided by three orthogonal leads, and (3) information about the location of the late potentials on the torso surface, which could be compared with the location of the arrhythmogenic site.

Methods

Patients. The study protocol, which was approved by an institutional committee on human research, was performed on two groups of subjects that had been informed of its purposes. The first group (table 1A) consisted of 15 normal subjects (five women, 10 men), aged between 20 and 35 years (28 ± 5), who...
TABLE 1A
Normal subjects

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<th>Sex/age (yr)</th>
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<th>V_{av} (μV)</th>
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BSPM = body surface potential mapping (A, sternal; B, anteroposterior).

TABLE 1B
Patients with VT

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<th>Sex/age (yr)</th>
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Before drugs | After drugs

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Before surgery | After surgery

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<th>V_{av} (μV)</th>
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<td>118</td>
<td>14</td>
<td>C</td>
<td>Post-Bas</td>
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MI site: A = anterior; I = inferior.
BSPM extrema: A = sternal; B = anteroposterior; C = close; C* = close but different than before surgery (see figure 8); D = distant.
Treatment: Amio = amiodarone; Sot = sotalol hydrochloride; Proc = procainamide; Mexi = mexilidene; Cryo = cryoablation; Aneu = aneurysmectomy; ERP = endocardial resection procedure.
Site of origin of VT: Sept = septal; Post-Bas = posterobasal; Ant = anterior; Post-Ap = posteroapical.
Other abbreviations as in table 1A.
The 63-lead array and format of the isopotential maps. The upper edge of a map corresponds to the top electrode row, the lower edge to the lower row, and the side edges to the right midaxillary line.

FIGURE 1. The 63-lead array and format of the isopotential maps. The upper edge of a map corresponds to the top electrode row, the lower edge to the lower row, and the side edges to the right midaxillary line.

front and sides of the torso and 20 electrodes on the back (figure 1). The electrodes were Ag-AgCl disks mounted on 12 vertical adhesive strips, with an interelectrode distance of 6 cm. The first strip was applied over the sternum with the top electrode over the suprasternal notch. The first electrode of each of the eleven other strips was applied at the same level. For each subject, at least three consecutive recordings were performed during normal sinus rhythm in the supine position.

The electrocardiograms were recorded and averaged with an integrated mapping system (CORDIC) developed at the Institut de génie biomédical. This system is based on a Z80 microprocessor with a 64 kbytes memory, a 10 Mbytes hard disk, and a 64 channel data acquisition system with programmable gains and bandwidths. The functions performed by the CORDIC software include data acquisition with simultaneous display of the signals, classification and averaging of the recorded beats, and generation of isopotential maps. With this system, the electrocardiograms were amplified, filtered with a bandwidth of 0.05 to 200 Hz, sampled at 500 Hz, digitized with a resolution of 2.5 μV and stored on a hard disk for periods of 52 sec.

**Data processing.** A program was used to classify normal and ectopic beats in up to four different categories according to the RR interval, QRS duration, and R and S wave amplitudes. For each category, beats were averaged separately for each lead to improve the signal-to-noise ratio. Averaging was performed after aligning each beat with the template by using a cross-correlation technique. The averaged signals, which included 59 ± 8 beats, were corrected for baseline shift by subtracting a straight line joining two isoelectric segments.

The signals were then transferred by a telephone line to a PDP 11/34 minicomputer for further processing. Digital filtering was performed on each of the 63 electrocardiograms to reject low frequency components and 60 Hz interference. Filtering was performed in a bidirectional manner to reduce the effects of ringing; an autoregressive filter was forward-decimated until 0.44 msec after the QRS onset, then reset and moved backwards until the same time instant (the QRS onset was automatically determined on the root-mean-square of the 63 averaged signals). The filter was a fourth-order Butterworth with a high-pass cutoff frequency of 25 Hz, with the addition of a first-order narrow band rejection filter centered at 60 Hz. We found that in electrocardiograms without 60 Hz interference, this additional band-reject filter did not alter the signal.

Two different methods were used to compute three vectorcardiographic leads. (1) Three X,Y,Z bipolar leads were obtained by subtracting the filtered electrocardiograms measured at the right and left midaxillary lines for the X lead, at the suprasternal notch and the umbilicus for the Y lead, and at the posterior chest and the sternum for the Z lead. (2) The other method was developed to reduce the noise by taking into account all the leads; the three X,Y,Z signals were computed by a least-square fit between all the 63 filtered electrocardiograms and three bipolar potential distributions generated on the surface of a realistic torso model, including regions of lower electrical conductivity representing the lungs.

The three X,Y,Z signals were used to determine the QRS duration and the average voltage of the terminal part of the QRS in the following manner. The vector magnitude of the three filtered vectorcardiographic components was computed and the QRS onset and offset automatically determined as the points where the magnitude function exceeded the mean plus 3 SDs of a noise sample measured during an isoelectric segment. An operator examined and eventually edited these QRS limits after inspecting on a scope the magnitude function and the three X,Y,Z signals (figure 2). The QRS duration was then obtained from these QRS limits. The root-mean-square of the magnitude function for the last 40 msec was computed and is referred to as $V_{50}$ in the rest of this article.

**FIGURE 2.** The three X,Y,Z signals and their magnitude function for a patient with VT. The vertical lines indicate the QRS onset and offset that were automatically detected and may be manually edited by the operator.
To characterize the spatial distribution of the late potentials, isopotential maps of the 63 filtered electrocardiograms were drawn for each sampling instant between 80 msec after QRS onset and the QRS offset. In these maps, the torso surface is represented in a rectangular format (see figure 1). The isopotential lines that depict the potential distribution on the filtered electrocardiograms at a given time instant are obtained by cubic spline interpolation and are drawn for intervals of 5 μV. The zero-potential line is identified by a heavier trace and plus and minus signs indicate the locations of the maximum and minimum.

Each recording was repeated at least three times during the same session to verify the reproducibility of the measurements. The vectorcardiographic measurements from each patient are the mean values for a single session. The results for each subgroup are given as the mean ± 1 SD. The statistical analyses were performed with the Student t test for either paired or unpaired observations according to the case.

Results

Vectorcardiographic measurements. The QRS duration and V_{40} were measured on the filtered vectorcardiograms for every recording of all normal subjects and patients with VT (three to four recordings for each individual, for a total of 131 recordings). A small but significant difference in QRS duration (p < .05) was found between the vectorcardiogram obtained with three bipolar leads (114 ± 26 msec) and that obtained with 63 unipolar leads (116 ± 29 msec). However, no significant difference was found between the V_{40} obtained with the two methods: 52 ± 48 μV for three leads and 54 ± 53 μV for 63 leads.

Since the QRS duration measured with three leads is only 2 msec shorter than that with 63 leads, and since measurements from three bipolar leads have previously been used in most studies, the QRS duration and voltage values in the rest of this article were obtained with the three bipolar leads.

Incidence of late potentials. The results for the QRS duration and V_{40} measured on the filtered QRS complexes are presented in table 2. The QRS duration was significantly longer (p < .005) for patients with VT (118 ± 20 msec) than for normal subjects (99 ± 10 msec), whereas a less significant difference (p < .05) was found between these two groups for the V_{40} (43 ± 41 vs 76 ± 57 μV). The QRS duration was also slightly longer (p < .10) for the patients with inferior myocardial infarction (125 ± 18 msec) than for those with anterior myocardial infarction (112 ± 20 msec), whereas the V_{40} was significantly smaller (p < .025) for the group with inferior (22 ± 10 μV) than for the group with anterior myocardial infarction (56 ± 47 μV). Table 2 also presents the relative number of subjects with a QRS duration longer than 120 msec, a V_{40} smaller than 25 μV, and a V_{40} smaller than 40 μV for the different categories.

Isopotential maps during late QRS. The morphology of the isopotential maps of different filtered recordings from the same session were strictly reproducible during the last 10 msec of the QRS, whereas V_{40} values and QRS durations showed slight variations.

In the group of 15 normal subjects, all the late QRS maps were dipolar: they showed a single positive region and a single negative region. For 10 subjects, the extrema were close together on a vertical line over the sternum (figure 3, A). For the other five subjects, the extrema were more distant, with one over the sternum and the other over the middle of the back (figure 3, B).

In the group of 21 patients with VT, all the late QRS maps were also dipolar. For 15 patients the extrema were distant, with one over the precordial area and the other over the back (figure 4, A). For the other six patients the extrema were close together in the precordial or left midaxillary region (figure 4, B) and were not located vertically in the sternal region as for the normal subjects. For the 13 patients with anterior myocardial infarction, the maps showed distant extrema in seven and close extrema in six. For the seven patients with inferior and the one with both anterior and inferior myocardial infarction, the maps always showed distant extrema. For the 10 patients with a V_{40} under 25 μV, the maps showed distant extrema in nine (three with anterior, five with inferior, and one with both anterior and inferior myocardial infarction) and close extrema for one (anterior myocardial infarction). Maps with more than two extrema were found only once in a patient who had no conduction disturbance but did not belong to the previously described study group because he was already on drug treatment (figure 5). His maps were reproducible and neither noise nor artifacts could be seen on the electrocardiograms. The data from this additional patient are presented here to confirm the possibility of detecting multiple extrema with our mapping system.

Effects of drugs. Recordings were performed in 10

<table>
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<td>V_{40} &lt; 40 μV (%)</td>
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Ant. = anterior; Post. = posterior; MI = myocardial infarction.

*p < .05; ^p < .005.
antiarrhythmic surgery, the origin of the VT determined by epicardial and endocardial mapping was septal in one, anterior in one, posterobasal in two, and posterocapital in one. In one patient, VT could not be induced in the operating room and surgery was performed in the septal region on the basis of its anatomic appearance. For all patients, no VT could be induced during postoperative electrophysiologic control, and the body surface potential maps were recorded without pharmacologic treatment, both before and after surgery.

After surgery, the QRS duration was significantly reduced from 119 ± 26 to 99 ± 17 msec (p < .01). A shortening was seen in five patients (four with aneurysmectomy, one without), whereas the duration increased slightly for the patient who had a normal QRS duration, a $V_{40}$ over 25 $\mu V$, and no inducible VT at surgery. The effects of surgery on the $V_{40}$ were not significant; it increased in four patients and decreased in two, and the mean changed from 47 ± 47 to 64 ± 53 $\mu V$.

Before surgery, the maps showed distant extrema for the four patients who had septal or posterobasal VT

patients with VT before and after administration of antiarrhythmic drugs. The QRS duration was prolonged in eight patients and changed from 119 ± 21 to 129 ± 30 msec for the entire group (p < .02). The $V_{40}$ showed no significant changes for the entire group (50 ± 47 vs 39 ± 31 $\mu V$); the values increased in four and decreased in six. Among the five patients who had a $V_{40}$ less than 25 $\mu V$ without treatment, $V_{40}$ stayed under 25 $\mu V$ in four and increased to 29 $\mu V$ in one. The isopotential maps at QRS offset showed distant extrema in seven patients and close extrema in three. The morphology was the same after treatment. The maps for patient 7 after treatment with amiodarone (figure 6, right) are shifted by about 16 msec with respect to those before treatment (figure 6, left) and they retain the same morphology until QRS offset. The $V_{40}$ remained the same (from 32 to 35 $\mu V$).

Effects of surgery. For six patients who underwent

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**FIGURE 3.** Typical isopotential maps for normal subjects during the last 10 msec of the QRS. **A**, Morphology with two close sternal extrema. **B**, Morphology with two distant extrema. Each map corresponds to a specific instant relative to QRS onset, indicated over the upper left corner. The isopotential lines depict the potential distribution of the filtered electrocardiograms and are drawn for intervals of 5 $\mu V$. The zero-potential line is identified by a heavier trace and the plus and minus signs indicate the maximum and minimum.

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**FIGURE 4.** Typical isopotential maps for patients with VT during the last 10 msec of the QRS. **A**, Morphology with two distant extrema. **B**, Morphology with two close extrema.
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Generation of late potentials. In chronic myocardial infarction, late potentials appear to be produced by slow and delayed activity associated with reentrant arrhythmias. In animal preparations, fragmented, double, and late electrograms are mainly recorded at the border zone of the infarct and are related to the occurrence of VT.19-21 The source of these fragmented electrograms seems to be islands of viable myocardial cells with normal action potentials but disoriented activation within a fibrous zone.21 Surface recordings of low-level, high-frequency, and delayed potentials have been correlated with the presence of delayed conduction,22 and certain conditions such as variations in heart rate modify both late potentials and activation sequence.22, 23

In man, delayed and fragmented electrograms have been found during sinus rhythm in patients with coronary heart disease during intraoperative mapping, sites and close extrema for the two who had anterior or posteroapical VT sites (figure 7). After surgery, the maps showed a modified morphology in five patients (figure 8); four of them also had a shorter QRS duration. The last one retained his late potentials (V_{40.7} vs 14 μV) with the same map morphology but with a shorter QRS duration (138 vs 122 msec) after a successful posterior aneurysmectomy and cryoablation.

Discussion

This study provides new information about the spatial distribution of late potentials recorded on the body surface. Successive isopotential maps of filtered electrocardiograms have revealed the organization and location of late potentials that could not have been obtained with three orthogonal leads.

FIGURE 5. Multipolar morphology of the isopotential maps of a patient on amiodarone with VT during the last 10 msec of the QRS. Two maxima and two minima can be seen around 120 msec.

FIGURE 6. Effect of amiodarone on the isopotential maps of a patient with VT (No. 7) near QRS offset. Left, Without treatment. Right, With amiodarone. Note the similar morphology of the maps, which are shifted about 16 msec after treatment.
more frequently at the endocardium than at the epicardium and on wider surfaces in patients with spontaneous VT. The sites of origin of VT often correspond to the areas showing fragmented electrograms in sinus rhythm, but these areas do not always show the most delayed activity. When these electrograms are prolonged beyond the QRS complex and cover a wide endocardial region, they are detected on surface leads as late potentials. The timing of the late potentials corresponds to that of the fragmented electrograms. The prolongation of these potentials during rapid atrial pacing is related to the inducibility of VT by programmed stimulation and suggests its involvement in initiating VT. The duration of these late potentials has been correlated to the spontaneous VT cycle length. The incidence of surface-recorded late potentials after myocardial infarction varies with time, suggesting that anatomic changes such as scar formation are involved.

In most published reports, a time-averaging process is used to improve the detection of late potentials by reducing the noise level on the body surface recordings. However, the averaged signals cannot reveal the beat-to-beat variations in delay of late electrical activity such as would be possible with beat-to-beat analysis. El Sherif et al. showed in epicardial electrograms in the dog that the delay in late activity could change from beat-to-beat with a Wenckebach periodicity or higher-degree block. The same phenomenon was also observed in man. Likewise, Berbari et al. showed the variations in late activity after an atrial extrastimulus in the dog and the correspondence between epicardial and thoracic potentials.

**Vectorcardiographic measurements.** Late potentials are usually measured on the root-mean-square values of three orthogonal leads that are filtered at 25 Hz. Three variables are usually considered to identify high-risk patients: (1) the mean voltage of the last 40 msec, which should be smaller than 25 μV, (2) the QRS duration, which should be greater than 120 msec, and (3) the duration of potentials that stayed under 20 to 40 μV at QRS offset, which should be longer than about 40 msec.

In our study, the mean QRS duration and V\textsubscript{m} values for normal subjects are similar to those reported elsewhere, although the rate of false positives for V\textsubscript{m} in our group is greater (20% vs 6% to 12%). Different values are reported in the literature for the incidence of late potentials in patients with coronary heart disease and VT, as well as for the duration of the filtered QRS complex in absence of intraventricular conduction disorders. With filtering at 25 Hz, Simson\textsuperscript{5} found a V\textsubscript{m} less than 25 μV in 92% of subjects with VT compared with 7% in patients with myocardial infarction but without VT, and a QRS duration greater than 120 msec in 72% and 0% of the patients, respectively. With filtering at 40 Hz, Denes et al.\textsuperscript{7} found a V\textsubscript{m} of less than 20 μV in 83% and a QRS duration longer than 120 msec in 58% of patients with myocardial infarction and VT. For our patients, the V\textsubscript{m} was less than 25 μV in 48% and less than 40 μV in 71%. This was found more often in patients with inferior myocardial infarction.
FIGURE 8. Effects of antiarrhythmic surgery on the unfiltered electrocardiogram (top), the filtered electrocardiogram (middle), and the isopotential maps of the last 10 msec of the QRS of a patient with anterior myocardial infarction and anterior site of origin of VT (No. 18). Left, Before surgery. Right, After surgery.

(57% and 86%, respectively) than in those with anterior myocardial infarction (39% and 62%). The greater prevalence of late potentials in patients with inferior compared to anterior myocardial infarction was found by others in patients with and without VT. This can be caused by a conduction delay in areas that are normally depolarized the latest, and for some investigators it is a criterion to identify posteroinferior myocardial infarction.

Dipolar distribution of late potentials. The analysis of the 63 leads showed a coherent organization of the late potentials on the torso surface. The morphology of the late potentials seen on a given filtered electrocardiogram was always similar to that of the neighboring leads. This was expressed on the isopotential maps by dipolar distributions.

We computed the vectorcardiogram from 63 electrodes to determine whether late electrical events could be detected better than with the three usual bipolar leads. It appears that the QRS durations were slightly increased by this procedure and that the $V_{40}$ values were not significantly changed. This can be attributed to the dipolar distribution of the isopotential maps: the three orthogonal leads did measure the three vector components of a dipole source. The slightly longer duration with the 63-lead vectorcardiogram can be ascribed to the feasibility of detecting lower-level signals at the QRS end points when the noise level is reduced by using all 63 leads instead of three. However, even if the signal-to-noise ratio can be larger for some of the 63 leads, the late potentials were reasonably well detected by only three bipolar orthogonal leads.

On the body surface maps, the distance between extrema may be linked to the distance between the thoracic surface and the area of myocardium generating the electrical activity: sources close to the torso surface generate close extrema while more distant sources generate distant extrema. The isopotential maps of normal subjects presented characteristic dipolar aspects that correspond to the terminal activity at the base of the ventricles. For our patients with VT the maps showed distant extrema for all with inferior myocardial infarction and for seven of 13 with anterior myocardial infarction. In six with anterior myocardial infarction, close extrema were found. The same observation was made in the group of 10 patients with a $V_{40}$ less than 25 $\mu$V: distant extrema were seen in three with anterior, five with inferior, and one with anterior-inferior myocardial infarction. This suggests that the spatial distribution of late potentials may be related to a localized area of conduction delay that does not necessarily correspond to the site of infarction determined by the electrocardiogram. For the single patient with multiple extrema, existence of delayed activity in separate areas may be postulated.

Effects of antiarrhythmic drugs. After antiarrhythmic treatment, a significant QRS prolongation was observed in our patients without significant changes in $V_{40}$. However, the terminal isopotential maps were not affected either in their dipolar character or in their distribution. The limited size of our study group does not allow us to reach conclusions on the precise effects of antiarrhythmic drugs. Rather, our purpose is to illustrate the use of the mapping technique to better characterize drug effects on late potentials. Thus, even with variations in the QRS duration, the absence of
changes of the spatial distribution after drugs suggests that the location of the terminal activity was not changed.

The effects of antiarrhythmic drugs on late potentials have been studied in animal preparations as well as in man. The incidence was rarely modified by therapy. Measurements in sinus rhythm were either slightly changed or showed a prolongation of the QRS duration with a $V_{40}$ decrease, especially with quinidine, procainamide, and amiodarone; however, no relationship was found with either inducible or spontaneously occurring VT. Only Cain et al., with a special frequency analysis technique, have reported that antiarrhythmic drugs decrease the high-frequency components of the averaged electrocardiogram. Studies on the reproducibility of late potential measurements showed spontaneous variations in duration and voltage.

**Effects of antiarrhythmic surgery.** After antiarrhythmic surgery, QRS duration was decreased and the body surface maps at QRS offset were modified. These modifications could indicate a change in the location of the terminal activity. In our patients, it was not possible to separate the effects of aneurysmectomy from those of selective exclusion of the arrhythmogenic foci in these alterations. However, these changes appear to be related to the noninducibility of VT. Others have investigated the effects of surgery on VT and late potentials. In patients who lost their late potentials after surgery, Marcus et al. found that VT was no longer inducible but that VT was also no longer inducible in about half the patients who retained late potentials. However, the three-lead recording technique did not allow detection of the changes in the spatial distribution of late potentials we have observed.

The spatial distribution of late potentials would seem to be related to the site of origin of VT determined at the time of surgery. Distant extrema were found on the maps of two patients with inferior myocardial infarction and posterobasal VT sites and on the maps of two patients with anterior myocardial infarction and septal VT sites. In contrast, close extrema were found for two patients with anterior myocardial infarction and VT sites that were anterior and posterobasal. As previously noted, the distance between the body surface extremum is linked to the distance separating the torso surface and the cardiac sources. This would suggest that if late potentials are generated near the site of origin of VT, anterior and apical sources that are near the torso surface would produce close extrema, whereas deeper basal and septal sources would produce distant extrema. Others have investigated the relationship between the VT site of origin and the areas showing fragmented and/or delayed electrograms. Kienzle et al. found a good correlation (80%) between the sites of origin of VT and areas of fragmented activity; however, the area with the most delayed activity in sinus rhythm did not always correspond to the site of origin of VT.

**Clinical implications.** Body surface potential maps show that spatial distributions of late ventricular potentials in patients with coronary artery disease and VT are dipolar. This demonstrates that late potentials can be detected reasonably well with only three orthogonal leads but that body surface potential maps provide additional information about their location and extent. After antiarrhythmic medication, signal-averaged electrocardiograms show modifications in QRS duration, but the spatial distributions of the late potentials remain the same, suggesting changes in the degree but not in the sites of the conduction delay.

After antiarrhythmic surgery, the body surface maps revealed changes in both the QRS duration and the morphology of the potential distribution at QRS offset, suggesting a displacement of the terminal activity. Based on our limited observations of six patients, it would seem possible that the spatial distribution of the late potentials, in particular the distance between the extrema, may be useful in locating the site of origin of VT.

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