Spatial Domain Analysis of Late Ventricular Potentials
Intraoperative and Thoracic Correlations

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For investigation of late potentials seen on the signal-averaged electrocardiogram, intracardiac and thoracic distributions of terminal activity were analyzed in 16 patients undergoing cryosurgery for ventricular tachycardia after remote myocardial infarction. The body surface potentials measured with 63 time-averaged unipolar leads were compared with epicardial and endocardial potential maps in six patients without and 10 patients with bundle-branch block. Intracardiac post-QRS activity, defined as extending beyond the thoracic QRS offset, was found in five of six patients without bundle-branch block (83%) and in five of 10 patients with bundle-branch block (50%), corresponding to 4±5% of the total number of electrograms in each patient. Fragmentation, double deflections, and single deflections were observed in 27%, 34%, and 39%, respectively, of these post-QRS electrograms. Post-QRS activation patterns that were stable from beat to beat showed slow propagation around or within areas of conduction block. Post-QRS activity was most often observed on both epicardial and endocardial surfaces (five of 10 patients). In the six patients without post-QRS activity, an area of late activity displaying low-amplitude deflections that were masked by the terminal activation of the normal myocardium was identified. Isopotential maps of the high-pass–filtered (55-Hz) thoracic and intracardiac signals demonstrated a close spatial correlation between the location, amplitude, and orientation of the potential extrema observed over the thoracic, epicardial, or endocardial surfaces during post-QRS activity. The thoracic patterns were generally dipolar with close extrema for anteroseptal or apical sites of post-QRS activity and more distant extrema for other sites. We concluded that the spatial domain analysis of intracardiac and thoracic potential distributions contributes to the understanding of the electrogensis and electrocardiographic measurement of late potentials. (Circulation Research 1990;66:55–68)

Several studies have established that late potentials can be used as a sensitive marker for the identification of subjects prone to ventricular tachycardia or sudden cardiac death, especially after remote myocardial infarction.1–10 Time-domain2,4 and frequency-domain8,11 analyses of the signal-averaged electrocardiogram have been used to detect these low-level potentials at the end of the QRS complex, but both methods have shortcomings, especially in patients with bundle-branch block.12 Thus, in time-domain analysis late potentials must be sufficiently delayed beyond the end of the QRS complex to be detected, whereas in the frequency domain, the Fourier transform technique does not readily give information concerning the timing of late potentials.

In previous work, we demonstrated that the use of 63 thoracic electrodes instead of three bipolar leads provides additional information about the distribution and extent of late potentials on the torso surface.13,14 Thus, spatial domain analysis constitutes an attractive field of investigation to improve detection of late potentials. In the present study, we attempted to validate this new approach by use of intraoperative data. Analysis of intracardiac terminal activation patterns is of interest since the nature of the electrical sources of late potentials registered on the torso surface remains unclear. It has not yet been established whether thoracic late potentials are the direct expression of fragmented electrical activity described in experimental dog studies15,16 as well as during extracellular recording of superfused preparations of human hearts,17 or the expression of a more organized type of delayed activity. Previous investigators have shown the occurrence of epicardial or endocardial bipolar electrograms with delayed and fragmented
activity in patients with thoracic late potentials, but no data are available concerning the quantitative correlation in the time domain or the space domain between thoracic and intracardiac late activities. Thus, our purpose was fourfold: 1) characterization of the epicardial and endocardial distributions and morphologies of late activities in patients undergoing surgery for ventricular tachycardia; 2) determination of the influence of factors such as location of myocardial infarction, sinus node frequency, and presence of a bundle-branch block on these late activities; 3) determination of the temporal and spatial correlation between the late intracardiac activities and thoracic late potentials registered with body surface potential mapping (BSPM); and finally, 4) assessment of the reliability of the information supplied by BSPM about the localization of late potentials.

Patients and Methods

The study group consisted of 16 of 20 consecutive patients referred to our institution for management of recurrent sustained symptomatic ventricular tachycardia or aborted sudden cardiac death who underwent antiarrhythmic surgery. All of them had coronary artery disease with at least one previous myocardial infarction. One patient had had prior aneurysmectomy 10 years before the study. The patients were separated into two groups: Group 1 consisted of six patients without bundle-branch block, and group 2 consisted of 10 patients with bundle-branch block.

Preoperatively, an electrophysiological study was carried out with patients in the unsedated fasting state after antiarrhythmic medications had been discontinued for at least 48 hours. In four patients receiving amiodarone, the drug had been stopped 11 to 52 days before testing; one patient was evaluated while taking the medication. BSPM was performed during normal sinus rhythm. A standard protocol of ventricular stimulation was then applied, with one to three extrastimuli introduced after trains of eight beats at three different drive cycle lengths and two consecutive sites on the right ventricle. Sustained (i.e., >30 seconds) symptomatic ventricular tachycardia was induced in each patient.

A total of 56 episodes of ventricular tachycardia, corresponding to one to six (mean, 3.5) episodes per patient, were mapped during surgery in the two groups. All patients underwent cryoablation of the endocardial sites of earliest activation during ventricular tachycardia; coronary artery bypass grafting and possible aneurysmectomy were performed when necessary. Antiarrhythmic surgery was performed after unsuccessful medical treatment, defined as one to seven ineffective drug trials (mean, 3.8) per patient. Written and informed consent was obtained for electrophysiological testing, cardiac catheterization, and surgery in each patient. The study protocol was approved by our institutional committee on clinical research. The data on the clinical characteristics of the patients are summarized in Table 1.

Thoracic Data Acquisition

The recording techniques have been described previously and will be summarized here. The body surface potentials were measured with 63 unipolar leads referenced to the Wilson central terminal. The electrodes were mounted on 12 vertical strips with an interelectrode distance of 6 cm, with 43 electrodes on the front and sides of the torso and 20 electrodes on the back. Since previous BSPM investigations have shown that the thoracic distributions of late potentials are mostly dipolar, this electrode array was adequate for the purpose of this study, which was to describe qualitatively the main features of these potential distributions. The electrocardiograms were recorded in sinus rhythm with an integrated system (CORDIC), and were amplified, filtered with a bandwidth of 0.05–200 Hz, sampled at 500 Hz, digitized with a resolution of 2.5 μV, and stored on a hard disk for 52 seconds. The beats were classified into normal and ectopic categories and averaged separately for each category. A cross-correlation technique was used to align the beats before they were averaged. The averaged beats were corrected for baseline shift. Finally, the signals were transferred by a telephone line to a PDP 11/34 computer (Digital Equipment Corporation) for further processing.

Intracardiac Data Acquisition

Intraoperative cardiac mapping was performed during partial or total cardiopulmonary bypass at normothermia during stable sinus rhythm. Simultaneous multiple unipolar recordings were obtained by use of epicardial sock and endocardial balloon arrays. The sock electrode array consisted of 63 evenly spaced (interelectrode distance, 0.9–2.0 cm) unipolar recording contacts mounted on a nylon mesh. The balloon array comprised 32 or 63 unipolar electrodes (interelectrode distance, 0.7–2.4 cm) mounted on an inflatable latex support. The deflated balloon array was passed through a left atriotomy across the mitral valve into the left ventricle, or directly into the left ventricle through a ventriculotomy performed on the aneurysm. The balloon was inflated with a 9% saline solution at a pressure below 25 mm Hg. Projections of left anterior and posterior descending coronary arteries on the sock array and position of the reference mark of the balloon array were noted as anatomical landmarks. In all cases but one, epicardial and endocardial recordings were performed consecutively. In one patient for whom the two recordings were performed simultaneously, a reduced 32-electrode configuration was used for the sock array. In six patients, a sequential probe technique was used for endocardial mapping of the ventricular tachycardia pattern and also for endocardial sinus rhythm mapping.

The unipolar signals were amplified, filtered with a bandpass of 0.05–200 Hz, multiplexed, sampled at 500 Hz, and converted to a 10-bit digital format. Data were stored on a hard disk for 26 seconds and subsequently transferred to the PDP 11/34 computer.
TABLE 1. Characteristics of Population Studied

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**Mean±SD**

- 61±9
- 0.9±1.2
- 0.4±0.9
- 2.2±0.7
- 29±7
- 20±7
- 29±38

Number of diseased vessels was determined by major coronary arteries with at least 75%-diameter stenosis.

MI, myocardial infarction; SCD, sudden cardiac death; NYHA, functional status before surgery per New York Heart Association criteria; LVEF, left ventricular ejection fraction assessed by radionuclide angiography; LVEDP, left ventricular end-diastolic pressure; BBB, bundle-branch block; LAD, left axis deviation; F, female; M, male; Inf, inferior; Lat, lateral; Ant, anterior; R, right; L, left; AL, atypical left; N/A, not available.

**Signal Processing**

A time window comprising one cardiac cycle during normal sinus rhythm was manually selected on epicardial and endocardial recordings. Since the epicardial, endocardial, and thoracic signals were not recorded simultaneously, temporal alignment of these signals was performed as follows: Three signals representing the root-mean-square (rms) value of 63 leads computed at each time instant were displayed on a monitor for the thoracic, epicardial, and endocardial recordings. The QRS complexes of the three superimposed signals were then manually aligned, and the thoracic QRS onset and offset points were selected. This procedure was performed by two independent observers with an agreement of ±2 msec (Figure 1). The differences in the QT intervals that were usually observed on the three sets of recordings resulted from modifications due to thoracic and intracardiac surgical interventions of autonomous tone, ventricular temperature and pressure, heart rate, etc. These differences contrasted with the similar QRS durations found on the three sets of recordings.

For analysis of the intracardiac activation sequence, the local activation time on each electrogram was

**FIGURE 1. Time alignment of root-mean-square signals computed from 63 unfiltered leads recorded at endocardium (A), torso (B), and epicardium (C) during normal sinus rhythm in same patient. Onset and offset arrows indicate beginning and end of QRS complex as manually determined from thoracic unfiltered signal.**
automatically detected at the point of most rapid potential decrease (i.e., the intrinsic deflection). The threshold for local activation was set at -0.5 V/sec. The results were manually edited for correction of any artifacts, acceptance of activation times with slopes having values of -0.5 to -0.2 V/sec, and selection of the latest activation time in cases of multiple deflections. Isochronal maps depicting the cardiac activation sequences were plotted for each selected cycle. The QRS onset determined as in Figure 1 was used as the zero reference time. A polar format was used for both the epicardial and endocardial maps.

**Filtering Technique**

High-pass filtering is the usual signal-processing technique used for extraction of the late potentials from the low-frequency ST segment.\(^2\) The filtering technique proposed by Simson,\(^4\) which has been widely applied, reduces the effects of ringing on the measurement of the QRS duration by application of the filter in a bidirectional manner. The initial part of the QRS is filtered in the forward direction, and the terminal portion of the QRS (including late potentials) is filtered backward. This technique prevents ringing outside the QRS but not within, as can be seen in Figure 2B. A test signal (Figure 2A, left) is filtered backward, and the filter response shows ringing with a first overshoot that is higher than the signal itself. In consequence, the filter output appears time shifted to the left and reversed in polarity. This effect is quite apparent for the real late potentials signal (Figure 2A, right). Since the main objective of this study was the morphological analysis of late potentials, a new high-pass filter that preserves the polarity and timing of the local extrema (Figure 2C) was designed. It consists of a finite impulse response filter obtained by subtraction from the current sam-

**Figure 2.** Comparison of filtering techniques. Left panels show filtering of test signal (a 62.5-Hz sine wave); right panels show filtering of thoracic late potentials recorded during ST segment of one patient. Panel A: Original signal before filtering. Panel B: Response of standard fourth-order Butterworth 25-Hz high-pass filter used in a retrograde manner. Panel C: Output of a finite impulse response high-pass (55-Hz) filter. Deflections of signals are identified by plus and minus signs. In panel B, polarities of deflections are inverted, and output is time shifted compared with original signal. In panel C, polarity and timing of deflections are respected.

**Figure 3.** Identification of epicardial and endocardial post-QRS activities during sinus rhythm in a patient without bundle-branch block (group 1) (QRS duration, 98 msec). Onset and offset arrows indicate beginning and termination of unfiltered thoracic QRS (time aligned with intracardiac signals as described in Figure 1). Top Left: Isochronal map of 63 unipolar epicardial leads. A double epicardial breakthrough is visible with right lateral and posteroseptal locations. Bottom Left: Isochronal map of 63 endocardial leads. Isochronal lines are traced at 10-msec intervals. Apex is at center and base is along circumference. Thoracic QRS onset is used as zero reference time. Right: Selected unfiltered epicardial and endocardial electrograms displaying post-QRS activities in apicalateral area. Numbers on these signals indicate latest activation times. Dotted areas represent scar tissue. Patient is number 15 in Table 1. EPI, epicardial map; ENDO, endocardial map; LAD, left anterior descending coronary artery; PDA, posterior descending coronary artery; A, anterior; L, lateral; P, posterior; S, septal.
TABLE 2. Identification of Endocardial and Epicardial Post-QRS Activities

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Mean±SD 123±21 4.0±4.7 6.0±7.6 4.3±4.6 145±36 130±33

Ventriculotomy refers to endocardial mapping performed through a ventriculotomy. QRSD, duration of unfiltered thoracic QRS; EPI, epicardial; ENDO, endocardial; Ant, anterior.

ple of the output of a centered 30-msec Blackman-Harris window. In simpler terms, the average voltage of the preceding and following time instants is subtracted from the current sample. Thus, slow repolarization waveforms are attenuated because each sample is similar to its neighbors, and late potentials are extracted since they stray from the local average. The high-pass cutoff frequency of this filter is 55 Hz and results from a compromise: A higher frequency would have attenuated the amplitude of the late potentials, whereas a lower frequency would have made the filter more sensitive to the proximity of the QRS complex. As can be seen in Figure 2C, the polarity and timing are now preserved for both the test signal and the late potentials signal. For the test signal, ringing is still present on both sides of the sine wave (the impulse response is symmetrical and the filter is not directional), but the overshoots are now much smaller than the signal itself. Thus, this filter is more appropriate for the morphological analysis of late potentials than the bidirectional Butterworth filter. However, it is less appropriate than the bidirectional filter for QRS duration measurements since its output could respond to a nearby large QRS signal.

FIGURE 4. Identification of epicardial post-QRS activities during normal sinus rhythm in a patient with nonspecific intraventricular conduction disturbance (group 2) with QRS duration of 140 msec. Breakthrough of activation occurs on right ventricle at 30 msec. Abnormal electrograms are located along a strip crossing the scar tissue. Onset and offset arrows indicate beginning and termination of unfiltered thoracic QRS. Patient is number 4 in Table 1. LAD, left anterior descending coronary artery; PDA, posterior descending coronary artery.
This filtering technique was applied to the thoracic, epicardial, and endocardial signals. After filtering of the multiple leads, the data were presented as isopotential maps where isocontour lines join points having the same filtered potential at a specific instant. For the thoracic data, the maps were plotted in a rectangular format with the left half corresponding to the front of the torso and the right half to the back. For the epicardial or endocardial data, the maps were presented in a polar format as for the isochronal maps. In addition, the rms value of the 63 filtered leads was computed at each time instant for the thoracic, epicardial, and endocardial data for investigation of the temporal relation between late activity observed on those three recording surfaces.

Statistical Analysis

The results for each variable are given as the mean±1 SD. The statistical analysis was performed with Student's t test for either paired or unpaired observations.

Definitions

Bundle-branch block was defined as a QRS duration ≥120 msec on the standard ECG; hence, this definition included complete right or left bundle-branch blocks as well as nonspecific intraventricular conduction disturbances, usually consisting in terminal slurring of the QRS and regrouped with left bundle-branch blocks in the rest of the study. Left axis deviation was defined as a QRS axis ≤−30°.

Post-QRS activity was defined on the unipolar epicardial and endocardial electrograms as depolarization extending beyond the unfiltered thoracic QRS offset (Figure 1), including the fast waveforms occurring after the intrinsic deflection. With such a definition, in some electrograms displaying post-QRS activity, the local activation time was set before the offset of the thoracic QRS (see Figure 3, two bottom electrograms).

As will be seen in “Results,” the onset of the post-QRS activity could be traced back within the
## #13

SCL = 758 ms

[Diagram showing QRS complex with onset and offset marks, LAD, and PDA]

SCL = 681 ms

[Diagram showing QRS complex with onset and offset marks, LAD, and PDA]

**FIGURE 6.** Influence of a slight variation in sinus node frequency on post-QRS activity: epicardial activation at two different cycle lengths in a patient without bundle-branch block (QRS duration 104 msec). Upper panel: Isochronal map, two electrograms displaying post-QRS activities 110 and 112 msec after QRS onset (double electrogram), and a reference electrogram are shown at sinus cycle length of 758 msec. Lower panel: Decrease of cycle length from 758 to 681 msec is associated with shortening of activation time of post-QRS activities from 110 to 102 msec and 112 to 100 msec, whereas timing of reference electrograms remains unchanged. Onset and offset arrows indicate beginning and termination of unfiltered thoracic QRS. Patient is number 13 in Table 1. SCL, sinus cycle length; LAD, left anterior descending coronary artery; FDA, posterior descending coronary artery.

QRS complex by determination on the intracardiac isochronal and isopotential maps of the earliest instant when the post-QRS pattern and the normal activation pattern were clearly separated. Thus, late activity was defined as the localized abnormal activity occurring after this earliest instant.

## Results

**Intracardiac Data**

Post-QRS activities were found at the epicardium and/or the endocardium in five of six patients in group 1 and five of 10 patients in group 2, corresponding to 4±5% of the total number of electrograms in each patient (see Table 2). Among the electrograms showing post-QRS activity, 27% displayed fragmentations (i.e., multiple deflections), 34% displayed double deflections, and 39% displayed a single deflection (a rapid biphasic deflection following a wide QS potential). These three types of electrograms are depicted in Figures 3, 4, 6, and 7, with small arrows indicating the deflections. The peak-to-peak amplitude of these deflections was 3.2±3.2 mV with a maximum negative slope of -2.3±3.7 V/sec. The percentages of electrograms presenting post-QRS activity were slightly higher at the endocardium than at the epicardium (6.0±7.6% vs. 4.0±4.7%) and in patients from group 1 than from group 2 (5.3±3.4% vs. 3.7±5.1%), but these differences were not statistically significant. The activation times of these post-QRS activities were similar between two consecutive cycles of ventricular activation during stable sinus rhythm (i.e., without variation of the RR interval >2 msec).

On isochronal maps, post-QRS activities appeared to be located in a single left ventricular region in each patient. In five patients, post-QRS activities were observed both epicardially and endocardially, with the two sites close to each other. Figures 3 and 4 depict typical results of epicardial and endocardial maps in patients from the two groups. Electrograms showing only broad QS waves without intrinsic deflection were recorded over scar tissue. Figure 5 shows that the areas and distributions of post-QRS activity between epicardium, endocardium, and scar varied from one patient to another even in patients with similar scars. Apical or apicolateral locations of post-QRS activity were seen in anterior and also in inferior infarcts. Furthermore, the analysis of the activation patterns demonstrated different directions and velocities of propagation despite identical locations. In two cases, the pattern revealed post-QRS activities along a strip crossing the scar tissue.

The influence of limited variations in the sinus node frequency on the measurements was assessed in seven patients in whom a second acquisition had been performed at a slightly faster rate. In five of these seven patients (one from group 1 and four from group 2) for whom cycle length decrease was less than 15%, the activation patterns were identical. In the two other patients from group 1, differences were observed. In the first case (Figure 6), a rate increase from a sinus cycle length of 758 msec to 681 msec was associated with changes in the activation pattern of the anterobasal part of the two ventricles and with a shortening of about 10 msec in the activation times of post-QRS activity. The second case presented a decrease of the sinus cycle length from 676 to 517 msec that was associated with a shift of the latest epicardial activity to an adjacent electrode without modification of the morphology of the activation pattern or of the activation times.

Activation patterns were analyzed in patients in whom intracardiac recordings did not display post-QRS activity. Figure 7 depicts the epicardial activation in one of these patients from group 2 with a left bundle-branch block. The activation of the normal myocardium is ending in the laterobasal area. The thoracic QRS duration is 146 msec, and the latest activation time found on the left side of the heart is 132 msec. The analysis of the signals originating from the anterolateral edge of the scar reveals low-
amplitude signals with double deflections and isochronal lines close to each other. The activation times of this area are between 120 and 142 msec, but all activities end before the QRS offset and are of a low amplitude compared with the electrograms originating from the laterobasal part of the left ventricle.

These conduction abnormalities occurring within the limits of the QRS were interpreted as late activity masked by terminal activation of the normal myocardium usually caused by bundle-branch block. Such late activity was observed in the five patients from group 2 and in the one patient from group 1 in whom no post-QRS electrograms had been recorded. Endocardial data were not available in four of these patients. The late activity was located epicardially in five patients and endocardially in one patient. It was characterized by electrograms having amplitude and maximum negative slope of 3.7±3.2 mV and −2.3±2.5 V/sec, respectively. These values were not different from those observed in post-QRS activities. Multiple, double, and single deflections were obtained in 30%, 30%, and 40%, respectively, of a total of 17 electrograms recorded in the six patients. The relations between these electrograms and scar tissue were identical with those described for post-QRS activity.

By use of isopotential maps of the filtered electrograms and isochronal maps, the onset of late activity was determined as the earliest instant when the late activity pattern and the normal myocardium activation pattern were clearly separated. Two typical examples are presented in Figure 8. Figure 8A shows a sequence of maps for a patient from group 2 with a left bundle-branch block. A specific pattern (arrows) clearly emerges at 116 msec and extends beyond the end of the QRS, thereby indicating the presence of post-QRS activity. Figure 8B represents the same phenomenon in a patient without bundle-branch block. A specific pattern (arrows) appears at 76 msec, again demonstrating the presence of post-QRS activity. Figure 8C displays the late activity onset time for patients with and without left bundle-branch block. This instant occurred significantly earlier in patients without left bundle-branch block than in patients with this condition: 75±4.7 msec vs. 101±12.3 msec (p<0.001). The two patients with right bundle-branch block are not presented in the diagram; their abnormal activation started before 90 msec. The duration of late activity was also compared between patients with and those without left bundle-branch block, and the difference was not significant (60.1±32.4 msec vs. 44±13.2 msec). Analyzing the results of the five patients in whom post-QRS activities were found both epicardially and endocardially, we determined that activation of the abnormal area began 4±4.2 msec later at the epicardium than at the endocardium, but this difference was not statistically significant.

**Correlation With Thoracic Data**

At first, we compared the filtered intracardiac rms signals with the corresponding filtered rms thoracic signal. The three types of results are illustrated in Figure 9. In the lower panel, the endocardial post-QRS activities are in temporal agreement with thoracic late potentials. In the two other panels, despite the correct time alignment of the beginning of the QRS, thoracic and intracardiac post-QRS activities are not concomitant. On the left panel, epicardial post-QRS activities exceed in duration the thoracic late potentials. Conversely, on the right panel, late potentials cannot be related to any intracardiac post-
FIGURE 8. Determination of onset of late activity within QRS. Panel A: Sequence of epicardial filtered isopotential maps in patient with left bundle-branch block (QRS duration 142 msec). Panel B: Similar sequence of maps in patient with normal QRS duration (98 msec). Time values beside each map are referred to thoracic QRS onset. Intervals between isopotential lines are 5, 2, 2, and 0.5 mV from top to bottom in panel A and 0.5, 0.5, 0.2, and 0.2 mV in panel B. A separate activation pattern (arrow) emerges at 116 msec in panel A, extending beyond QRS offset and clearly indicating presence of post-QRS activity. Same phenomenon is visible in panel B, with beginning of a separate activation at 76 msec extending beyond QRS offset. Panel C indicates an earlier onset of late activity in patients without than in patients with left bundle-branch block. Patients are numbers 3 and 15 in Table 1; LAD, left anterior descending coronary artery; PDA, posterior descending coronary artery; QRS offset, end of filtered thoracic QRS; LBBB, left bundle-branch block; BBB, bundle-branch block.

QRS activity. These discrepancies could be due to variations in the sinus cycle length that change the coupling interval between post-QRS activities and the beginning of the QRS (Table 3).

For comparison of the thoracic and intracardiac post-QRS activities, the data were realigned by matching the thoracic with the corresponding epicardial and endocardial filtered isopotential maps. Late activity features such as changes in polarities, changes in the orientation of the extrema, and maximum and minimum potentials were time aligned on the different map sequences. In all cases except one (Table 3), this realignment was of 6 msec or less. In Figure 10, the thoracic map sequence was shifted by 2 msec so that the timing of the extrema coincided with that of the epicardial and endocardial data. The potential distributions over the three recording surfaces are in good agreement. Both the epicardial and endocardial maps show that terminal activation is located in the laterobasal part of the left ventricle. This activation is oriented with maxima that are anterior with respect to the minima. This is reflected on the body surface with positive potentials covering the anterior part of the torso and negative potentials over the back.

In the five patients of group 1 and in three of the five patients of group 2 in whom epicardial or endocardial post-QRS activities had been observed, we obtained terminal BSPM patterns that well reflected the intracardiac post-QRS distribution. In seven of these eight patients, a single monomorphic thoracic pattern was observed despite variations in its amplitude. In the other patient, two morphologically distinct patterns were consecutively obtained beyond the end of the unfiltered thoracic QRS and were associated with a large displacement of terminal activation (Figure 4). A phenomenon of polarity reversal due to the high-pass filtering of sudden changes in the signal amplitude was simultaneously observed on the intracardiac and thoracic maps in six patients. We examined the specificity of the thoracic maps with respect to the intracardiac site of post-QRS activity (Figure 11). Two main types of patterns could be identified: 1) a type with close extrema that corresponded to apical, anteroseptal, and anterolateral sites, and 2) type with distant extrema that corresponded either to inferior or laterobasal sites, which were characterized by a horizontal or vertical zero potential line, respectively. Differences in BSPM patterns for nearby intracardiac sites in different patients could be attributed to differences in the orientation of the activation. In two of the five patients in whom post-QRS activities were found both epicardially and endocardially, the thoracic pattern was related both to the epicardial and the endocardial patterns, which were similar (Figure 10). In the three other patients, the thoracic pattern was related to the prevailing intracardiac pattern according to the relative amplitude of the epicardial and endocardial signals.

In two of the five patients of group 2 with post-QRS activities, the thoracic map reflected the delayed activation of the normal myocardium induced by the bundle-branch block, without any relation with the late activities. In these two patients, the latest thoracic map was inscribed 12 and 14 msec after QRS offset, whereas in the three other patients in whom the thoracic pattern correlated with post-QRS activities, this delay was 24, 26, and 98 msec. Because of
the nonsimultaneity of the thoracic and intracardiac recordings, changes in the coupling interval between QRS and post-QRS activity could explain these observations. Also, this effect can be attributed to the characteristics of our filter, whose response is altered by the proximity of the QRS complex in the range of 10–14 msec. In the five other patients of group 2 without post-QRS activities, the terminal thoracic maps were also related to the bundle-branch block activation sequence. Three different types of thoracic patterns reflecting bundle-branch block activation were identified (Figure 12). A first type, characterized by extrema located in the right superior part and the left inferior part of the anterior torso, corresponded to right bundle-branch block. The other patterns, related to left bundle-branch blocks, were characterized by distant extrema, one over the front and the other over the back of the torso, with an oblique or vertical zero potential line depending on the presence or absence of a left axis deviation.

The analysis was completed by correlation of the rms signals after the time shift obtained from com-

![Figure 9](http://circres.ahajournals.org/)

**Figure 9.** Intracardiac and thoracic filtered root-mean-square signals with three types of temporal correlation. Top Left: Epicardial post-QRS activities are exceeding in duration the thoracic late potentials (arrows). Top Right: Despite thoracic activity extending beyond unfiltered QRS offset (arrow), no post-QRS activity is visible on intracardiac recordings. Bottom: Endocardial post-QRS activities are in time agreement with thoracic late potentials (arrows). Panel A: Root-mean-square signal of 63 unipolar filtered epicardial electrograms. Panel B: Root-mean-square signal of 63 unipolar filtered epicardial electrograms. Panel C: Selected unfiltered epicardial channel displaying late activity. Panel D: Root-mean-square signal of 32 unipolar filtered endocardial electrograms. Panel E: Selected unfiltered endocardial channel displaying post-QRS activity. Onset and offset arrows indicate beginning and termination of unfiltered thoracic QRS (time-aligned with intracardiac signals as described in Figure 1). Patients are numbers 4, 5, and 10 in Table 1. RMS, root-mean-square; EPI, epicardial; END0, endocardial.

### Table 3. Comparison Between Thoracic and Intracardiac Root-Mean-Square Filtered Signals After Map Alignment

<table>
<thead>
<tr>
<th>Patient number</th>
<th>Thoracic RRI (msec)</th>
<th>EPI RRI (msec)</th>
<th>ENDO RRI (msec)</th>
<th>Thorax vs. EPI</th>
<th>Thorax vs. END0</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Amp.</td>
<td>t (msec)</td>
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<tr>
<td>Group 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>660</td>
<td>614</td>
<td></td>
<td></td>
<td>24.8</td>
</tr>
<tr>
<td>5</td>
<td>960</td>
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</tr>
<tr>
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<td>690</td>
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<tr>
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</tr>
<tr>
<td>3</td>
<td>928</td>
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<td>518</td>
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<td>530</td>
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<td>14</td>
<td>784</td>
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RRI, mean RR interval during signal acquisition; EPI, epicardial; END0, endocardial; Amp., amplitude ratio between epicardial or endocardial post-QRS activities and thoracic late potentials; t, time adjustment between the two root-mean-square signals obtained from comparison of maps; r, correlation coefficient corresponding to linear regression between the two root-mean-square signals.
Discussion

Comparison of the potential distributions recorded with multiple unipolar leads over the torso, the epicardium, and the endocardium provides additional information about the electrogensis of the late potentials observed on the signal-averaged ECG. In summary, we observed that post-QRS intracardiac activity that is stable from beat to beat, propagates slowly in a relatively organized manner around or within regions of scar tissue, and generates epicardial and endocardial potentials in the millivolt range with small and delayed RS deflections, at times doubled or multiphasic, produces potential distributions on the thoracic surface that are mainly bipolar with amplitudes that are typically reduced 100-fold with respect to the intracardiac signals. The BSPM patterns were specific to the site and orientation of this post-QRS activity. In all the patients without post-QRS activity, an area around or within the scar tissue displaying similar electrograms with small and late deflections was identified. Obviously, thoracic and intracardiac potential distributions cannot be recorded simultaneously, and the electrophysiological changes that may occur between the recording sessions constitute a basic limitation for similar studies.

Previous studies have emphasized the fragmented nature of the intracardiac electrograms during normal sinus rhythm in patients with ventricular tachycardia and late potentials on the signal-averaged ECG. We also detected fragmentation during post-QRS activity, but a more organized type of activation with single or double RS deflections was more prevalent. In some patients, this organization was also reflected on the isochronal and isopotential maps, which showed activation fronts moving along a constant direction (Figure 4) or generating stable BSPM patterns (Figure 10). Similarly, Klein et al have also reported that delayed activation, either single or double, as well as fragmentation, is a characteristic finding of sinus rhythm mapping in patients with ventricular tachycardia. When compared with our unipolar recording technique, the bipolar electrodes and analog bandpass filters that were used in the other studies could reduce signal amplitude, produce ringing, and, finally, accentuate the fragmentation.

Like other investigators, we found that post-QRS activity was located in well-localized areas in the proximity of scar tissue. Both the epicardial and endocardial regions were involved, and our results support the role of the subepicardial region in the electrogensis of late potentials, which has been questioned in some studies. However, the lack of standard definitions may explain the wide range (from 10% to 95%) of prevalence of epicardial delayed activity found in patients with ventricular tachycardia.

Beat-to-beat variability and decremental conduction in abnormal areas are phenomena well described in the experimental and clinical literature. We have not observed beat-to-beat variations during stable sinus rhythm in any of our patients. This finding suggests that the phenomenon may be rare and, thus, validates the use of averaging techniques.

Figure 10. Terminal intracardiac and thoracic filtered isopotential maps in a patient with QRS duration of 104 msec. Panels A, B, and C represent endocardial, epicardial, and thoracic data, respectively. Epicardial and endocardial maps have same polar format as in preceding figures. Torso surface is represented with a rectangular format: left part of map corresponds to front of torso and right part to back. Interval between isopotential lines is indicated at bottom of each sequence of maps. Time value indicated over each map is with respect to thoracic QRS onset (see Figure 1). Plus and minus signs indicate positive and negative extrema. Zero potential line is identified by a heavier trace. A time adjustment of 2 msec between intracardiac and thoracic sequences was made so that maximum filtered values would occur simultaneously. Potential distributions over the three surfaces are in good agreement, showing a terminal activation in laterobasal part of left ventricle. Patient is number 13 in Table 1. A, anterior; L, lateral; P, posterior; S, septal; LAD, left anterior descending coronary artery; PDA, posterior descending coronary artery.
However, shorter RR intervals might induce alternans. Our results indicate that a cycle length decrease of over 15% is usually required for induction of changes in the coupling interval between late potentials and QRS of up to 20 msec (see Table 3). However, like other investigators, we reported two examples (Figure 6) where limited variations in basic rate had similar effects. Also, we have shown variations in coupling between QRS and late potentials that cannot be uniquely attributed to alterations in propagation in the areas of slow conduction, but may be due to modifications affecting ventricular depolarization in a global fashion (Figure 6).

Mapping of late activity within the QRS leads to a wider concept of late potentials based on localized areas displaying electrograms whose morphology is altered and amplitude and slope are reduced. The identification of such late activity within the QRS limits in all our patients without post-QRS activity suggests that the lack of thoracic late potentials sometimes reported in patients with ventricular tachycardia may be attributed to a masking effect of the QRS and not to an insufficient amplitude or to alternans precluding the averaging technique. Furthermore, since late activity begins about 25 msec later in patients with left bundle-branch block than in those without it, and since it has a similar duration in both cases, the lower prevalence of post-QRS activity in patients with left bundle-branch block implies that the masking effect of the widened QRS is predominant, despite this delayed onset. Thus, these data may have implications for the development of techniques for detection of late potentials during the QRS, notably in patients with bundle-branch block.

The organized nature of epicardial and endocardial activity generating thoracic late potentials warrants the registration of monomorphic terminal patterns of the torso surface, as reported in seven of eight of our patients. As previously noted, these patterns are dipolar, and the distance between the

**Figure 11.** Nine body surface potential mapping patterns recorded in eight patients during terminal activation and corresponding endocardial (right) and epicardial (left) sites of post-QRS activity. Anterior, apical, and anterolateral patterns showing close extrema can be distinguished from inferior and lateral patterns characterized by distant extrema. Polar format of maps as in preceding figures. Interval between isopotential lines is 2 µV in all cases. ANT-SEP, anteroseptal; API, apical; ANT-LAT, anterolateral; LAT, lateral; INF, inferior; LAD, left anterior descending coronary artery; PDA, posterior descending coronary artery.

**Figure 12.** Morphological analysis of thoracic patterns depicting terminal activation in patients with bundle-branch block (group 2). Panel A: Patterns related to intracardiac post-QRS activity. Panel B: Patterns reflecting right bundle-branch block terminal activation. Panel C: Patterns related to left bundle-branch block with normal QRS axis. Panel D: Patterns corresponding to left bundle-branch block with left axis deviation. Interval between isopotential lines is 2 µV in all cases. All 10 patients in group 2 are represented.
thoracic extrema is linked to the distance separating the torso surface and the cardiac source. In comparison with the standard time domain and frequency domain techniques or more recent spectrotomographic techniques, spatial analysis could be useful for the detection of late potentials, especially in patients with bundle-branch block. This spatial delineation of late potentials is based on the identification of terminal activation patterns different from those related to bundle-branch block activation sequences (Figure 12). Limitations exist for such a technique; for example, the activation pattern of late potentials in the left laterobasal area (Figure 11) could not be distinguished from normal myocardium activation pattern in left bundle-branch block with normal QRS axis (Figure 12C). The relevance of application of the BSPM technique for localization of the arrhythmogenic substrate in patients who will undergo antiarrhythmic surgery remains to be demonstrated.

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