Body Surface Mapping of High-Frequency Components in the Terminal Portion During QRS Complex for the Prediction of Ventricular Tachycardia in Patients With Previous Myocardial Infarction

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To study the clinical significance of terminal QRS high-frequency components for the prediction of ventricular tachycardia, an 87-lead body surface signal-averaged mapping was performed in 21 healthy subjects (control) and in 41 patients with previous myocardial infarction (anterior, 20; inferior, 21). Mapping data were analyzed and averaged (129.7±26.5 beats) for 160 seconds, and the signal-averaged beat was filtered with a bidirectional bandwidth (80–250 Hz) digital filter. J-point was determined from the 87-lead RMS voltage of nonfiltered QRS. For each lead, we calculated the sum of the absolute value of filtered QRS from 20 msec ahead of the J-point to the J-point (J–A–20). The body surface distribution of A–20 was expressed as A–20 map. The maxima in A–20 maps were mainly located on the upper sternal region in healthy subjects, on the left anterior chest in patients with previous anterior myocardial infarction, and on the central anterior chest in patients with previous inferior myocardial infarction. In the patients in both the group with anterior myocardial infarction and the group with inferior myocardial infarction, the value of maximum was significantly greater than in the subjects in the control group (0.181±0.086 and 0.138±0.048, respectively, vs. 0.075±0.031 mV·msec; p<0.01). In patients with myocardial infarction (n=41), the value of maximum was significantly greater with ventricular tachycardia (n=11) than without ventricular tachycardia (n=30) (0.240±0.076 vs. 0.130±0.043 mV·msec; p<0.01). It was suggested that the maximum value of A–20 map is useful for the prediction of ventricular tachycardia in myocardial infarction. (Circulation 1990;82:2084–2092)

Many investigators have reported that low-amplitude high-frequency components observed at the end of QRS complex, termed “late potentials,” are associated with ventricular arrhythmias in coronary artery disease.1–24 Body surface distributions of such components, however, have not been investigated satisfactorily.19 Generally, late potentials are seen in the early portion of ST segment, and are continuous from the end of QRS complex. Some late potentials, however, may appear only in the terminal portion of QRS complex, which has not been examined. To evaluate body surface distributions of terminal QRS high-frequency components, we developed an 87-lead signal-averaged mapping system. Using this system, we analyzed high-frequency components of the terminal portion during QRS complex in healthy subjects and patients with previous myocardial infarction (MI). Then we investigated the clinical significance of these components for the identification of patients with ventricular tachycardia (VT).

Methods

Subjects
The control group consisted of 21 healthy subjects (age range, 20–56 years; mean±SD, 28.0±9.0 years) who had no history of heart diseases and had normal physical examinations, normal standard 12-lead electrocardiograms (ECGs), and normal echocardiograms.
Forty-one patients with previous MI were studied. Each patient had a documented transmural MI that was more than 4 weeks old. None had bundle branch block or intraventricular conduction disturbances on ECG. According to the location of infarcts, patients were divided into two groups, 20 patients with anterior MI (AMI) (age range, 41–81 years; mean ± SD, 58.6 ± 10.1 years) and 21 patients with inferior MI (IMI) (age range, 38–79 years; mean ± SD, 59.3 ± 9.3 years). Eleven patients had at least one documented episode of spontaneous VT, four patients with sustained VT and seven patients with nonsustained VT. VT was defined as ventricular premature contractions more than five beats per run.

**Body Surface Electrocardiographic Recording**

Eighty-seven unipolar lead body surface ECGs were recorded with a VCM-3000 system (Fukuda Denshi, Tokyo) during normal sinus rhythm. Eighty-seven body surface leads were arranged in a lattice-like pattern (13 × 7 matrix), except for four lead points in the midaxillary lines, and covered the entire thoracic surface (anterior, 59; back, 28). ECGs from 87 unipolar leads were sampled simultaneously with Wilson's central terminal as reference. This system consisted of amplifiers that had a frequency response between 0.05 and 500 Hz, the analog-to-digital converter (12-bit samples, 5 μV/bit), and a sampling interval of 1 msec. All subjects were kept relaxed in the supine position at the time of recording.

By using the VCM-3000 system, body surface ECG data were sampled for 8 seconds and recorded on a 1-MB floppy disk. This procedure was repeated 20–40 times. Data recorded on 20–40 floppy disks were transferred to a PC-9801 VX4 (NEC, Tokyo, Japan) and stored on a hard disk unit (total 20–40 MB). The stored data were copied on a cassette tape and later analyzed. The results of the analysis were printed by a laser printer (Canon, Tokyo) (Figure 1).

**Signal Averaging and Digital Filtering**

The following analyses were performed by a 16-bit personal computer (PC-9801 VX4, NEC). One lead

![Diagram of the 87-lead body surface signal-averaged mapping system.](image)

**FIGURE 1.** Diagram of the 87-lead body surface signal-averaged mapping system.

![Illustration of the last 20-msec area. The QRS onset and offset were determined from the 87-lead nonfiltered RMS voltage. The upper panel illustrates nonfiltered QRS, and the lower panel illustrates filtered QRS (high-frequency components) with a bidirectional bandwidth of 80–250 Hz. In the lower panel, the shaded area shows the sum of the absolute value of filtered QRS from 20 msec ahead of the J-point to the J-point (last 20-msec area, A–20). RMS, root mean square.](image)

**FIGURE 2.** Illustration of the last 20-msec area. The QRS onset and offset were determined from the 87-lead nonfiltered RMS voltage. The upper panel illustrates nonfiltered QRS, and the lower panel illustrates filtered QRS (high-frequency components) with a bidirectional bandwidth of 80–250 Hz. In the lower panel, the shaded area shows the sum of the absolute value of filtered QRS from 20 msec ahead of the J-point to the J-point (last 20-msec area, A–20). RMS, root mean square.
on the left anterior chest (lead G4) was used for the processing of signal averaging. We selected a template beat, and all subsequent beats were tested against the template. A 75% point of maximal dV/dt in the template beat (X) and that in a successive beat (X') were selected for the temporary reference point. We moved X' from (X−10) msec to (X+9) msec (total, 20 points). Then, 20 correlation coefficients between the template beat and a subsequent beat were calculated by using 160 pairs of potential data from (X−75) msec to (X+84) msec. The signal averaging was performed at the time when the highest correlation coefficient was attained. Each lead was filtered with a bidirectional, bandwidth (80–250 Hz, 6 dB per octave) digital filter described by Pynsent and Hanka.29 A beat was excluded from the averaging when the highest correlation coefficient was less than 0.990, or when the RR interval between the beat and the former beat changed more than 20% compared with between a template and the former cardiac beat.

Last 20-msec Area Map

The QRS onset and offset were determined visually from the 87-lead root-mean-square (RMS) voltage of nonfiltered QRS. In Figure 2, the upper panel illustrates nonfiltered QRS and the lower panel illustrates filtered QRS (QRS high-frequency components). In the lower panel of Figure 2, the shaded area shows the sum of the absolute value of the filtered QRS from 20 msec ahead of the J-point to the J-point (last 20-msec area, A−20). For each lead, A−20 was calculated. The body surface distribution of A−20 was expressed as the last 20-msec area map (A−20 map). Late potentials were defined as high-frequency components extending beyond the nonfiltered QRS offset.

Statistical Analysis

Quantitative data were expressed as the mean±SD. Statistical differences between the three groups for maximum value and location of the maxima were examined by the unpaired t test and nonparametric Wilcoxon rank sum test, respectively. Significance refers to a p value less than 0.05.

For the use of the nonparametric Wilcoxon rank sum test, horizontal mapping lead positions A, B, C, . . . , and M were assigned to 1, 2, 3, . . . , and 13, respectively. Then, the mean±SD of vertical and
horizontal position numbers for the maxima of A–20 map was calculated in each group.

**Results**

The number of beats averaged was 130.4±14.5 in the subjects in the control group, 124.0±36.2 in the group of patients with AMI, and 134.4±23.7 in the group of patients with IMI, and the difference was not significant among these groups.

Figure 3 (upper left, lower left, and right panels) shows examples of high-frequency components that occur during the terminal QRS. The upper and lower parts of each panel show nonfiltered (original) and filtered waveforms, respectively. The onset and offset arrows indicate beginning and end of the QRS determined from nonfiltered RMS voltage. The upper left, lower left, and right panels (Figure 3) show examples of E6 lead waveforms in a normal subject, G4 lead waveforms in a patient with AMI, and E6 lead waveforms in a patient with IMI, respectively. E6, G4, and E6 are leads of maximum values in A–20 maps in the normal subject, the patient with AMI, and the patient with IMI, respectively. In the upper and lower left panels (Figure 3), the high-frequency components exist during QRS and do not occur during the ST segment. In contrast, in the right panel (Figure 3), the high-frequency components extend into the ST segment. In all three panels (Figure 3), the amplitudes and morphologies of the high-frequency components during the terminal 20 msec of QRS are different from each other. Although late potentials did not exist in the ST segment, the patient with AMI in the lower left panel had VT episodes and a maximum value of 0.187 mV · msec in A–20 map. The patient with IMI in the right panel did not have VT episodes. Late potentials, however, existed in the ST segment.

Figure 4 illustrates examples of A–20 maps for each group. The upper map is from a normal control subject. The maximum of 0.076 mV · msec is located on the upper sternal region. The middle panel is from a patient with AMI. The maximum is located on the left anterior chest, and the maximum value is greater (0.133 mV · msec). The lower panel is from a patient with IMI. The maximum of 0.111 mV · msec is located on the central anterior chest.

Figure 5 represents the distributions of the sites of maxima for each group. Open circles indicate subjects in whom maxima are equal or greater than 0.1 mV · msec. In the normal control group, all the maxima were located on the upper sternal region. The sites of most maxima in patients were distributed over the precordial region. In the group of patients with AMI, they were located mainly on the left anterior chest, and in the group of patients with IMI, they were located mainly on the central anterior chest. There was a significant difference in location of maxima between the control group of subjects and the group of patients with AMI in both vertical (6.0±0.6 vs. 4.4±0.7, p<0.001) and horizontal (5.1±0.5 vs. 6.5±1.1, p<0.001) positions. In location of maxima for the group of control subjects and the group of patients with IMI, there was no significant difference in horizontal position. In these groups of patients, however, there was a significant difference in vertical position (6.0±0.6 vs. 4.7±1.0, p<0.001). In location of maxima for the groups of patients with AMI and IMI, there was no significant difference in vertical position. There was a significant difference in location of maxima, however, between the group of patients with AMI and the group of patients with IMI in horizontal position (6.5±1.1 vs. 5.6±0.9, p<0.001).

Figure 6 shows the values of maxima in A–20 maps of the three groups. Open circles indicate patients with VT. In both the group of patients with AMI and the group of patients with IMI, the mean value of the maxima was significantly greater than in the control group of subjects (0.181±0.086 and 0.138±0.048 vs. 0.075±0.031 mV · msec, p<0.01). There was no significant difference in the mean value of maxima between the group of patients with AMI and the group of patients with IMI.

We divided the 41 patients into two groups, 11 patients with VT and 30 patients without VT. Figure 7 shows the values of maxima in A–20 maps of the two groups. Open circles indicate patients with sustained VT. The values of maxima in patients with VT were significantly greater than in patients without VT (0.240±0.076 vs. 0.130±0.043 mV · msec, p<0.01). The values of maxima in patients with sustained VT showed a tendency to be greater than in patients with nonsustained VT.

**Discussion**

The association of low-amplitude high-frequency components in the terminal portion of QRS complex (late potentials) with VT has been reported by many investigators.1–24 These late potentials have been detected by epicardial and endocardial mapping20,22–24 and body surface ECGs1,10,12–19 in patients with VT and in experimental animals.

The purpose of this study was 1) to examine high-frequency components that occur during the terminal QRS by using a new index (A–20), 2) to compare the body surface distributions of A–20 among control subjects and patients with AMI and IMI, and 3) to study the clinical significance for the identification of patients with VT in MI.

**Last 20-msec Area (A–20)**

Although several methods have been developed for analyzing high-frequency ECGs, there is no study to examine the clinical significance of high-frequency components existing exclusively in the last portion of the nonfiltered QRS. Rozanski et al23 examined the waveform activity that existed beyond the termination of nonfiltered QRS. Simson et al5 and others9,10,14,15,24 investigated only filtered QRS, especially its terminal portion, and did not examine the significance of high-frequency components during nonfiltered QRS. Simson et al5 described that when
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Control  K.M. 21y, Man

Anterior MI  A.S. 74y, Man

Inferior MI  H.T. 75y, Man

Front  Back
(interval=5mV-ms)

FIGURE 4. Examples of A–20 maps for each group. The upper map is from a normal control subject. The maximum is located on the upper sternal region. The middle map is from a patient with anterior MI. The maximum is located on the left anterior chest. The lower map is from a patient with inferior MI. The maximum is located on the central anterior chest. Numeral on each map indicates the maximum value. MI, myocardial infarction.

fragmented electrograms were brief in duration, no distinctive signals were identified on the body surface ECG. We also assumed that the beginning of late potentials was within QRS complex. Thus, we calculated the sum of the absolute value of filtered QRS from 20 msec ahead of J-point to J-point of nonfiltered QRS (A–20) for each lead and examined the body surface distributions of A–20. A–20 was considered to reflect the average absolute amplitude of high-frequency components during the 20-msec interval. The present study, different from those of most investigators,1–24,30,31 provided new information about terminal QRS high-frequency components.

It was at first problematic how long we should measure the last phase of QRS complex. We constructed the last 10-, 20-, 30-, and 40-msec area maps, and the values of the maxima in these maps were calculated. The values of the maxima in the last 20-msec area maps showed the best differentiation between the groups of patients with MI (groups of patients with AMI and IMI) and the control group of normal subjects. Therefore, we used the last 20-msec area map.

Distributions of A–20

In 1986, Faugère et al19 reported the spatial distribution of late potentials by 63 unipolar lead body surface mapping in patients with VT. According to their report, the distance between extrema on isopotential maps is linked to the distance between the thoracic surface and the area of myocardium generating the electrical activity.

In the present study, the body surface distributions of A–20 were investigated. The maxima of A–20 maps were located mainly on the upper sternal region in the normal subjects in the control group, on the left anterior chest in the patients in the AMI group, and on the central anterior chest in the patients in the IMI group (Figure 5). It is evident that the maxima in the normal subjects in the control group are derived from the excitation of the pulmonary conus, where the depolarization is most delayed.
The sites of maxima in the patients in the AMI group were located mainly on the left anterior chest and corresponded to just over or the circumscribed area of abnormal Q waves. The sites of maxima in the patients in the IMI group, however, were located mainly on the central anterior chest and did not always correspond to the abnormal Q waves.

**Prediction of Ventricular Tachycardia**

In the previous studies, the presence of late potentials is considered to be a marker for VT and sudden death.\(^1\)\(^{-24}\) The presence of late potentials is clinically useful for the prediction of VT and sudden death. We also examined the relation between the value of maximum in A-20 map and the occurrence of VT.

We demonstrated that patients with VT had significantly greater values of maxima in A-20 maps than patients without VT. The values of maxima for patients with VT were greater than 0.100 mV · msec (Figure 6), and the values of maxima for patients with sustained VT were greater than 0.200 mV · msec (Figure 7). It was concluded that the maximum value in A-20 map is useful for the prediction of VT in patients with MI.

**Conclusion**

Our 87-lead body surface signal–averaged mapping system gave us new information that had not been
obtained by the conventional body surface mapping. The maximum value of A–20 map was considered to be a useful index for the prediction of VT. Further study is required to find a more useful index than the maximum value in A–20 map for the prediction of VT by means of this system.

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References


**KEY WORDS** • signal averaging • body surface mapping • ventricular tachycardia
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