Relation Between Recovery Sequence Estimated From Body Surface Potentials and T Wave Shape in Patients With Negative T Waves and Normal Subjects

Michiyasu Yamaki, MD; Isao Kubota, MD; Takio Endo, MD; Yukio Hosoya, MD; Kozue Ikeda, MD; and Hitonobu Tomoike, MD

Background. Advances in analytical methods of the epicardial electrical potentials allowed us to demonstrate spatial distributions of local recovery. Because local recovery will be reflected in events on body surface ECG mapping, abnormalities in recovery sequence that may be responsible for the origin of negative T waves can be detected from body surface potentials.

Methods and Results. Eighty-seven unipolar ECGs were recorded simultaneously from the entire thorax in patients having negative T waves on left anterior precordial leads and in normal subjects. These included 40 patients with anterior myocardial infarction (MI), 21 patients with left ventricular hypertrophy (LVH), and 44 male volunteers. We measured $T_{max}$ time, defined as the instant of maximal first derivative of the T wave as the index of local recovery (Wyatt’s method). Parameters related to T wave potentials were positive T wave amplitude, negative T wave amplitude, and T integral. Significantly correlations were observed between the $T_{max}$ time and each of the T wave potentials. The T wave potentials were dependent on $T_{max}$ times. In the anterior MI, the late $T_{max}$ times were located on the upper left anterior chest and early $T_{max}$ times on the lower right lateral chest. In the LVH, the area showing a delayed recovery was displaced in a left downward direction compared with anterior MI.

Conclusions. Body surface $T_{max}$ time distributions clearly separate two negative T wave groups, i.e., anterior MI and LVH. Appearance of the negative T waves correlates well with the presence of the area with delayed $T_{max}$ time on the spatial distribution. (Circulation 1992;85:1768–1774)

Key Words • mapping, body surface • T waves • myocardial infarction • hypertrophy

It is accepted that the T wave originates from an inhomogeneous electrical recovery of the ventricle$^{1–3}$ and that T wave inversion is intimately related to the presence of delayed recovery resulting from various pathological states.$^{3,4}$ The presence of myocardial infarction (MI) or left ventricular hypertrophy (LVH) frequently accompanies negative T waves. Although pathological events based on negative T waves are quite different between MI and LVH, differentiation of these diseases solely on the basis of conventional ECGs is occasionally difficult.

A series of experimental$^{5–7}$ and simulation$^{8}$ studies indicated that the time instant of maximal $dV/dt$ in the ST-T segment on epicardial unipolar electrograms closely corresponds to the instant of local ventricular recovery. Namely, the activation recovery intervals defined as the duration between the activation time and the recovery time correspond well with action potential durations during cycle length changes,$^{6,7}$ norepinephrine infusion,$^6$ sympathetic nerve stimulation,$^{6,7}$ and myocardial ischemia.$^7$

Recently, body surface mapping, which can clarify the changes in local electromotive force,$^{9–19}$ has enhanced the diagnostic performance of ECG when it was applied to patients with myocardial infarction,$^{9–13}$ effort angina pectoris,$^{16}$ LV hypertrophy,$^{17}$ or accessory atrioventricular pathways.$^{18,19}$ In the present study, we measured body surface recovery time, which we designated “$T_{max}$ time,” as the index of local recovery. Recovery sequence estimated from body surface potentials was studied in patients with MI and LVH in reference to the characteristics of negative T waves. We were intrigued by the possibilities that the T wave shape depends on the mode of recovery sequence and that the body recovery sequence could differentiate negative T wave in patients between MI and LVH.

Methods

Normal Subjects

Forty-four normal male volunteers aged 22–51 years (mean, 32 years) were studied. None of the volunteers had any history of cardiac disorders or systemic arterial hypertension. All of them showed normal physical examination and 12-lead ECG findings.
Patients With Negative T Waves

Sixty-one consecutive patients with anterior MI or LVH accompanying negative T waves in all leads from V₄ to V₆ were enrolled in the present study. The criterion for the negative T waves was T negativity with amplitude >0.2 mV.

Anterior MI. Forty anterior MI patients aged 35–70 years (mean, 56 years) satisfying the following criteria were defined as the infarction group: 1) a clinical diagnosis of MI accompanying typical chest pain and serum enzyme changes; 2) no other heart disease such as congenital heart disease, myocardial disease, valvular heart disease, or hypertensive heart disease; 3) no conduction disturbances such as bundle branch block or Wolff-Parkinson-White syndrome; and 4) abnormal Q waves in V₂ and V₃ but no abnormal Q waves in II or aVF. All patients in the infarction group underwent diagnostic cardiac catheterization within 2 weeks before or after the map recording.

LVH. The LVH group consisted of 21 patients with increased LV wall thickness (septal thickness ≥13 mm or posterior wall thickness ≥13 mm) on echocardiography, including 19 patients with hypertrophic cardiomyopathy and two patients with hypertensive heart disease. Their age ranged from 16 to 70 years (mean, 48 years), and patients in this group satisfied the following criteria: 1) no other heart disease such as congenital heart disease or ischemic heart disease, 2) no conduction disturbances such as bundle branch block or Wolff-Parkinson-White syndrome, and 3) no enlargement of the right ventricular dimension (>27 mm) as estimated by an M-mode echocardiogram.

All normal subjects and patients with negative T waves gave their informed consent to the procedures before the study commenced.

Body Surface Mapping

Recording. Body surface mapping was done with a body surface potential mapping system, the HPM-5100 unit (Chunichi Denshi Co., Nagoya, Japan). This system consists of a 96-channel amplifier, sample-hold circuit, multiplexer, A-D converter, 48-kbyte random access memory, and eight-bit microprocessor. Resolution of the system was 0.01 mV, with a dynamic range of ±5 mV. Eighty-seven body surface leads were arranged on each patient's body in a lattice-like pattern (13×7 matrix) except for the lead points in the midaxillary line. The body surface leads covered the entire thoracic surface (59 leads located on the anterior chest and 28 leads on the back; Figure 1). ECGs from these 87 unipolar leads with Wilson's central terminal as reference, standard 12-lead ECGs, and Frank X, Y, and Z lead scalar ECGs were recorded simultaneously. Sampling frequency was 250 samples per second per channel, and these data were acquired at the expiratory period state in the resting supine position. When noises such as baseline drift, electromyograms, or 50-Hz AC interference were detected in any of the signals, data acquisition was repeated. The flat portion of the TP segment was defined as the baseline. After a baseline adjustment was made, data were stored on magnetic cassette tape in digital format.

The mapping data were processed off-line on a minicomputer VAX 11/750 (Digital Equipment Co., Maynard, Mass.) to which a laser printer (LBP-406, Canon, Tokyo, Japan) and a high-resolution graphic terminal (System-3400, Lexidata, Billerica, Mass.) were connected.

Data Analysis

The QRS onset, QRS offset, and T onset were visually determined from the superimposed Frank X, Y, and Z leads and spatial magnitude. We calculated the mean potentials of the T waves in each subgroup (normal, anterior MI, or LVH) to which QRS offset was assigned as the reference point for averaging potentials in each study group. The following parameters were measured on each lead of each patient's ECG and of group mean ECG: positive T wave amplitude, negative T wave amplitude, and integral of T potentials during the ST-T segment (the segment from QRS offset to T offset). The positive T wave amplitude was measured as the maximal positive amplitude, and negative T waves were measured as the maximal negative amplitude with respect to the horizontal line through the TP segments. The T max time was defined as the instant of maximal dV/dt near the peak T wave, in which QRS onset was assigned as time zero. Appropriateness of the T max time measurement was checked in each lead on a high-resolution graphic terminal displaying ECGs and their first derivatives. When the computer determined the wrong time, the cursor was manually moved to the peak of the first derivative of the T wave. The number of inappropriate time determinations was usually two or three leads out of 87 leads, and these resulted from a noise that made another peak on the first derivative or ST elevation that obscured the peak of the first derivative. For the latter case, we took the T max time as the point beyond which the T wave slope became negative, according to Haws and Lux.
For statistical analysis, we used two-way ANOVA to analyze differences in T_{max} time and the T wave amplitudes between anterior MI and LVH. In the analysis, the parameters were T_{max} time, positive T wave amplitude, negative T wave amplitude, and T integrals, and examined factors were the subgroups studied and the lead points. The differences in parameters between subgroups were determined independently of the effect of the lead point difference. Linear regression analysis was applied to examine the relation between T_{max} time and T wave parameters on the group mean ECGs. A value of $p<0.05$ was considered significant.

**Results**

**Recovery Time Distribution**

The mean T_{max} time map obtained from normal subjects is shown in the upper panel of Figure 2. The earliest T_{max} times appeared on the upper anterior chest. The isochrone lines propagated toward the lower left lateral chest and then toward the left side of the back. The T_{max} times on the right shoulder area were much delayed.

In the anterior MI group (Figure 2, middle panel), the latest T_{max} times of about 360 msec were detected preferentially on the upper left anterior chest. The earliest T_{max} times were noted on the lower right lateral chest, whereas the latest T_{max} times were distributed close to the area with earliest T_{max} times.

In the LVH group (Figure 2, lower panel), the latest T_{max} times were observed on the left lateral chest. The area characterized by a T_{max} time of more than 360 msec was displaced in a left downward direction compared with that in the anterior MI group.

Two-way ANOVA (Table 1) indicated a significant difference in T_{max} time between the two negative T groups ($p<0.00001$).

**T Wave Shapes**

The mean T waves of the normal subjects are shown in Figure 3. Positive T waves were observed mainly on the left anterior chest and on the lower thorax. The highest peak of positive T wave was located on lead F_5, where the earliest T_{max} time was noted. The negative T waves were located mainly around the right shoulder area. The deepest negative T wave was recorded on lead D_4, where the latest T_{max} time was detected.

In patients in the anterior MI group, negative T waves occurred on the upper left anterior chest, where the latest T_{max} times coincided topographically (Figure 4). The positive T waves were located mainly on the right anterior chest and on the lower thorax.

In patients in the LVH group, negative T waves were detected on the left lateral chest, where the latest T_{max} times were observed (Figure 5). The positive T waves were noted on the right anterior chest and the right back, including the right shoulder.

The negative T waves in the LVH group tended to be detected in the left downward direction compared with those in the anterior MI group. Two-way ANOVA (Table 1) indicated a significant difference in positive T wave amplitude or negative T wave amplitude between two negative T groups (each, $p<0.0001$).

**Relation Between T_{max} Time and T Wave Shape**

In normal subjects, T_{max} times were closely correlated with the amplitudes of positive T wave ($r=0.85$) or amplitudes of negative T wave ($r=0.81$) and the integrated potentials during the ST-T segment ($r=0.82$). The T_{max} time in the anterior MI group was also closely correlated with the amplitudes of negative T wave ($r=0.78$) or the integrated potentials of the T waves ($r=0.77$). Correlation with the amplitudes of positive T wave was not high ($r=0.34$). The T_{max} times in the LVH group correlated with the amplitudes of negative T waves and the T integral values (both $r=0.75$), whereas

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**TABLE 1. Results of Two-Way ANOVA on the Effect of Subgrouping (AMI or LVH) and Lead Points**

<table>
<thead>
<tr>
<th></th>
<th>Anterior MI or LVH</th>
<th>Lead points</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>F</td>
<td>p</td>
</tr>
<tr>
<td>T_{max} time</td>
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</tr>
<tr>
<td>Positive T wave amplitude</td>
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<tr>
<td>Negative T wave amplitude</td>
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<td>0.0001</td>
</tr>
<tr>
<td>T wave integral</td>
<td>0.9</td>
<td>0.34</td>
</tr>
</tbody>
</table>

AMI, myocardial infarction; LVH, left ventricular hypertrophy.
Normal

Figure 3. Tracings of mean T wave shape of 44 normal subjects. The positive T waves were shown mainly on the left anterior chest and lower thorax. The highest positive T wave was located on lead F5, where the earliest \( T_{\text{max}} \) time was shown. Negative T waves were located mainly around the right shoulder portion. The deepest negative T wave was shown on lead D7, where the latest \( T_{\text{max}} \) time occurred.

Anterior MI

Figure 4. Tracings of mean T wave shape of 40 patients in the anterior myocardial infarction (MI) group. The negative T waves occurred on the upper left anterior chest, where the delayed \( T_{\text{max}} \) times were observed. The positive T waves were located mainly on the right anterior chest and lower thorax.
the correlation with positive T wave amplitudes was not high ($r=0.56$).

**Discussion**

We studied the body surface distribution of electrical recovery, especially the $T_{\text{max}}$ times. The results suggested that the T wave potentials were dependent on the $T_{\text{max}}$ time, and the recovery sequence was spatially different between MI and LVH despite the presence of negative T waves in leads $V_{1}$-$V_{6}$.

**A New ECG Measurement, $T_{\text{max}}$ Time**

Local electrical recoveries have been studied on recordings of monophasic action potentials using a suction electrode or of an epicardial ECG from pairs of closely affixed bipolar electrodes. These techniques have been invaluable, but their application to patients was limited because of their invasive nature. Body surface mapping based on unipolar electrodes is clearly noninvasive and is applicable to ill patients or to chronic examinations for follow-up purposes.

We previously demonstrated that the body surface ECG measurement of activation time, which was defined as the time of minimal $dV/dt$ in the QRS complex, was clinically useful for recognizing noninvasively the local abnormalities in the activation sequence.\textsuperscript{16,17,21} Theoretically, the cardiac generator can be viewed as a dipole consisting of a positive and a negative charge separated by a small distance. During activation, when the excitation approaches, the electrical potential shows a positive deflection. When it passes and recedes, the deflection rapidly becomes negative and then returns to baseline. Therefore, the instant of maximal derivative of ECGs was used as an index of local activation. The difference in $T_{\text{max}}$ time measured as the time of maximum $dV/dt$ of the T wave would be that it is largely passive, has slow velocity, and is of opposite polarity. Because of the opposite polarity in repolarization, maximal $dV/dt$ should be used as a measurement.

Millar et al\textsuperscript{6} and Haws and Lux\textsuperscript{7} described the relation between the recovery time and local repolarization in an experimental model. They found a very high correlation ($r=0.99$) between the activation recovery intervals and refractory periods or transmembrane action potential durations. They also found high correlations not only during cycle length change, norepinephrine infusion, or sympathetic nerve stimulation, but also in ischemia. Chen et al\textsuperscript{4} showed a high correlation between epicardial activation recovery interval and monophasic action potential duration on the right ventricle in patients with right ventricular hypertrophy.

**Figure 5.** Tracings of mean T wave shape of 21 patients in the left ventricular hypertrophy (LVH) group. The location of negative T waves was shifted left downward, and they were distributed over the entire left anterior chest. The right anterior chest and the right back including the right shoulder had positive T waves.
Some concern may be raised regarding the accuracy in the present estimation of local recovery by the peak of T wave derivatives. A simulation study carried out by Steinhaus\textsuperscript{8} suggested that errors can be made in the measurement of the recovery time when the cardiac generator includes nonuniform propagations. Nevertheless, the reported estimations of recovery time from epicardial potentials in ischemia\textsuperscript{7} and hypertrophy,\textsuperscript{4} which obviously include the nonuniform propagation, were sufficiently accurate in predicting a local recovery. Another methodological concern was the accuracy of estimation of the recovery sequence from body surface potentials. Burgess et al\textsuperscript{22} measured the recovery times recorded from distant electrodes in a model of an isolated dog heart placed in a tank filled with a conductive water. They concluded that recordings from distant leads (4.5 cm from the cardiac surface) still electrically represent details of the local recovery, and they suggested the possibility of applying this concept to body surface potentials.

Body Surface \( T_{\text{max}} \) Time Distribution

Regional differences in the local electrical recovery were clearly shown in normal subjects. Early recovery was detected on the upper anterior chest, and delayed recovery was reflected on the left lateral chest. Toyoshima et al\textsuperscript{3} constructed an epicardial isochrone map of recovery time estimated from multiple records of monophasic action potentials. They noted an early recovery at the base and a delayed recovery at the apex. Accordingly, the body surface distribution of recovery time is assumed to reflect regional differences in epicardial distribution of the recovery time. The right shoulder area, where the ECG reflects the potentials of the endocardial surface of the LV apex, corresponded to the area with markedly delayed \( T_{\text{max}} \) time. This suggests a delayed recovery at the endocardium of the LV apex.

We observed delayed \( T_{\text{max}} \) times on the upper anterior chest of the anterior MI group. Gough et al\textsuperscript{4} reported prolongation of the recovery time on the infarcted area in dogs. In their study, the refractory periods increased in duration from the normal zone to the infarcted zone as concentric rings. The surviving myocardium may correspond to the delayed recovery times. In the LVH group, the delayed \( T_{\text{max}} \) times were observed on the left lateral chest. A computer model\textsuperscript{3} suggested that the action potential duration on hypertrophic myocardium should be prolonged for the formation of negative T waves. Chen et al\textsuperscript{4} recorded long activation–recovery intervals on the right ventricle in patients with right ventricular hypertrophy at the time of cardiac surgery. Thus, the delayed \( T_{\text{max}} \) time in patients with LVH in the present study should reflect delayed repolarization on the LV.

Relation Between \( T_{\text{max}} \) Time and T Wave Shape

Inhomogeneous ventricular recovery is thought to be the origin of the T wave.\textsuperscript{1,2} The present study showed that T wave potentials are dependent on \( T_{\text{max}} \) time and that the T wave shape is determined by recovery sequence. However, the correlation coefficient, when it was calculated between positive T wave amplitude and \( T_{\text{max}} \) time, was lower in anterior MI (\( r=0.34 \)) and LVH (\( r=0.56 \)) than in normal subjects (\( r=0.85 \)). This discrepancy may be explained by the presence of injury currents reflecting the presence of ST elevation. Such effects were less marked in the LVH group and were not present in normal subjects.

The delayed \( T_{\text{max}} \) times in the LVH group were distributed in a left downward direction compared with the anterior MI group. The regional differences in \( T_{\text{max}} \) times between the two groups were accompanied by regional differences in positive and negative T wave amplitudes. The present finding suggested a difference of the location relating to abnormal repolarizations. Namely, we think that it will be rational to determine negative T waves in MI and LVH on the body surface mapping. In particular, the \( T_{\text{max}} \) time might be the most effective parameter for separating MI and LVH. However, further examination will be required to clarify this point.

Acknowledgment

We would like to thank Dr. Shoji Yasui for encouragement and helpful discussions.

References


Relation between recovery sequence estimated from body surface potentials and T wave shape in patients with negative T waves and normal subjects.
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_Circulation_. 1992;85:1768-1774
doi: 10.1161/01.CIR.85.5.1768

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1992 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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