Detection of local abnormalities in ventricular activation sequence by body surface isochrone mapping in patients with previous myocardial infarction

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ABSTRACT Body surface isochrone mapping was performed in 36 normal subjects and in 85 patients with previous myocardial infarction. Eighty-seven unipolar electrocardiograms distributed over the anterior chest and the back were recorded simultaneously. For each lead, activation time was measured as the time from the onset of QRS to the peak of the R wave. The lead points where R waves were not observed were designated the "no R wave area" (NR area). Isochrone maps of normal subjects had a consistent pattern, with isochrone lines extending from the right upper anterior chest to the left anterior chest and then to the back. NR area was small and was located only on the right upper chest or the upper back. On the isochrone maps of patients with myocardial infarction, abnormal findings were observed; NR area was found in 26 of 28 patients with anterior infarction on the upper to middle anterior chest, in 13 of 22 patients with inferior infarction on the lower chest, and in 24 of 25 patients with anterior and inferior infarction on the upper to lower anterior chest. Activation time was delayed near the NR area (peri-NR area delay) in 37 patients. In patients with apical infarction, an islandlike zone of delayed activation was typically found on the left precordium. These abnormal patterns are considered to indicate local abnormalities in the activation of infarcted myocardium; the NR area indicates dead unexcitable scar, and the peri-NR area delay and islandlike zone of delayed activation indicate partially infarcted myocardium of slow activation. Patients with NR area had greater degree of left ventricular asynergy and lower ejection fraction than those without. Patients with peri-NR area delay had higher incidence of ventricular arrhythmia than those without. Body surface isochrone mapping provides new evidence of myocardial infarction that is not available by the conventional analysis of the electrocardiogram.

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BODY SURFACE potential mapping has been used increasingly for the diagnosis of myocardial infarction. In the analysis of mapping data, the most conventionally used technique is the instant-by-instant observation of the isopotential maps. Isopotential display has the advantage of presenting simultaneous comparisons of potential at many body surface sites and can demonstrate abnormal potential distribution in patients with myocardial infarction who had no classical diagnostic Q wave in standard 12-lead electrocardiograms (ECGs). However, detection of subtle disorders in the activation sequence may not always be easy in isopotential mapping because it is very time consuming to analyze many isopotential maps of various times. Comparison of time factors such as local activation time between different leads is difficult in the isopotential map display.

Flowers et al. analyzed isopotential maps of anterior and inferoposterior myocardial infarction by departure map technique and found areas of abnormal positivity at the mid and late QRS period. They proposed that the mid and late activation changes detected by the departure map were related to ischemically induced alterations in the temporal sequence of ventricular activation. In this study we attempted to detect the local abnormalities in ventricular activation sequence directly by body surface isochrone mapping.

The isochrone map is a display of the distribution of ventricular activation time, and many investigators...
have performed epicardial isochrone mapping in patients with Wolff-Parkinson-White syndrome, bundle branch block, and ventricular tachyarrhythmias. However, few have performed body surface isochrone mapping to detect abnormalities in the ventricular activation sequence. We made body surface isochrone maps of patients with myocardial infarction and tried to detect local abnormalities in ventricular activation, which are hard to detect on standard 12-lead ECGs and isopotential map displays. Information about the local ventricular activation sequence may help to determine the extent and severity of ischemic damage and to predict the occurrence of ventricular arrhythmias after infarction.

Materials and methods

Subjects. From 614 consecutive patients who underwent left ventriculography and selective coronary arteriography in Yamagata University Hospital from August 1979 to July 1984, 85 satisfying all of the following criteria were selected for this study: (1) a clinical diagnosis of myocardial infarction established by typical chest pain and serum enzyme changes; (2) asynergic site proved by left ventriculography; in this study, asynery implies akinesis or dyskinesis according to the reporting system of the American Heart Association; (3) significant stenosis of at least one major coronary artery; (4) no other heart disease such as congenital heart disease, myocardial disease, or valvular heart disease; and (5) no conduction disturbance such as right bundle branch block, left bundle branch block, or Wolff-Parkinson-White syndrome. The QRS duration was less than 0.12 sec.

The patient group consisted of 73 men and 12 women 35 to 69 years of age (mean 55). The time from the onset of the myocardial infarction to the cardiac catheterization ranged from 2 to 11 months (mean 5.1).

Thirty-six clinically normal subjects were also studied (the normal group). They were all men, 22 to 51 years of age (mean 33). All had normal physical and electrocardiographic findings. None of them had a history of heart disease or hypertension.

Informed consent was given by all subjects before the study commenced.

Left ventriculography and coronary arteriography. Biplane left ventriculograms in the 30 degree right anterior oblique and 60 degree left anterior oblique projections were recorded on 35 mm film taken at 50 frames/sec with the Toshiba 9 inch image amplifier system (Angiorex-U-arm). The left ventricle was opacified with contrast medium (megilumine diatrizoate) injected into the left ventricle at a rate of 13 ml/sec for 3 sec. The left ventricular ejection fraction was calculated from biplane left ventriculograms according to the area-length method of Dodge et al., with the use of Siemens cardiac catheterization SICOR system. Left ventricular wall motion was evaluated qualitatively by three or more observers who had no knowledge of mapping data, according to the reporting system of the American Heart Association. Regional wall motion was graded as follows: normal = 0, hypokinesis = 1, akinesis = 2, and dyskinesis = 3. These scores were given separately to the seven segments (anterobasal, anterolateral, apical, diaphragmatic, posterobasal, septal, and posterolateral) of the left ventricular wall. The asynery index was determined by adding the seven scores.

In this study, segments of akinesis or dyskinesis were referred to as the asynergic sites. Asynery of the anterobasal, anterolateral, and/or septal segments with or without apical asynery was referred to as anterior asynery. Asynery of the diaphragmatic, posterobasal, and/or posterolateral segments with or without apical asynery was referred to as inferior asynery. Asynery of only the apical segment was referred to as apical asynery.

Selective coronary arteriographic studies were carried out in multiple projections according to the Judkins technique. Coronary arterial narrowing of 70% or more in the luminal diameter was considered significant.

Body surface mapping

Map recording. Body surface mapping was performed with a body surface potential mapping system, HPM-5100S unit (Chunichi Denshi Co.) within a week before the cardiac catheterization. Because the procedure for data sampling and processing has been described in detail elsewhere, it will be reviewed here only briefly. 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 (59 leads on the anterior chest and 28 leads on the back). ECGs from these 87 unipolar leads with Wilson’s central terminal as reference, standard 12-lead ECGs, and the Frank X, Y, Z ECGs were sampled simultaneously. The stored signals of each ECG were then displayed on a graphic terminal (Tektronix 4006-1). If noise was detected in any of the signals, data sampling was repeated. The flat portion of the PQ segment was chosen for the baseline. After the baseline adjustment, the data were recorded in a magnetic cassette tape in a digital format. This system had a resolution of 10 μV, in the dynamic range ±5 mV, with a sampling rate of 1000 samples/sec/channel. The data sampling was done at the resting expiratory level and in the supine position.

Data analysis. The map data were processed off-line on a minicomputer (Texas Instrument 980-B) by means of the program for isochronal map analysis developed at our institution. For this study, the onset of QRS was determined from superimposed Frank X, Y, Z leads and the spatial magnitude and was called “zero time.” For each lead, the duration from zero time to the time of the peak of the R wave was measured and was designated “activation time.” When two R waves were present in a lead, the greater R wave was chosen for the measurement. When the R wave was absent in a lead, the area where the lead points were located was called the “no R-wave area” (NR area). The body surface distribution of the activation times was displayed as an isochrone map. On the display of isochrone maps, the rectangular area represented the torso surface, with the left half reflecting the anterior chest and the right half the back. Thus both the right and left edges represent the right midaxillary line. Each contour line connected points of equal activation time. The interval of isochrone lines was 5 msec. NR areas were indicated by crosses.

Examinations for ventricular arrhythmia. Holter monitoring was done in 25 patients and treadmill exercise testing was done in all 85 patients within a week before or after the cardiac catheterization. Twenty-four hour ambulatory ECGs of modified leads V1 and V5 were recorded by a Holter recorder (Avionics model 445B). All recordings were analyzed by a computer-assisted system (Avionics Cardioscanner) as to the presence of ventricular premature contractions (VPCs). Submaximal treadmill exercise was performed according to Bruce’s protocol. The ECG of lead CM5 was continuously monitored before, during, and at least 10 min after the exercise.

All patients were followed for 6 months to 5 years (mean 3.1 years) after the onset of myocardial infarction as to the occurrence of ventricular tachycardia.

Statistical analysis. Group mean data were expressed as mean ± SD. Statistical difference was examined by the un-
Results

**Isochrone maps of normal subjects.** Figure 1 represents isochrone maps of normal subjects. Figure 1, A, is an isochrone map from a 44-year-old man. On this map, activation time was shortest on the right upper anterior chest. Isochrone lines extended from the right upper anterior chest to the left anterior chest in the leftward and downward direction. After they reached the left lateral chest, isochrone lines propagated to the back in a rightward and upward direction. The activation time was longest on the upper back.

Figure 1, B, is another example of normal isochrone map obtained from a 26-year-old man. Although there were slight interindividual differences, the total distribution pattern of the isochrone map was almost the same as that in the first case. Activation time was shortest on the right upper anterior chest. Isochrone lines spread from the right upper chest over the left anterior chest and finally to the back. Small NR areas were found on the upper back (only two lead points).

As shown in figure 1, isochrone maps of normal subjects had a consistent distribution pattern, extending from the right upper anterior chest toward the left anterior chest and then to the back. At the fifth intercostal level (row 4, indicated by arrows), the 30 msec isochrone line crossed near the anterior median line, the 40 msec isochrone line passed nearby the left axillary line, and the 50 msec isochrone line crossed near the posterior median line. The difference between the activation times of neighboring lead points did not exceed 20 msec except on the right axillary zone and upper back. NR area was found in 24 of the 36 normal subjects on the upper right anterior chest or on the upper back. All these NR areas were small (three lead points in four subjects, two lead points in seven, and one lead point in 13). NR area did not exist on the right lower anterior chest, central sternal region, left anterior or left lateral chest, or lower back.

**Isochrone maps in patients with myocardial infarction.** The patients were divided into four groups according to the asynergic site of the left ventricle: group 1 (anteroasynery group), 28 patients; group 2 (inferior asynergy group), 22 patients; group 3 (anterior and inferior asynergy group), 25 patients; group 4 (apical asynergy group), 10 patients.

**Group 1 (anterior asynergy group).** Group 1 consisted of 28 patients, 25 men and three women 41 to 67 years of age.

Figure 2 shows the isochrone map obtained from a patient in group 1. The left ventricular asynergic site was located in the anterolateral and apical segments (both were dyskinetic). On the isochrone map, NR area lay over the upper right anterior chest and central sternal regions. The earliest isochrone line appeared on the left upper lateral chest at 40 msec from the onset of QRS. Isochrone lines extended from the left upper anterior chest to the left lateral chest and then to the back. On the middle right to lower anterior chest, isochrone lines propagated from the lower chest toward the NR area, in the opposite direction from the normal isochrone maps. Isochrone lines crowded on the lower portion of the right anterior chest. On the lower side of the NR area, activation time was much delayed (70 to 80 msec); this phenomenon was referred to as “peri–NR area delay.” Peri–NR area delay was judged to be present when a zone of three or more leads of delayed activation was found near the NR area; delayed activation was considered significant when the activation time was obviously outside what was found in normal subjects for that torso area with coexistent increased density of isochrone lines. Contrary, on the left side of the NR area, isochrone lines were suddenly interrupted and peri–NR area delay was not observed on the isochrone map.
FIGURE 2. Isochrone maps from a patient in group 1 (anterior asynergy). An NR area was located on the upper to middle anterior chest. Peri–NR area delay was observed on the right anterior chest.

Figure 3 represents an isochrone map of another patient in group 1. The asynergic site was anterolateral segment (akinesis). NR area was found on the upper anterior chest. Activation times on the left upper chest were slightly longer than those on the left middle anterior chest. Isochrone lines propagated on the left anterior chest in the upward direction and finally toward the NR area, as if they moved around the NR area. We thought that slight peri–NR area delay was present in this case.

In 26 of the 28 patients (93%) of group 1, NR area was found on the upper to middle anterior chest. The mean size of NR area in these 26 patients was 11.6 ± 3.0 leads. Peri–NR area delay was found in 10 patients (36%).

Group 2 (inferior asynergy group). This group consisted of 22 patients, 20 men and two women 40 to 69 years of age.

Figure 4 is an isochrone map of a patient in group 2. The asynergic site was located in the apical, diaphragmatic, and posterobasal segments (akinesis, akinesis, and dyskinesis, respectively). NR area was located widely on the lower chest. Isochrone lines were converged on the inferior chest near the NR area. Activation time was much delayed on the back (over 70 msec), especially in the inferior portion. Peri–NR area delay was judged to be present on the back in this case.

Of the 22 patients in group 2, 13 patients (59%) had NR area on the lower chest. The mean size of NR area was 11.5 ± 3.2 leads. Peri–NR area delay was observed in 11 patients (50%).

Group 3 (anterior and inferior asynergy group). Group 3 consisted of 25 patients, 18 men and seven women 35 to 69 years of age.

Figure 5 represents an isochrone map of a patient in group 3. The asynergic site was located in the anterolateral, apical, and diaphragmatic segments (akinesis, dyskinesis, and dyskinesis, respectively). A large NR area lay over almost the entire anterior chest except for the left lateral portion. Isochrone lines extended from the left upper chest to the back. But on the lower portion of the left lateral chest, a zone of delayed activation time was present. This was also determined to be peri–NR area delay.

Figure 6 is another isochrone map of a patient in group 3. The asynergic site was located in the anterolateral, apical, and diaphragmatic segments (all dyskinesis). NR area was located on the upper portion of the anterior chest. Activation time was earliest in the
left-lateral inferior chest and the lower back. Isochrone lines extended from inferior to superior toward the NR area. On the left anterior chest, activation time was much delayed beside the NR area. In this case, the area of the peri-NR area delay was extremely wide and occupied almost all the anterior chest except for the NR area.

NR areas were found in 24 of 25 patients (96%) in group 3 and extended over the upper to lower right and left anterior chest. The group mean size of NR area was 21.0 ± 3.3 leads and significantly greater than those in groups 1 and 2 (p < .01, p < .01). Peri-NR area delay was found in 16 patients (64%).

**Group 4 (apical asynergy group).** Group 4 consisted of 10 patients, all men, 45 to 66 years of age.

Figure 7A represents an isochrone map of a patient in group 4. The asynergic site was located in the apical segment. A small NR area was found on the right anterior chest, but on the left anterior chest no NR area was present. Isochrone lines crowded on the left anterior chest and formed an island-like zone of delayed activation time (indicated by an arrow). As was shown in figure 7B standard 12-lead ECGs failed to detect this abnormal activation pattern.

Out of the 10 patients in group 4, four (40%) had NR areas on the anterior chest. The mean size of the NR area in the four patients was 7.0 ± 2.0 leads, significantly smaller than those in groups 1, 2, and 3 (p < .05, p < .05, and p < .01, respectively). Peri-NR area delay was not observed in any case. Crowds of isochrone lines were considered significant when activation time was delayed by over 20 msec between neighboring leads except on the right axillary zone and upper back. Isochrone lines crowded on the left anterior chest in nine patients and formed an island of delayed activation as in figures 7A and 7B in five patients.

Table 1 summarizes the abnormal findings on the body surface isochrone maps in patients with myocardial infarction (NR area, peri-NR area delay, crowd of isochrone lines, and islandlike zone of delayed activation). Islandlike zones of delayed activation were found in nine patients, all of whom had asynergy in the apical segment. Of the 85 patients, 83 (98%) had abnormal patterns on isochrone maps.

**Correlation of the isochrone map findings with asynergy index, ejection fraction, and incidence of ventricular arrhythmias.** Patients were divided into three sets of subgroups according to the findings of the isochrone
maps: those with and without NR area, those with and without peri-NR area delay, and those with and without crowds of isochrone lines. Between each subgroup, asynergy index, left ventricular ejection fraction, and the incidence of VPCs were compared (table 2).

The group with NR area had a higher asynergy index (p < .01) and lower ejection fraction (p < .01) than the group without NR area. The group with peri–NR area delay had a higher asynergy index (p < .01) and lower ejection fraction (p < .01) than the group without peri–NR area delay. However, a significant difference was not found in asynergy index or ejection fraction between the groups with and without crowds of isochrone lines.

The incidence of VPCs was significantly higher in the group with peri–NR area delay than in the group without (p < .05). It also tended to be higher in the group with crowds of isochrone lines than in the group without, but the difference was not significant. There was no significant difference in the incidence of VPCs between the groups with and without NR area.

During the follow-up period, episodes of ventricular tachycardia were found in three patients in group 3 (anterior-inferior asynergy). All of them had peri–NR area delay.

### Discussion

The purpose of this study was to detect local abnormalities in ventricular activation sequence in patients with previous myocardial infarction by body surface isochrone mapping.

**Interpretations of the body surface isochrone maps.** From the results of this study, body surface isochrone maps of normal subjects showed a consistent pattern in which isochrone lines propagated from the right upper anterior to left lateral chest and finally to the back. The leftward extension of the isochrone lines is considered to reflect the ventricular excitation from the septum to the left ventricular free wall. We could not separate the excitation of the right ventricle by this analysis. Thus we focused our attention on the relationship between the left ventricular asynergic site and the distribution pattern of the isochrone lines in patients with previous myocardial infarction.

The results in patients revealed that the NR area was usually located on the upper to middle anterior chest in group 1 (anterior asynergy), on the lower anterior chest and the back in group 2 (inferior asynergy), and on the upper to lower anterior chest in group 3 (anterior and inferior asynergy). NR area was found in 93% of group 1, 59% of group 2, and 96% of group 3 patients. Sometimes activation time was very delayed near the

### TABLE 1

Abnormal findings on the isochrone maps

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>NR area</th>
<th>Peri–NR area delay</th>
<th>Crowd of isochrone lines</th>
<th>Islandlike zone</th>
<th>One or more abnormalities(^a)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>28</td>
<td>26 (93)</td>
<td>10 (36)</td>
<td>11 (39)</td>
<td>1 (4)</td>
<td>28 (100)</td>
</tr>
<tr>
<td>2</td>
<td>22</td>
<td>13 (59)</td>
<td>11 (50)</td>
<td>9 (41)</td>
<td>0 (0)</td>
<td>20 (91)</td>
</tr>
<tr>
<td>3</td>
<td>25</td>
<td>24 (96)</td>
<td>16 (64)</td>
<td>17 (68)</td>
<td>3 (12)</td>
<td>25 (100)</td>
</tr>
<tr>
<td>4</td>
<td>10</td>
<td>4 (40)</td>
<td>0 (0)</td>
<td>9 (90)</td>
<td>5 (50)</td>
<td>10 (100)</td>
</tr>
<tr>
<td>total</td>
<td>85</td>
<td>67 (79)</td>
<td>37 (44)</td>
<td>46 (54)</td>
<td>9 (11)</td>
<td>83 (98)</td>
</tr>
</tbody>
</table>

Numbers in parentheses are percentages of the group.

\(^a\)Number of patients who had at least one of the abnormal findings.

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### TABLE 2

Correlation of the isochrone map findings with the findings of left ventriculography and the incidence of VPCs

<table>
<thead>
<tr>
<th></th>
<th>NR area</th>
<th>Peri–NR area delay</th>
<th>Crowd of isochrone lines</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>+</td>
<td>–</td>
<td>+</td>
</tr>
<tr>
<td>AI</td>
<td>7.1 ± 3.3(^b)</td>
<td>4.8 ± 2.3</td>
<td>8.6 ± 3.0(^b)</td>
</tr>
<tr>
<td>EF (%)</td>
<td>47.0 ± 12.8(^b)</td>
<td>56.0 ± 11.5</td>
<td>43.7 ± 12.3(^b)</td>
</tr>
<tr>
<td>VPC (%)(^c)</td>
<td>36</td>
<td>33</td>
<td>49(^a)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

\(^a\)Incidence of VPCs during Holter monitoring or treadmill test.

\(^b\)p < .05; \(^c\)p < .01 compared with the negative subgroup.

\(^b\) = asynergy index; EF = left ventricular ejection fraction.

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NR area (peri–NR area delay). This delay was most frequently found in patients with larger infarction, as in group 3 (67%). On the other hand, in cases of small infarction of the apical segment (group 4), NR area was less frequent (40%). But a crowd of isochrone lines frequently existed on the left anterior chest (nine of 10, 90%). In typical cases, local delay of the activation time was indicated by an island of isochrone lines on the left precordial region. As shown in Table 2, patients with NR area had a greater asynergy index and lower ejection fraction than those without. Furthermore, patients with peri–NR area delay had a greater asynergy index and lower ejection fraction.

We propose the following interpretations for these findings: the NR area indicates the central portion of a large infarction, which was almost unexcitable scar; the peri–NR area delay, crowds of isochrone lines, and islandlike zones of delayed activation indicate the area of partial or small infarction, where excitation propagated slowly through the surviving myocardial cell. These hypotheses agree closely with the results of Durrer et al. on epicardial and intramural ECGs in experimental chronic myocardial infarction. They concluded that QS complexes on epicardium were present only where the infarction was transmural. Where living intramural muscle fibers were present, excitatory waves followed circuitous routes and the mean conduction velocity was reduced.

Local delay of activation has been considered to have a close relationship with ventricular arrhythmias. Patients with peri–NR area delay had a higher incidence of VPCs than those without. All the patients with episodes of ventricular tachycardia had peri–NR area delay. Although these results are preliminary, they suggest that peri–NR area delay will be an aid in identifying high-risk patients with ventricular tachyarrhythmias.

When NR area is not associated with peri–NR area delay, the excitation may be suddenly interrupted beside the infarcted area or may be delayed in a very small site near the infarcted area, undetectable by the body surface isochrone map. Further study must be conducted to address this problem.

**Influences of the time after infarction on the isochrone maps.** QRS waveforms sometimes change, especially in the acute phase of myocardial infarction, as well as the extent of asynergy of ventricular wall. Figure 8 represents isochrone maps of a patient who underwent left ventriculography twice. The first left ventriculographic study was performed 4 weeks after the onset of myocardial infarction. Anterolateral and apical segments were dyskinetic and ejection fraction was 46%.

**FIGURE 8.** Isochrone maps from a patient in group 1 recorded at 4 weeks (A) and 7 months (B) after the infarction. In B, the size of NR area becomes smaller than in A. Several leads on the left and lower part of the NR area in A showed the development of R waves (B, indicated by asterisk), indicating a zone of slow conduction.

Figure 8, A, is the isochrone map at that time. NR area was located on the upper to middle anterior chest. Isochrone lines appeared on the lower anterior chest at 25 msec and propagated leftward and upward. On the left lateral chest, activation time was later near the NR area and peri–NR area delay was judged to be present. On the right lower chest, isochrone lines crowded and slowly propagated toward the NR area.

Six months later, the patient underwent a second left ventriculographic study. Asynergy of anterolateral and apical segments was reduced to akinesis and ejection fraction increased to 61%. Figure 8, B, is the isochrone map at the second left ventriculographic examination 7 months after the onset of infarction. NR area decreased in size, especially on the left anterior chest, and was replaced by a zone of slow conduction (indicated by an asterisk) and a crowd of isochrone lines. On the right anterior chest the distribution pattern of isochrone lines was not much different from the first time.

We thought that the NR area detected at the first study included the electrically silent myocardium with reversible damage as well as the dead unexcitable myocardium. We supposed that during the interval between the two recordings, reversibly damaged myocardium had recovered and the irreversibly damaged
area still existed. At the second ventriculographic study, we thought that the NR area indicated the infarcted scar and the crowd of isochrone lines indicated the partially infarcted slow conduction area.

To avoid the influence of short-term reversible change on the isochrone maps, we analyzed the ECGs at the early chronic phase of myocardial infarction (2 to 11 months) in this study. However, comparison of isochrone map patterns at the acute and chronic phases may be used to evaluate the balance of reversible and irreversible damage in the infarcted myocardium.

**Limitations of body surface isochrone maps.** Isochrone maps have been made from epicardial bipolar ECGs. In this study, we tried to construct isochrone maps from body surface unipolar ECGs. Designation of the activation time on the body surface unipolar ECGs is a problem,\(^6\) and we chose the peak of the R wave because it has been conventionally used to measure the ventricular activation time in clinical electrocardiology.\(^2\) On the precordial ECG, the deflection from the peak of the R wave to the termination of the R wave or to the nadir of the S wave is called “intrinsicsoid deflection,” which is assumed to occur when the depolarization force reaches the myocardium beneath it. The peak of the R wave is the onset of the intrinsicsoid deflection and is considered to represent the arrival of the depolarization force at the myocardial surface under the precordial lead.

Therefore the existence of the QS complex implies that the activation is proceeding away from the recording site and does not necessarily mean that the area is not activated. In fact, the NR area is sometimes seen in normal subjects on the right upper anterior chest and upper back, where the ECG leads are supposed to face the endocardial surface. But when the NR area is located on the other part of the torso surface of a patient with myocardial infarction, it may be reasonable to assume that the area reflects the inactive myocardium damaged by the infarction.

The potential on the epicardial surface is transmitted through the lungs, muscle layer, and skin to the body surface. The body surface potential at a point is mainly determined by the epicardial potentials beneath it, although it is also affected by potentials of the other part of the epicardium to some extent. The activation time measured in this study may be somewhat different from the local activation time of the corresponding point of the epicardial surface. However, the results of this study indicated that body surface isochrone maps reflected the normal left ventricular activation sequence from septum to the free wall and depicted abnormalities corresponded well with the asynergic site in patients with myocardial infarction. Thus we believe that body surface isochrone maps can be used clinically to determine the activation sequence of the myocardium.

**Clinical implications.** Peri–NR area delay and the island of delayed activation or the crowd of isochrone lines are not easily detected by the conventional analysis of ECGs. The island of delayed activation on the precordium was characteristic of apical infarction. It has been difficult to diagnose the infarction of the apical segment by the ECG. Thus body surface isochrone mapping will be a useful analytic method for apical infarction.

Patients with peri–NR area delay had a higher incidence of VPCs than those without. All the patients with episodes of ventricular tachycardia had peri–NR area delay. Local delay of activation indicated by the body surface isochrone map may be used to predict the occurrence of serious ventricular arrhythmias. Furthermore, as mentioned above, isochrone mapping may aid in evaluating the balance of reversible and irreversible damage in the infarcted myocardium. Further study will be carried out to address these points.

Although body surface isochrone mapping has several limitations, it is able to represent local abnormalities in ventricular activation sequence. Information from the body surface isochrone maps will provide new evidence for the occurrence of myocardial infarction.

**References**


9. Wyndham CRC, Smith T, Meenan MK, Mammana R, Levitsky S,

Erratum
In an article by Eichler et al. that was published in the April 1985 issue of Circulation (71: 779, 1985), a sentence in the Abstract appeared incorrectly. The sentence should have read: “The hemodynamic effects of this drug (1 mg/kg followed by 25 μg/kg/min over 36 hr) were studied, in 30 patients randomly assigned to a tiapamil or control group within 12 hr of the onset of acute myocardial infarction, by Swan-Ganz catheterization and gated blood pool scans.”

DIAGNOSTIC METHODS—ELECTROPHYSIOLOGY

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