New Body Surface Isopotential Map Evaluation Method to Detect Minor Potential Losses in Non–Q-Wave Myocardial Infarction

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Background—Potential losses caused by stable non–Q-wave myocardial infarction (MI) are too small to diagnose with the use of standard ECG. The aim of the present study was to obtain accurate diagnostic criteria for this prognostically important disease with the help of body surface mapping.

Methods and Results—Body surface potentials were recorded with the use of 63 unipolar leads in 45 patients with a non–Q-wave MI (41 to 75 years old); 24 healthy adults, 42 patients with unstable angina, and 70 patients with Q-wave MI served as reference groups. Qualitative pathological features of the isopotential maps, such as onset time and site and magnitude of the first right-anterior/anterior minimum, as well as pathological negativities at that time, were defined in non–Q-wave MI cases. These features, which account for the activation sequence and the body surface projections of specific cardiac regions (Selvester classification), showed a 91% sensitivity and an 88% specificity for the detection of non–Q-wave MI. In comparison, the different departure maps (first third QRS, QRS, and QRST isoarea) resulted in less favorable specificities (50% to 58%). Concordance between the isopotential maps and the acute-phase ECG (90%), hypokinesis (64%), fixed perfusion defects (59%), and significant stenosis of the infarct-related coronary artery (87%) supported the concept that these isopotential map changes correspond to the supposed sites of MI. There were pathological features in 69% of patients with unstable angina, with similar concordances as in non–Q-wave MI.

Conclusions—Isopotential maps revealed characteristic features that were suitable for the detection and localization of non–Q-wave MI in the clinical setting of unstable coronary artery disease. (Circulation. 2000;101:1115-1121.)

Key Words: angina ■ mapping ■ myocardial infarction ■ potentials

Montague et al1 suggested that non–Q-wave myocardial infarction (NQMI) is a distinct, heterogeneous pathophysiological entity, whereas others2 consider NQMI to be smaller in measure than Q-wave myocardial infarction (QMI). Because of their common features, NQMI and unstable angina (UA) are grouped under the term “unstable coronary artery disease” (UCAD).3 The identification of NQMI is of paramount clinical importance, because it is more frequently associated with future UA, malignant arrhythmias, and sudden cardiac death than is QMI.4 The diagnosis of acute NQMI is generally based on clinical observations, laboratory test results, and acute ST-T changes on the ECG.5 However, ECG changes are missing when chronic NQMI is being considered. Therefore, attempts have been made to diagnose chronic NQMI through other noninvasive methods, such as echocardiography,6 thallium scintigraphy,7 radionuclide ventriculography,1,7 and body surface potential mapping (BSPM).2,8–12 The use of BSPM may detect electrical abnormalities such as the loss of electrical potential or altered activation sequence during depolarization. “Potential loss” is the reduction in the electrical activity of the heart due to a functional or structural lesion that can arise from limited necrosis or ischemia. These electrical abnormalities involving ≥1 cardiac region correspond to myocardial damage insufficient to cause a Q wave on the standard 12-lead ECG. However, BSPM can be suitable to detect these abnormalities because of its higher spatial resolution. The position of the initial potential minimum, as suggested by Osugi et al8 and Hirai et al,9 or differences in isoarea maps, as suggested by De Ambroggi et al,10,11 were helpful in the diagnosis of old anterior and inferior NQMIs (without more accurate localization), but sensitivity was low.

The aim of the present retrospective study was to analyze body surface isopotential maps in clinically proved NQMI during the chronic phase and to identify characteristic fea-
tures specific to each cardiac region. The localization was supported by concordances with different invasive and non-invasive methods. Our hypothesis was that a series of isopotential maps should yield more information than the isoarea departure maps. Thus, we also evaluated different departure maps and compared the sensitivities and specificities for the diagnosis of NQMI. The isopotential map features that were found to be characteristic of potential loss in NQMI were also investigated in the normal control group and in 2 reference groups of patients with ischemic heart disease (UA and QMI).

Methods

Patient Selection
All of the patients were hospitalized and were from the Hopital du Sacré-Cœur de Montréal. The NQMI group consisted of 45 patients (32 men and 13 women, age range 41 to 75 years, mean age 61 years), all of whom had enzymatically proved NQMI. One of the reference groups, the UA group, consisted of 42 patients (24 men and 18 women, age range 41 to 74 years, mean age 58 years) who had clinical signs of the UA but no proved myocardial enzyme elevation. In these 2 groups of patients with UCAD, there were no pathological Q waves, all patients underwent coronary angiography and ventriculography, and 79 patients underwent exercise $^{201}$Tl scintigraphy. The other reference group, the QMI group, consisted of 70 patients with remote QMI (32 anterior: 28 men and 4 women, age range 33 to 76 years, mean age 55 years; 38 inferoposterior localization: 33 men and 5 women, age range 48 to 69 years, mean age 60 years).

The BSPM and the other clinical examinations were performed with the patient in a clinically stable state, without acute ST-T changes. No patient had a cardiac disorder other than ischemic heart disease. Exclusion criteria were atrial fibrillation, intraventricular conduction disturbances, and signs of ventricular hypertrophy/strain on the ECG.

The normal control group consisted of 24 healthy adults with a normal ECG (16 men and 8 women, age range 17 to 38 years, mean age 31 years).

Body Surface Potential Mapping

Recording and signal processing of the body surface potentials were performed according to the Montreal system (63 unipolar leads, amplifier 0.05 to 200 Hz, signal averaging 52 seconds). Different types of body surface maps (isopotential, isoarea, and departure) were generated. On these maps, the torso surface is represented by a rolled-out cylinder cut along the right midaxillary line: the left side of the map corresponds to the anterior torso. The zero level is identified with a heavier line, and contour lines are drawn at regular intervals. Plus and minus signs indicate the locations of the maximum and minimum, respectively. Isopotential maps were evaluated every 2 ms. In addition, 3 isoarea maps were constructed: these maps represent the body surface distribution of the time integral of the potential during the first third of the QRS complex, the entire QRS, and the combination of the QRS complex and T wave. Finally, departure maps were generated with the use of mean ± 2 SD values; the averages of the maps of 24 normal subjects at each lead were used.

Evaluation of the Isopotential Maps

The first point of interest was the appearance time (initial, early, normal, or late) of the first right-anterior/anterior minimum. Secondary discriminative signs are site and magnitude of this minimum, as well as minor negativities and positivities at abnormal sites at the same time point (arrows). + and – indicate potential maximum and minimum, respectively. Inf. indicates inferior; neg., negative; post., posterior; pos., positive; Ant, anterior; sup., superior; and lat., lateral.

![Figure 1. Method of localization of potential losses in NQMI with the use of isopotential maps. Schematic display of normal and pathological isopotential maps and corresponding pathologic localization. First step is evaluation of appearance time (initial, early, normal, or late) of first right-anterior/anterior minimum. Secondary discriminative signs are site and magnitude of this minimum, as well as minor negativities and positivities at abnormal sites at same time point (arrows). + and – indicate potential maximum and minimum, respectively. Inf. indicates inferior; neg., negative; post., posterior; pos., positive; Ant, anterior; sup., superior; and lat., lateral.](image)

<table>
<thead>
<tr>
<th>Time /ms/</th>
<th>Normal maps</th>
<th>Pathological features</th>
<th>Localization</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-14</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10-22</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20-26</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>24-40</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30-40</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

regions were taken into account. The sites of potential loss were grouped according to the scheme of Selvester et al., which is based on combinations of 4 longitudinal segments (anteroseptal, anterosuperior, posterolateral, and inferior) and 3 transversal segments (basal, middle, and apical). The beginning of the depolarization was defined as the map recorded 6 ms before the first 0.1-mV positivity.

For numerical evaluation of the features, the averaged potential value for each pathological value and the z value for each individual NQMI patient were computed $\{z = (individual potential value-normal average potential value at the same site and time)/SD of this normal average\}$. The specified electrode site and normalized time point of the characteristic features are given in Table 1. Individual timing was normalized [time in the individual case×average normal QRS duration/individual QRS duration]. The average QRS duration in our normal control group was 83±18 ms.

The sensitivity, specificity, and positive and negative predictive values of the characteristic isopotential map features were evaluated and compared with different types of departure maps. Concordances were evaluated between the localized sites of potential losses through the use of isopotential maps and noninvasive or invasive cardiac methods such as acute-phase ECG (ST elevation in 14, ST depression in 10, and deep negative T waves in 21 participants), hypokinesis (it was evaluated with the use of left ventriculography; midseptal and posterolateral middle/basal locations were not evaluated), fixed perfusion defect (it was evaluated with the use of exercise thallium test scintigraphy), and significant stenoses of >80% (they were evaluated with the use of coronary arteriography).
Statistical analyses were performed with use of the $z$ score, Student’s $t$ test of the mean, unequal variance, and the $\chi^2$ test.

**Results**

Characteristic isopotential map features were found in 40 of 44 NQMIIs. (In 1 patient, the low potentials resulted in an atypical change.) The typical abnormalities that suggest a loss of electrical potential in different regions are displayed in Figure 1 schematically and in Figure 2 with individual maps from patients with NQMI. Computed isopotential maps of the averaged potential values of our normal control group are displayed in Figure 3 at the diagnostically important time points.

### Description of Isopotential Map Features

**Initial Anterior Minimum (0 to 16 ms After QRS Onset, With a Midpresence Time Point of 8 ms)**

An initial anterosuperior minimum slightly to the right of the midfrontal area indicates a potential loss for the midseptal area (5 cases; Figures 1a and 2a).

### Table 1. Time, Site, and Value of Isopotential Map Features in Different Regions With Potential Loss, Corresponding Averaged Normal Values, Difference Between Pathological and Normal Values, and Sensitivity of $z_i$ Score

<table>
<thead>
<tr>
<th>Localization</th>
<th>No. of Localizations</th>
<th>Characteristic Features</th>
<th>Normal Value, $\mu V$</th>
<th>Difference Between Values ($P$)</th>
<th>Sensitivity of $z_i$ Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Midseptal</td>
<td>5</td>
<td>Sup mid ant</td>
<td>$-56 \pm 28$</td>
<td>$+70 \pm 62$</td>
<td>$&lt;0.01$</td>
</tr>
<tr>
<td>Anterior paraseptal</td>
<td>8</td>
<td>Sup left ant</td>
<td>$-95 \pm 37$</td>
<td>$+97 \pm 76$</td>
<td>$&lt;0.01$</td>
</tr>
<tr>
<td>Anteroseptal/inferior middle</td>
<td>22</td>
<td>Mid right ant</td>
<td>$-144 \pm 51$</td>
<td>$-90 \pm 282$</td>
<td>NS</td>
</tr>
<tr>
<td>Anteroseptal apical</td>
<td>9</td>
<td>Inf right ant</td>
<td>$-56 \pm 48$</td>
<td>$+68 \pm 39$</td>
<td>$&lt;0.01$</td>
</tr>
<tr>
<td>Infraapical</td>
<td>4</td>
<td>Inf mid post</td>
<td>$-45 \pm 38$</td>
<td>$+67 \pm 52$</td>
<td>$&lt;0.01$</td>
</tr>
<tr>
<td>Anteroseptal apical</td>
<td>3</td>
<td>Mid mid post</td>
<td>$-100 \pm 58$</td>
<td>$+168 \pm 202$</td>
<td>$&lt;0.05$</td>
</tr>
<tr>
<td>Infraapical</td>
<td>4</td>
<td>Sup left ant</td>
<td>$-319 \pm 76$</td>
<td>$-505 \pm 575$</td>
<td>NS</td>
</tr>
<tr>
<td>Infraapical</td>
<td>3</td>
<td>Sup right ant</td>
<td>$-645 \pm 48$</td>
<td>$-480 \pm 353$</td>
<td>NS</td>
</tr>
<tr>
<td>Posterolateral</td>
<td>5</td>
<td>Sup right post</td>
<td>$-457 \pm 140$</td>
<td>$-317 \pm 222$</td>
<td>NS</td>
</tr>
</tbody>
</table>

Sup indicates superior; inf, inferior; mid, middle; ant, anterior; and post, posterior.

**Figure 2.** Individual pathological isopotential maps with different location of potential loss. a, Midseptal (anterosuperior initial minimum). b, Anterior paraseptal (left anteroseptal initial minimum). c, Anteroseptal/inferior middle (slight early right-sided minimum plus inferior right anterior and inferior midposterior negativities). d, Anteroseptal apical (inferior right anterior negativity plus elevated midposterior positivity). e, Infraapical (contiguous inferior negativity). f, Anteroseptal middle/basal (minimum arrives from above with contiguous superior negativity). g, Inferior basal (late, considerable, and persisting right superior minimum). h, Posterolateral middle/basal (minimum persists on back after maximum positivity). Discriminative features and corresponding regions are connected by arrows. + and − indicate potential maximum and minimum, respectively. Time after QRS onset (bottom), values between 2 lines, and maximum and minimum values for all maps (top) are given.
and left-sided minimum indicates an anterior paraseptal potential loss (8 cases; Figures 1b and 2b). For normal cases at this time point, the minimum is on the back (Figure 3A).

Early, Slight Right Anterior Minimum (10 to 22 ms After QRS Onset)
This minimum (its absolute value is \(<0.22\) mV) and an inferior right anterior and inferior midposterior negativity at this time indicate a potential loss in the anteroseptal/inferior midregion (the most frequent location: 22 cases; Figures 1c and 2c). For normal cases, the first right anterior minimum has a more negative value and comes later; there is only positivity on the inferior torso (Figures 3B and 3C).

Normal (Time and Measure) Right Anterior Minimum (20 to 24 ms After QRS Onset)
At this time, a right-sided inferior negativity with midposterior elevated positivity indicates a potential loss in the anteroseptal/inferior midregion (the most frequent location: 22 cases; Figures 1c and 2c). For normal cases, the first right anterior minimum has a more negative value and comes later; there is only positivity on the inferior torso (Figures 3B and 3C).

Right Anterior/Anterior Minimum Appears Late
A minimum from above is often inserted between 2 neighboring positivities at 24 to 42 ms after QRS onset. At this time, there is a contiguous negativity on the total superior torso that corresponds to an anterosuperior-middle/basal localization (4 cases, Figures 1f and 2f). A late (34 to 40 ms after QRS onset) and considerably negative (\(<-0.6\) mV) minimum that persists on the superior right anterior torso and is followed by a short incisura (smaller minimum) on the minimum time curve (when the minimum moves to the midanterior part) indicates an inferobasal localization (3 cases, Figures 1g and 2g). The minimum that persists on the back later (until \(\approx 40\) ms) after the onset time of the highest maximum indicates a posterolateral middle/basal potential loss (5 cases, Figures 1h and 2h). For normal cases, the minimum appears on the right anterior side in normal time (at 20 to 24 ms but no later than the time of maximum positivity); it moves to the middle part of the anterior chest, and its negativity increases continuously (Figures 3C to 3F).

The combination of some of these features suggests multiple potential losses: midseptal plus anteroseptal/inferior middle (4 cases), midseptal plus anteroseptal/inferior middle plus anteroseptal-apical (1 case), anterior paraseptal plus anteroseptal/inferior middle (1 case), anterior paraseptal plus anteroseptal/inferior middle plus anteroseptal apical (3 cases), anterior paraseptal plus anterosuperior middle/basal (2 cases), anteroseptal/inferior middle plus anteroseptal apical (2 cases; Figure 4a), anteroseptal/inferior middle plus inferior apical (2 cases; Figure 4b), and anterosuperior middle/basal plus posterolateral middle/basal (1 case). In total, 60 regions with potential losses were observed in 44 patients with NQMI.

The numerical values of the pathological features with the corresponding average normal values, the significance level of the differences, and the sensitivity of the significant (\(z\) scores are displayed for each region in Table 1. The numerical values of the pathological features with the corresponding average normal values, the significance level of the differences, and the sensitivity of the significant (\(z\) scores are displayed for each region in Table 1. The sensitivity, specificity, and positive and negative predictive values of the characteristic BSPM changes in the NQMI group were 91%, 88%, 93%, and 84%, respectively (Table 2). The pathological changes (\(\pm 2\) SD) of the first third QRS and QRST isoarea departure maps resulted in lower specific-
ity (50% to 54%, \( P < 0.05 \); Table 2). Concordances between isopotential maps and other noninvasive or invasive methods are given in Table 3.

**What Does the Standard 12-Lead ECG Reveal in NQMI?**

According to BSPM features, the potential losses of different regions resulted in minor changes at different times during the QRS interval. The initial depolarization was involved only in cases of midseptal and anterior paraseptal potential losses: a slight r-wave reduction was revealed in leads V1 and V2. However, true pathological Q waves were seen in the leads situated above them (according to the leads V1 and V2). Consequently, these cases correspond to Kornreich’s “missed” QMIs, in contrast with the other cases in which the initial period was intact and pathological Q waves were nowhere to be seen (ie, true NQMI). For anteroseptal/inferior middle and anteroseptal apical potential losses, the r wave in leads V1 to V3 was narrower, similar to the right-sided leads. For inferoapical and inferobasal sites, a notch could appear in the inferior ECG leads. For the anteroseptal middle/basal sites, an r-wave reduction of varying degrees could be seen in leads V2 to V4. For posterolateral middle/basal sites, the r wave in leads V1 and V2 was wider and a little taller, and the real posterior leads had a wider Q wave because of the persistence of the back-sided minimum.

**How Did the Signs of Potential Losses Appear in UA Patients?**

Twenty-nine UA patients (69%) showed similar pathological features in the isopotential maps. Five of them showed multiple changes, but the ratio of multiple changes was significantly lower than that in NQMI (\( P < 0.05 \)). Together, the potential losses involved 34 regions (midseptal 1, anterior paraseptal 3, anteroseptal/inferior middle 8, anteroseptal apical 6, anterosuperior middle/basal 3, posterolateral middle/basal 7, and inferobasal 6). Atypical changes (low potentials) were detected in 1 patient. The proportion of the \( z_i \) score significance was 2:3 in anterior paraseptal, and there was no significant \( z_i \) score in the other regions. Potential loss abnormalities were detected in 62% with the first third QRS departure map, in 74% with the entire QRS departure map, and in 79% with QRST departure maps. The concordances of localization by isopotential maps and other methods were similar to those in NQMI: 83% for the acute-phase ECG, 58% for the hypokinesis, 62% for the perfusion defect, and 85% for the detection of a significant lesion of the infarct-related coronary artery.

**TABLE 2. Sensitivities, Specificities, and Positive and Negative Predictive Values of New Signs by Body Surface Isopotential Maps and >2 SD Differences by Different Types of Departure Map**

<table>
<thead>
<tr>
<th>Location</th>
<th>No. of NQMI Patients</th>
<th>No. of Normal Patients</th>
<th>Sensitivity, %</th>
<th>Specificity, %</th>
<th>Positive Predictive Value, %</th>
<th>Negative Predictive Value, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Isopotential map</td>
<td>44</td>
<td>24</td>
<td>91</td>
<td>88</td>
<td>93</td>
<td>84</td>
</tr>
<tr>
<td>Isoarea departure map</td>
<td>First-third QRS</td>
<td>43</td>
<td>24</td>
<td>84</td>
<td>54</td>
<td>77</td>
</tr>
<tr>
<td></td>
<td>QRS</td>
<td>43</td>
<td>24</td>
<td>86</td>
<td>58</td>
<td>79</td>
</tr>
<tr>
<td></td>
<td>QRST</td>
<td>43</td>
<td>24</td>
<td>88</td>
<td>50</td>
<td>76</td>
</tr>
</tbody>
</table>

**TABLE 3. Concordance of Potential Losses Identified With Isopotential Maps in 44 NQMI Patients With Abnormalities Identified Through Different Methods: Acute-Phase ECG, Hypokinesis With Ventriculography, Fixed Perfusion Defect With Thallium Exercise Testing, and Coronary Artery Status**

<table>
<thead>
<tr>
<th>Location</th>
<th>Acute-Phase ECG</th>
<th>Hypokinesis</th>
<th>Fixed perfusion defect</th>
<th>Significant coronary stenosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Midseptal</td>
<td>5/5</td>
<td>7/8</td>
<td>20/22</td>
<td>7/9</td>
</tr>
<tr>
<td>Anterior/Inferior Middle</td>
<td>8</td>
<td>22</td>
<td>4</td>
<td>9</td>
</tr>
<tr>
<td>Anteroseptal Apical</td>
<td>7/9</td>
<td>4/4</td>
<td>2/3</td>
<td>1/2</td>
</tr>
<tr>
<td>Anterosuperior Middle/Basal</td>
<td>4</td>
<td>4</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Inferoapical</td>
<td>3/8</td>
<td>2/3</td>
<td>1/2</td>
<td>2/3</td>
</tr>
<tr>
<td>Inferobasal</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Posterolateral Middle/Basal</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All</td>
<td>52/60 (87%)</td>
<td>55/60 (92%)</td>
<td>27/46 (59%)</td>
<td></td>
</tr>
</tbody>
</table>

Coronary arteries: R indicates infarct related; C, collateral; and I, independent.
How Were the NQMI Features Revealed in QMI?
The initial superior minimum of the midseptal and anterior paraseptal potential loss appeared in anterior QMI where the septum was involved according to the ECG (QS in leads V1 and V2, 21 of 32 cases). In extensive anterior QMI, the isopotential maps showed an extended right anterior negativity that did not allow us to discriminate among anteroseptal/inferior middle, anteroseptal apical, and anteroseptal middle/basal lesions (12 of 32 cases). The breakthrough arrived from above in 6 anterior QMI cases as in the anteroseptal middle/basal NQMI lesion. The clearly inferior QMI (22 of 38) cases showed similar inferior negativity as was seen in inferoapical NQMI. The characteristic right upper late negativity of the inferior basal NQMI location was not revealed in QMI cases. The characteristic late, back-sided minimum of posterolateral basal NQMI was even later in 10 cases with a tall R wave in lead V2. In summary, some of the pathological NQMI changes appeared in QMI, but in general, the larger extent of the necrosis masked the subtle pathological signs of the smaller regions.

Discussion
The main abnormality revealed by isopotential maps in NQMI was the loss of electrical potential, but different features were characteristic for each region of the heart. Therefore, distinctive features are described as both diagnostic and localization criteria for NQMI.

The initial minimum refers directly to the cardiac site of the potential loss, because these regions (midseptal and anterior paraseptal) are the earliest activating part of the heart.14

The early right-sided, slight potential minimum that we associated with an anteroseptal/inferior middle cardiac potential loss was mentioned by Hirai et al9 and De Ambroggi et al11 as a sign of anteroseptal necrosis but by Osugi et al8 as an inferior necrosis sign. Normal isopotential maps did not show this early right-sided minimum.14 How can 2 different types of necrosis result in similar negativeties? In fact, these necrosis sites are close to each other. In both, the early resultant vector is directed toward the apex because of the right-sided anteroseptal or inferoseptal potential loss. However, the resultant vector directs a bit more downward in anteroseptal middle necrosis. Their discrimination is more probable in cases in combination with anteroseptal apical or inferoapical potential losses (Figure 4).

More considerable differences in the vector direction determine the distinctive BSPM features between potential losses of anteroseptal apical and inferoapical regions.

The appearance time of the anterose superior middle/basal potential loss is variable (24 to 42 ms after the QRS onset) depending on the region involved. For instance, with more lateral or basal potential losses, the minimum appears later.

In the quantitative evaluation of minor potential losses, except for 3 regions (midseptal, anterior paraseptal, and anteroseptal apical), the sensitivity of the \( z \) score was very low, probably because of the high SD of the normal values, changes in neighboring leads, or both. The differences in the average values between the normal and pathological groups resulted in better discrimination. How can such minor changes in the isopotential maps indicate potential losses with good sensitivity and specificity? The reason is their very well defined time point, which makes these features discriminative. For example, the minimum value did not differ significantly between posterolateral NQMI and the normal group, but the late timing in the NQMI cases makes this feature a good marker.

Why do isoarea departure maps have worse specificity than the isopotential maps? The isoarea departure maps represent binary decision-making and exclude borderline cases from the subsequent analysis. The period of the entire QRS isopotential map is long, and a minor change lasting only 10 to 20 ms is probably lost within this period. The QRST isoarea maps are overwhelmed by the S wave. In cases of combined potential losses, the different changes can mask or neutralize each other on the isoarea maps.

Confirmation of the diagnosis of old NQMI is problematic. Good concordance with invasive and noninvasive investigations only increases the probability of old NQMI. Coronary occlusion does not cause an infarction in all cases, whereas hypokinesis can also be seen in myocardial hibernation. Moreover, the negativity of these tests does not exclude the existence of NQMI, because spontaneous or pharmacological thrombolysis can open coronary arteries, and hypokinesis or fixed perfusion defect does not necessarily develop.

The total concordance of the hypokinesis (64%) increases when only the middle regions are considered. This means that the potential losses are reflected more readily in hypokinesis for the middle regions. The concordances of the hypokinesis and the fixed perfusion defect were worst for the apical regions (46% and 45%, respectively) because of the proximity of perfusion territories in the apical regions. Collateral and independent coronary arteries together with the infarct-related coronary artery were involved in 67% of cases, which suggests that minor potential losses can appear frequently in multiple vessel disease.

Potential losses of certain specific neighboring regions can manifest themselves in the same acute-phase ECG or coronary state; therefore, the good concordance of BSPMs with acute-phase ECG (90%) and coronary angiography (87%) does not really help much in identification of the actual anatomic regions.

The considerable proportion of isopotential map changes (69%) in patients with UA and their concordances with the other invasive and noninvasive methods that result in similar values as those in NQMI support the suspicion that some patients with UA might have had an undiagnosed NQMI. The other possible reason for the common features is that potential loss may result from severe ischemia without necrosis.

These results may suggest that NQMI is not a distinct pathophysiological entity. There may be potential losses with different extensions in the subendocardial region. The potential losses might also have transmural extensions without pathological Q waves if the regions do not involve the first activating parts of the heart. The common features with UA justify the use of the term “UCAD” for NQMI and UA.

Study Limitations
Because of the detailed localization, the number of cases for some locations is small. We have no autopsy or histological
data. The members of the control group were younger and high standard deviations of the averaged values.

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References
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