Increase in Electrocardiographic R-Waves After Revascularization in Patients With Acute Myocardial Infarction

Satoshi Isobe, MD*,†; Yasuo Takada, MD**; Akitada Ando, MD*; Satoru Ohshima, MD*; Kiyoyasu Yamada, MD*; Mamoru Nanasato, MD*; Kazumasa Unno, MD*; Takuo Ogawa, MD†; Takahisa Kondo, MD*; Hideo Izawa, MD*; Yasuya Inden, MD*; Makoto Hirai, MD††; Toyoaki Murohara, MD*

Background The physiological mechanism of the increase in the electrocardiographic (ECG) R-wave voltage after revascularization in patients with acute myocardial infarction (MI) needs to be elucidated.

Methods and Results One hundred and thirty-eight MI patients (83: anterior MI, 45: inferior MI, 10: lateral MI) underwent ECG and echocardiography in both the acute and subacute phases after emergency revascularization, as well as a resting thallium-201/iodine-123 15-p-iodophenyl-3-(R,S)-methyl pentadecanoic acid myocardial scintigraphy in the acute phase. The total sum of the R-wave voltage (ΣR) was calculated over multiple leads on ECG for each infarcted lesion. Scintigraphic defect on each tracer was expressed as the percentage (%) defect of the total left ventricular (LV) myocardium. The % defect-discordance on both images in the acute phase and the % increase in ΣR and the absolute increase in LV ejection fraction from the acute to the subacute phase (ΔEF) were also calculated. The ΣR in the subacute phase was significantly greater than that in the acute phase (p<0.0001). The % increase in ΣR significantly correlated with the ΔEF (r=0.57, p<0.0001). The % increase in ΣR also correlated with the % defect-discordance (r=0.68, p<0.0001).

Conclusions The increase in the ECG R-wave voltage reflects not only the improvement in myocardial perfusion but also the presence of salvaged myocardium after revascularization in acute MI patients. (Circ J 2006; 70: 1385–1391)

Key Words: Acute myocardial infarction; Echocardiography; Revascularization; R-wave voltage; Tl/BMIPP scintigraphy

A spontaneous increase in the electrocardiographic (ECG) R-wave voltage after acute myocardial infarction (MI) is a unique phenomenon, and regeneration of R-waves or disappearance of Q-waves after acute ischemic events has been reported.1-7 With the prevalence of interventional therapy, it is thought that this phenomenon is increasing, but little is known regarding the physiological mechanism involved.

Patients with acute MI always undergo echocardiography in order to evaluate changes in wall motion, left ventricular (LV) cavity, and LV function over time. Myocardial scintigraphy provides important information about myocardial perfusion and/or metabolism. Specifically, a combined study of myocardial metabolic tracer with perfusion tracer provides clues about the myocardial status in the healing stage of MI8-10. Perfusion–metabolism mismatch of the perfusion/metabolic dual-tracers represents impaired but viable myocardium in the risk area, which is involved in functional recovery after revascularization in patients with acute MI.11-13 Therefore, both examinations are cardinal diagnostic modalities for patients after acute MI.

In this study, we performed dual-isotope scintigraphy and echocardiography, and compared the findings with ECG results to determine the physiological mechanism of the increase in ECG R-wave voltage during the acute to the subacute phase after revascularization in patients with acute MI.

Methods

Population One hundred and seventy-one consecutive patients with first onset of acute MI who underwent emergency coronary revascularization, dual-isotope scintigraphy, and echocardiography between January 1998 and March 2004 were retrospectively examined. Acute MI was diagnosed by typical chest pain for >30 min, ECG ST-elevation of ≥0.1 mV (1 mm) in at least 2 contiguous leads, and a characteristic rise in plasma creatine kinase-MB activity. All patients underwent emergency coronary angiography (CAG) immediately after the ECG diagnosis and subsequent coronary revascularization. In the emergency CAG, antegrade flow was characterized using the Thrombolysis In Myocardial...
Infarction (TIMI) study grading system\textsuperscript{14} Anterior MI was defined by confirming left anterior descending artery (LAD) occlusion, inferior MI by confirming right coronary artery (RCA) occlusion, and lateral MI by confirming left circumflex (LCx) artery occlusion. Proximal culprit lesions were defined as #6 as left anterior descending artery (LAD); #11 as left circumflex (LCx), #1–2 as right coronary artery (RCA); Distal, #7–10 as LAD, #12–15 as LCx, #3–4 as RCA; EF, ejection fraction; CK, creatine kinase.

Electrocardiography

The R-wave voltage in each lead was visually measured using a magnifier on the ECG by 2 observers unaware of the scintigraphic and echocardiographic data. If the patient had an anterior MI, the total sum of the R-wave voltage ($\Sigma R$) in the chest leads (V1–6) was measured, for an inferior MI $\Sigma R$ in II, III, and aVF was measured, and for a lateral MI $\Sigma R$ in I, aVL, V5, and V6 was measured. The $\Sigma R$ was calculated for both the acute and subacute phases. The percentage ($\%$) increase in the $\Sigma R$ was calculated as follows: $\%$ increase in $\Sigma R$ = ([Sum of R-wave voltage ($\Sigma R$) in subacute phase – $\Sigma R$ (acute)]/ $\Sigma R$ (acute)) x 100.

<table>
<thead>
<tr>
<th>Location of MI</th>
<th>Anterior</th>
<th>Inferior</th>
<th>Lateral</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proximal</td>
<td>83 (60%)</td>
<td>45 (33%)</td>
<td>10 (7%)</td>
</tr>
<tr>
<td>Distal</td>
<td>58 (42%)</td>
<td>35 (26%)</td>
<td>47 (32%)</td>
</tr>
<tr>
<td>Distal, #7–10</td>
<td>58 (42%)</td>
<td>35 (26%)</td>
<td>47 (32%)</td>
</tr>
<tr>
<td>Proximal, #6</td>
<td>83 (60%)</td>
<td>45 (33%)</td>
<td>10 (7%)</td>
</tr>
<tr>
<td>Proximal, #11</td>
<td>83 (60%)</td>
<td>45 (33%)</td>
<td>10 (7%)</td>
</tr>
<tr>
<td>Proximal, #12–15</td>
<td>58 (42%)</td>
<td>35 (26%)</td>
<td>47 (32%)</td>
</tr>
<tr>
<td>Proximal, #3–4</td>
<td>58 (42%)</td>
<td>35 (26%)</td>
<td>47 (32%)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>TIMI classification on emergency CAG</th>
<th>Incomplete (0, 1, 2)</th>
<th>Complete (3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>114 (83%)</td>
<td>24 (17%)</td>
</tr>
</tbody>
</table>

Collateral flow

(+): 56 (41%); (-): 82 (59%)

Culprit lesion

Proximal: 80 (58%); Distal: 58 (42%)

Time to reperfusion (h): 4.1±2.6

EF (%): 48±9

Peak CK (IU/L): 2,707±1,402

<table>
<thead>
<tr>
<th>Electrocardiography</th>
<th>Acute</th>
<th>Subacute</th>
<th>$p$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total MI (mV)</td>
<td>1.9±1.7</td>
<td>2.7±2.4</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Anterior MI (mV)</td>
<td>2.7±1.6</td>
<td>3.9±2.3</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Inferior MI (mV)</td>
<td>0.2±0.2</td>
<td>0.4±0.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Lateral MI (mV)</td>
<td>2.9±0.8</td>
<td>3.6±1</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Scintigraphy</th>
<th>% Defect on TI (%)</th>
<th>% Defect on BMIPP (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>23±11</td>
<td>35±14*</td>
</tr>
</tbody>
</table>

Data are mean ±SD.

$\Sigma R$, total sum of R-wave voltage; LVEDD, left ventricular end-diastolic dimension; LVESD, left ventricular end-systolic dimension; LVEF, left ventricular ejection fraction; TI, thallium-201; BMIPP, iodine-123 15-p-isodophenyl-3-(R,S)-methyl pentadecanoic acid. Other abbreviation see in Table 1.

*p<0.0001 vs (%) defect on TI.

Echocardiography

LV dimensions and ejection fraction (LVEF) were measured by an expert in both the acute (1 week) and subacute phases (4 weeks) using echocardiography (Hewlett-Packard, Sonos 2500, Andover, MA, USA) with a 2.5–3.75 MHz transducer. These functional parameters were obtained according to the standard method. In brief, LV end-diastolic and end-systolic diameters were determined in M-mode, and the LVEF was calculated by the method of Teicholz et al.\textsuperscript{15} An absolute increase in LVEF from the acute to the subacute phase ($\Delta$EF) was defined as: $\Delta$EF=LVEF (subacute)−LVEF (acute). Patients who showed a $\Delta$EF $\geq$5% were defined as having experienced a functional recovery whereas those with a $\Delta$EF $<5\%$ showed no recovery.\textsuperscript{16}

Thallium-201 (TI)/Iodine-123 15-p-Isodophenyl-3-(R,S)-Methyl Pentadecanoic Acid (BMIPP) Myocardial Scintigraphy

Resting TI and BMIPP scintigraphy were performed on the same day during the acute phase of MI (7.3±1.9 days after the onset of MI). TI (111 MBq) was injected intravenously and imaging was initiated 15 min later. BMIPP (148 MBq) was injected intravenously and imaging was initiated 30 min later. Imaging was obtained with a single-head rotating gamma camera (Millenium VG, GE Yokokawa, Hino, Japan) equipped with a low-energy, multipurpose collimator. TI-201 images were acquired over a 180-degree arc with an acquisition time of 30 s/image, whereas BMIPP had an acquisition time of 45 s/image. Energy discrimination was provided by a 20% window centered at 70 keV for TI and 159 keV for BMIPP imaging. Data from both im-

<table>
<thead>
<tr>
<th>Gender (M/F)</th>
<th>105/33</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>62±9</td>
</tr>
</tbody>
</table>

### Table 2 Comparison of Changes on ECG, Echocardiography, and Scintigraphy

<table>
<thead>
<tr>
<th></th>
<th>Acute</th>
<th>Subacute</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total MI (mV)</td>
<td>1.9±1.7</td>
<td>2.7±2.4</td>
</tr>
<tr>
<td>Anterior MI (mV)</td>
<td>2.7±1.6</td>
<td>3.9±2.3</td>
</tr>
<tr>
<td>Inferior MI (mV)</td>
<td>0.2±0.2</td>
<td>0.4±0.4</td>
</tr>
<tr>
<td>Lateral MI (mV)</td>
<td>2.9±0.8</td>
<td>3.6±1</td>
</tr>
<tr>
<td>Scintigraphy</td>
<td>% Defect on TI (%)</td>
<td>% Defect on BMIPP (%)</td>
</tr>
<tr>
<td></td>
<td>23±11</td>
<td>35±14*</td>
</tr>
</tbody>
</table>
agages were collected in a 64×64 matrix. Tomographic slices (6 mm thick) of both images were reconstructed by filtered back-projection using a ramp filter relative to the anatomic axis of the LV. Later, vertical long-axis, horizontal long-axis and short-axis slices were generated.

For quantitative analysis of the tracer distribution, a circumferential profile curve was generated from the apical to basal short-axis slices and was used to create a bull’s eye polar map with 100% as a maximum count in each of the TI and BMIPP distributions.7 The areas with less than 80% of the maximum count were defined as defective.18 The extent of the defective areas was expressed as the % defect of the whole myocardium. The % defect-discordance was calculated as the difference in the % defect on both images: (TIMI grade = 3) – (TIMI grade = 0–2) and 24 (17%) patients complete perfusion. Seventeen (83%) patients showed incomplete perfusion (TIMI grade = 0–2) and 24 (17%) patients complete perfusion. One hundred and sixty (78%) patients showed defect discordance exhibited a significantly greater % increase in \( \Delta R \) than those without it (p<0.0001) (Table 3).

### Statistical Analysis

Continuous variables are expressed as the mean ± SD. Comparisons of the data from the acute and subacute phases were done using the paired t-test for variables that were normally distributed. Because echocardiographic parameters (LVEF and LV dimensions) are not distributed normally, the Wilcoxon signed rank test was used to assess differences. Difference in the % defect between TI and BMIPP was analyzed by Student’s t-test. Correlations between continuous variables were assessed using a linear regression analysis. A cut-off value for the % increase in \( \Delta R \) in distinguishing patients with a \( \Delta R \) >5% from those without was defined using receiver-operating characteristic analysis. A p-value <0.05 was considered statistically significant.

### Results

#### Patient Demographics (Table 1)

Of the 138 patients, 83 (60%) had an anterior MI, 45 (33%) had an inferior MI, and 10 (7%) had a lateral MI. Forty-one (30%) patients had a history of pre-infarction angina pectoris, 80 (58%) patients had a proximal occlusion and 58 (42%) had a distal occlusion. One hundred and fourteen (83%) patients showed incomplete perfusion (TIMI grade = 0–2) and 24 (17%) patients complete perfusion (TIMI grade = 3); retrograde collateral flow was observed in 56 (41%) patients on emergency CAG. All patients showed TIMI grade 3 flow after revascularization. Time to reperfusion was ≤6 h in 112 (81%) patients and >6 h in 26 (19%) patients, and the mean time to reperfusion was 4.1±2.6 h. Four patients (3%) only underwent coronary thrombolysis (intra-venous coronary thrombolysis: 2 patients; intra-coronary thrombolysis: 2 patients). 32 (23%) patients had rescue PCI (stent implantation: 21 patients), and 102 (74%) patients had direct PCI (stent implantation: 91 patients).

### Change in ECG R-Wave Voltage

The \( \Sigma R \) showed an excellent correlation between the first and second analysis by the same observer (r=0.99, p<0.0001) or between 2 independent observers (r=0.99, p<0.0001). The Bland-Altman plots demonstrated no significant bias or correlation between mean values and difference for \( \Sigma R \). A significant increase in \( \Sigma R \) was observed from the acute to the subacute phase in all patients (p<0.0001) (Table 2). Significant increases in \( \Sigma R \) were also observed in each patient (p<0.01, respectively) (Table 2). The % increase in \( \Sigma R \) was significantly greater in patients who had complete TIMI flow, retrograde collateral flow, and a distal culprit lesion than in those who did not have these factors (p<0.01) (Table 3). The % increase in \( \Sigma R \) was also significantly greater in patients with pre-infarction angina and an early time to reperfusion (<6 h) than in those without (p<0.0001) (Table 3). For all patients, the sensitivity, specificity and accuracy for distinguishing patients with a \( \Delta R \) >5% from those without was 96%, 44%, and 81%, respectively, at 10% cut-off value for the % increase in \( \Sigma R \). The sensitivity, specificity and accuracy for distinguishing patients was 95%, 43% and 81%, respectively, for anterior MI patients, and 96%, 41% and 89%, respectively, for inferior MI patients at 10% cut-off value for the % increase in \( \Sigma R \). For lateral MI patients, the sensitivity, specificity and accuracy was 83%, 100%, and 80%, respectively, at 8% cut-off value for the % increase in \( \Sigma R \). One hundred and seventeen (85%) patients showed % increase in \( \Sigma R \) ≥10%.

### Changes in Cardiac Functional Parameters

The LVEF show an excellent correlation between the first and second analysis by the same observer (r=0.98, p<0.0001). The Bland-Altman plot demonstrated no significant bias or correlation between mean values and difference for LVEF. LV end-systolic dimension significantly decreased from the acute to the subacute phase (p<0.0005), whereas the LV end-diastolic dimension did not change significantly (Table 2). LVEF significantly improved from the acute to the subacute phase (p<0.0001) (Table 2). Ninety-eight (71%) patients showed a \( \Delta E F \) ≥5% and 40 (29%) a \( \Delta E F \) <5%. Patients with a \( \Delta E F \) ≥5% exhibited a significantly greater % increase in \( \Sigma R \) than those with a \( \Delta E F \) <5% (p<0.0001) (Table 3).

### Dual-Tracer Findings

One hundred and eight (78%) patients showed defect discordance on both images. The % defect on BMIPP was significantly greater than that on TI (p<0.0001) (Table 2). A significant correlation was observed between the % defect on TI and that of BMIPP (r=0.86, p<0.0001). Patients with defect discordance exhibited a significantly greater % increase in \( \Sigma R \) than those without it (p<0.0001) (Table 3).

### Table 3 Difference in % Increase in \( \Sigma R \)

<table>
<thead>
<tr>
<th>Variable</th>
<th>TIMI flow</th>
<th>Collateral</th>
<th>( \Delta E F )</th>
<th>Defect-discordance</th>
<th>Culprit lesion</th>
<th>Pre-infarction angina</th>
<th>Time to reperfusion</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>III</td>
<td>0–II</td>
<td>5%≤</td>
<td>5%&gt;</td>
<td>Proximal</td>
<td>Distal</td>
<td>(+)</td>
</tr>
<tr>
<td>% Increase in ( \Sigma R ) (%)</td>
<td>51±22</td>
<td>33±21</td>
<td>&lt;0.001</td>
<td>47±19</td>
<td>37±23</td>
<td>&lt;0.0001</td>
<td>57±30</td>
</tr>
<tr>
<td>p value</td>
<td>&lt;0.001</td>
<td>&lt;0.0001</td>
<td>&lt;0.0001</td>
<td>&lt;0.0001</td>
<td>&lt;0.0001</td>
<td>&lt;0.0001</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

Data are mean ± SD.

% Increase in \( \Sigma R \), percentage increase in total sum of R-wave voltage; \( \Delta E F \), absolute increase in ejection fraction. Other abbreviations see in Table 1.
Comparison of ECG Results With Echocardiographic or Scintigraphic Findings

The % increase in $\sum R$ significantly correlated with the $\Delta EF$ ($r=0.57, p<0.0001$) (Fig 1A) and the % defect on Tl ($r=0.40, p=0.0003$), whereas it did not correlate with the % defect on BMIPP ($r=0.08, p=NS$). The % increase in $\sum R$ also significantly correlated with the % discordance on TI/BMIPP ($r=0.68, p<0.0001$) (Fig 2A).

When we analyzed these relationship only in the patients with anterior MI, significant correlations were observed in (A) all patients, (B) anterior myocardial infarction (MI) patients, (C) inferior MI patients, (D) lateral MI patients.

Fig 1. Significant correlation between the % increase in the total sum of the R-wave voltage ($\sum R$) and subacute phase ($\Delta EF$) in (A) all patients, (B) anterior myocardial infarction (MI) patients, (C) inferior MI patients, (D) lateral MI patients.

Fig 2. Significant correlation between the % increase in the total sum of the R-wave voltage ($\sum R$) and the % defect-discordance in (A) all patients, (B) anterior myocardial infarction (MI) patients, (C) inferior MI patients, (D) lateral MI patients.
Increase in ECG R-Waves After MI

Circulation Journal   Vol.70, November 2006

between the % increase in $\Sigma R$ and the $\Delta$EF and between the $\%$ increase in $\Sigma R$ and the $\%$ defect-discordance on TI/BMIPP ($p<0.0001$, respectively) (Figs 1B,2B). When we analyzed them only in patients with inferior MI, both the correlation between the $\%$ increase in $\Sigma R$ and the $\Delta$EF and between the $\%$ increase in $\Sigma R$ and the $\%$ defect-discordance on TI/BMIPP was still significant, but became worse ($p<0.001$, respectively) (Figs 1C,2C). When we analyzed them only in patients with lateral MI, the aforementioned correlations were excellent ($p<0.001$, respectively) (Figs 1D,2D).

We show the ECG findings and bull’s-eye polar map images of a typical patient in Fig 3A and B, respectively. The $\Delta$EF of this patient was 9%.

**Discussion**

With recent technical progress in interventional therapy, the assessment of salvageable myocardium is important for judging the efficacy of treatment. This assessment plays a pivotal role in the clinical decision-making process. Myocardial scintigraphy can provide more detailed information about myocardial status, such as the extent of impaired myocardium. In particular, a combined study of metabolic tracer with perfusion tracer is useful for assessing salvaged myocardium after revascularization in patients with MI. However, dual-isotope myocardial scintigraphy takes time, is somewhat expensive, and uses radiation. Echocardiography is normally used for the evaluation of wall motion recovery and LV functional evolution, but is technician-dependent and imaging may be subject to unavoidable operator-dependent error. Another disadvantage of echocardiography is the variable acoustic windows in patients with underlying lung diseases, which technically limits the studies because of poor definition of the heart. Electrocardiography, which is less expensive, very convenient, and the most available and widespread modality, is useful in diagnosing acute MI in patients having acute chest pain. Regeneration of the R-waves instead of disappearance of the Q-waves has often been reported after acute MI, but the exact mechanism in relation to the time course of such ECG changes or the clinical relevance of this phenomenon has not been well characterized. Therefore, it is of great interest to determine the physiological mechanism of the increase in R-wave voltage after acute MI, as well as investigating whether the evaluation of such ECG findings could...
provide information regarding myocardial status or functional evolution after revascularization in acute MI patients.

In our previous study involving only patients with acute anterior MI, R-wave recovery correlated with both the extent of viable myocardium and functional recovery. In the present study, similar results were obtained, indicating that R-wave recovery reflects viable myocardium and subsequent functional recovery in any type of infarction. Moreover, the magnitude of the R-wave recovery is greater in acute MI patients with distal culprit lesions or pre-infarction angina than in those without it. R-wave recovery also reflects the extent of impaired myocardium and the presence of ischemic preconditioning.

Previous studies have demonstrated that ECG evaluations do not show sufficiently close correlations with LV function or infarct size.22,23 The discrepancies of the present results and those previous studies may be explained by the more advanced techniques of PCI or other therapeutic strategies after MI, earlier reperfusion and a larger study population in our study.

A previous study reported that patients showing T-wave normalization despite persistent abnormal Q-waves had improvement in LVEF and regional wall motion.24 We did not evaluate the time course of changes in the T-wave. However, some patients (23) without an increase in R-waves showed improvement in LVEF, which may accord with the results of the previous study.

The increase in R-wave voltage significantly correlated with defect on TI but not with that on BMIPP. The increase in R-wave voltage also correlated with scintigraphic discordance. The defect on perfusion tracers indicates the infarct area whereas that on BMIPP reflects the risk area at concordance. The defect on perfusion tracers indicates the extent of impaired myocardium and the presence of ischemic preconditioning.

The local reversible loss of electrical activity in impaired myocardium.25 Although we did not assess the time course of changes in myocardial perfusion, a smaller defect on TI than BMIPP in the acute phase can indicate the recovery of perfusion in the risk area after revascularization. In addition, patients with complete TIMI flow or retrograde collateral flow on initial CAG showed a greater increase in the R-wave than those without it. Our results support those of a previous study that demonstrated that disappearance of Q-waves is consistent with improved myocardial perfusion as assessed by TI scintigraphy.5 In general, perfusion–metabolism mismatch findings indicate the presence of salvaged myocardium in the risk area after acute MI11–13 Such an area is called “impaired but reversible” myocardium in the early healing stage of MI. The R-wave recovery may relate to the presence of reversible myocardium after acute MI. Thus, assessing the time course of the changes in ECG may allow us to infer functional evolution and to estimate the magnitude of salvaged myocardium.

We could not determine the underlying physiological mechanism of the increase in the ECG R-wave voltage, although some possibilities have been proposed. Reversible ischemic myocardial hemorrhage and edema may produce a temporary Q-wave or dynamic changes in the vessel tone of the infarct-related artery may play a role in the alterations of electrical voltage in the damaged myocardium.27 The local reversible loss of electrical activity in impaired but reversible myocardial tissue may be involved in electrophysiological changes such as a regeneration of R-waves instead of disappearance of Q-waves.27 Presumably, dynamic and temporary changes in the vascular area or myocardium would appear because all patients successfully underwent revascularization. The increase in R-wave voltage that was frequently found in our study relates to an amelioration of the impaired vascular tone and myocardium because all patients showed both residual stenosis <50% and TIMI grade 3 flow after revascularization.

A major limitation of this study was the small patient population, especially only 10 patients with a lateral MI. We did not estimate the time course of changes in the dual-isotope images because we performed them only once weekly after MI. As electrical voltage cancellation may be a problem in calculating R-wave amplitude and marked changes in cardiac size after MI may hinder the precise evaluation of R-wave changes, we focused on patients in a stable condition after acute MI and excluded those with multivessel disease or congestive heart failure. It is reported that acute MI patients with intermediate QRS prolongation with bundle-branch block have a lower 6-month mortality than those without bundle-branch block.28 We could not address the clinical significance of this phenomenon because we did not follow patients over a long enough period. This warrants further investigations.

In summary, an increase in the ECG R-wave is associated with improvement in myocardial perfusion. The change in R-wave voltage is associated with not only LV functional recovery but also the presence of salvaged myocardium in the risk area. Monitoring the ECG complements results from echocardiography or myocardial scintigraphy and can be an important clinical tool in evaluating the evolution of cardiac function or prognosis after revascularization in patients with acute MI. ECG examination is a less expensive, very convenient, and a useful modality for assessing myocardial status.

Acknowledgments
We wish to thank Joey Franzone II for reviewing the manuscript and providing invaluable suggestions.

References


