RELATIONSHIP BETWEEN CHARACTERISTICS OF ENDOCARDIAL ELECTROGRAMS AND REGIONAL WALL MOTION ABNORMALITIES IN OLD MYOCARDIAL INFARCTIONS

CHIHARU CHINO, M.D.

In 40 patients with old myocardial infarction (MI), we evaluated the relationship between characteristics in endocardial electrograms and regional wall motion abnormalities at the MI site. Left ventricular ejection fraction (LVEF) was greater in the late-potentials (LPs) negative group (n=21) than in the LPs positive group (n=19), but this difference was not significant (57.6±19.8% vs 47.9±16.8%). Regional wall motion was assessed by the radial method. We used the percent left ventriculogram (%LVG) area change of each segment as a marker of regional wall motion. Correlation coefficients between electrographic amplitude and %LVG area change, electrographic duration and %LVG area change, and amplitude/duration ratio and %LVG area change were 0.3999 (p<0.001), −0.2519 (p<0.02), and 0.4312 (p<0.001), respectively. Thus, characteristics of endocardial electrograms were significantly related to regional wall motion abnormalities in MI sites. In MI sites, as regional wall motion was reduced, the electrographic amplitude became smaller and the electrographic duration became longer. However, this relationship was not linked to that between LPs and LVEF. (Jpn Circ J 1994; 58: 877–884)

VENTRICULAR arrhythmias, such as ventricular tachycardia (VT) and ventricular fibrillation, are the main causes of death in patients who survive an acute myocardial infarction. Signal-averaged electrocardiography (SA-ECG) has recently been developed as a technique for evaluating ventricular arrhythmias, complementing studies of ambulatory ECG and programmed electrical stimulation.1,2 Body surface late potentials (LPs) on SA-ECG have been reported to predict the occurrence of VT and sudden death in old myocardial infarction (OMI) noninvasively3,4 Both left ventricular dysfunction and LPs can predict the prognosis

<table>
<thead>
<tr>
<th>TABLE I PATIENTS' CHARACTERISTICS</th>
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<tbody>
<tr>
<td>Patients (N)</td>
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<tr>
<td>Mean age (yr)</td>
</tr>
<tr>
<td>Gender (N)</td>
</tr>
<tr>
<td>Male</td>
</tr>
<tr>
<td>Female</td>
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<tr>
<td>Infarct location (N)</td>
</tr>
<tr>
<td>Anterior</td>
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<tr>
<td>Inferior</td>
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<tr>
<td>Anterior+Inferior</td>
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<td>LVEF (%)</td>
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<tr>
<td>Total</td>
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<tr>
<td>Anterior</td>
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<td>Inferior</td>
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<td>Anterior+Inferior</td>
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LVEF=left ventricular ejection fraction.

Key words:
Endocardial electrogram
Regional wall motion
Signal-averaged electrocardiogram
Late potential
Left ventricular dysfunction

(Received October 20, 1993; accepted March 18, 1994)
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Fig. 1. Scheme for evaluating regional wall motion of the anterolateral, septal, diaphragmatic, and posterolateral regions by the radial method. The shaded areas represent the infarction area. MI, myocardial infarction.

Fig. 2. Scheme for endocardial catheter mapping. Left ventricle is divided into 12 segments according to the recommendations of the Committee on Nomenclature of Myocardial Wall Segments of the International Society of Computerized Electrocardiology. Anteroseptal, anterosuperior, inferior and posterolateral correspond to septal, anterolateral, diaphragmatic and posterolateral in Fig. 1, respectively.

of these events after myocardial infarction (MI) as a predictor, LPs have been reported to be independent of ventricular dysfunction while other reports have found a good correlation between the incidence of LPs and impaired left ventricular function. Zimmermann et al reported a higher incidence of LPs in patients with left ventricular aneurysm in the absence of VT. The incidence of LPs is reported to be influenced by the site of MI. In cases of OMI, LPs have been considered to originate with abnormal endocardial electrograms, such as fragmented or delayed electrograms of injured myocardium. Therefore, in this study, we evaluated the relationship between the characteristics of left ventricular endocardial electrograms and regional wall motion abnormalities in patients with OMI.

METHODS

Study Population

The study group consisted of 40 patients with previous MI who were admitted to the Shinshu University Hospital between October 1989 and February 1992 (Table I). The diagnosis of acute myocardial infarction was based on the occurrence of chest pain, ST elevation on ECG, and a significant increase in the level of total serum creatine kinase. Infarct location was anterior in 22 patients, inferior in 14, and both anterior and inferior in the remaining 4. None of the patients had bundle branch block and all were in sinus rhythm. There were 33 men and 7 women with a mean age of 58 ± 9 years, (range 38 to 77 years). All of the patients underwent left ventriculography (LVG), selective coro-
Endocardial Electrograms and Regional Wall Motion Abnormalities

Fig. 3. Endocardial electrogram (LV). II and V₅ are surface electrocardiographic recordings.

Fig. 4. Comparison of LVEF between LPs-positive patients and LPs-negative patients. Data are expressed as the mean ± SD. LVEF, left ventricular ejection fraction; LPs, late potentials.

Oblique (RAO) projection and the 60-degree left anterior oblique (LAO) projection. Left ventricular volumes and ejection fraction (LVEF) were computed according to the method of Chapman. Only sinus beats were used for analysis and premature ventricular contractions and post premature beats were disregarded. Left ventricular wall motion abnormalities were assessed by the radial method.

Regional Wall Motion Analysis
Cine films were projected and both end-diastolic and end-systolic endocardial contours were traced. A normal non-post-premaature beat was used. Analysis by the radial (polar) system was performed using the Cardio 200 from Contron Electronics (Munich, Germany). In the RAO 30-degree view, the major axis was defined as the line from the apex to the midpoint of the aortic valve plane. Lines drawn from the midpoint of the major axis to the ventricular outline were used to divide it into 12 segments. In each of the 12 segments, the percent left ventriculogram (%LVG) area change was measured. As illustrated in Fig. 1, the %LVG area change of one segment was used to reflect anterolateral wall motion and diaphragmatic wall motion. In the LAO 60-degree view, the major axis was not defined and the center of gravity was used as a reference point. Again, the ventricular outline was divided into 12 segments and %LVG area change of each segment was measured. The %LVG area change of one segment was

Cardiac Catheterization
CAG and LVG were performed by the femoral approach, using conventional techniques. Left ventriculograms were performed in both the 30-degree right anterior

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TABLE II CHARACTERISTICS OF ENDOCARDIAL ELECTROGRAMS FOR MI SITES AND NMI SITES

<table>
<thead>
<tr>
<th></th>
<th>MI site (N=111)</th>
<th>NMI site (N=70)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amplitude (mV)</td>
<td>1.2±0.9</td>
<td>2.7±1.9</td>
<td>p&lt;0.001</td>
</tr>
<tr>
<td>Duration (msec)</td>
<td>63.4±15.9</td>
<td>54.7±11.1</td>
<td>p&lt;0.001</td>
</tr>
<tr>
<td>Amplitude/duration</td>
<td>0.021±0.017</td>
<td>0.050±0.034</td>
<td>p&lt;0.001</td>
</tr>
</tbody>
</table>

MI, myocardial infarction; NMI, non-myocardial infarction.

used to reflect septal wall motion and posterolateral wall motion. Anterolateral and septal walls were defined as MI sites in anterior MI, and diaphragmatic and posterolateral walls were defined as MI sites in inferior MI.

Endocardial Catheter Mapping
After CAG and LVG, a quadripolar catheter (5F USCI) was inserted percutaneously into the femoral artery and advanced to the left ventricle under fluoroscopic guidance. The catheter had an interelectrode distance of 5 mm and the electrode rings were 2-mm wide. Left ventricular mapping was performed according to the recommendations of the Committee on Nomenclature of Myocardial Wall Segments of the International Society of Computerized Electrocardiology (Fig. 2). Only endocardial electrograms recorded in the shaded areas in Fig. 1 were included in the analysis. The catheter site was verified by multiple-plane fluoroscopy, in essentially the RAO 30-degree and LAO-60 degree views. Stability was ensured by recording for a minimum of 5 sec. Electrograms were recorded with a 5-mm interelectrode distance between the distal and second electrode rings. All electrograms were filtered at 50 to 300 Hz and recorded at a paper speed of 100 mm/sec. We evaluated the characteristics of endocardial electrograms as specified by Cassidy et al. Electrophographic amplitude (in mV) was defined as the peak-to-peak deflection. Electrophographic duration (in msec) was defined as the time from the earliest electrical activity that deviated from a stable baseline to the onset of the amplification signal decay artifact (Fig. 3).

SA-ECG

Signal-averaging of the surface QRS complex was performed using a VCM 3000 (Fukuda Denshi Company, Tokyo), according to the method described by Simson! The signal of three standard orthogonal bipolar X, Y, and Z leads for 200 beats was amplified, digitized, and filtered. A bandpass filter of 40 to 300 Hz was used. The filtered leads were combined into a vector magnitude $\sqrt{X^2+Y^2+Z^2}$. The onset and termination of the QRS complex were determined at positions that were more than twice the amplitude of baseline noise. The presence of LPs was defined as $\text{RMS}_{40} \leq 16 \mu \text{V}$.

Statistical Analysis
All results are expressed as the mean±SD. Student's t-test for unpaired data was used.

RESULTS
The mean total LVEF in all 40 patients was 53.9±18.6%. The mean LVEF in anterior, inferior, and both anterior and inferior infarction was 53.0±19.9%, 61.6±12.7% and 31.3±8.5%, respectively (Table I). LPs was positive in 19 of 40 patients (47.5%). Ten of 22 patients with anterior infarction (45.5%) and 8 of 14 patients with inferior infarction (57.1%) had a positive LPs. Of the 4 patients with both anterior and inferior infarctions, one was LPs positive (25%). LVEF in the LPs-positive patients was 49.7±16.8% and that in the LPs-negative group was 57.6±19.8% (N.S., Fig. 4).

A total of 181 electrograms were obtained from all of the patients for quantitative analysis of the characteristics of amplitude and duration. To estimate the relation between endocardial electrograms and regional wall motion abnormalities, electro-
Endocardial Electrograms and Regional Wall Motion Abnormalities

![Graph 1: Correlation between the amplitude of endocardial electrograms and %LVG area change.](image)

![Graph 2: Correlation between the amplitude/duration ratio of endocardial electrograms and %LVG area change.](image)

sites vs NMI sites was $1.2 \pm 0.9 \text{ mV} \text{ vs } 2.7 \pm 1.9 \text{ mV} \quad (p < 0.001)$. The mean electrographic duration at MI sites vs NMI sites was $63.4 \pm 15.9 \text{ msec} \text{ vs } 54.7 \pm 11.1 \text{ msec} \quad (p < 0.001)$. The mean amplitude/duration ratio at MI sites vs NMI sites was $0.021 \pm 0.017 \text{ vs } 0.050 \pm 0.034 \quad (p < 0.001) \quad (\text{Table II}).$

**Regional Wall Motion**

Percent LVG area change at MI sites vs NMI sites was $13.2 \pm 20.2\% \text{ vs } 50.1 \pm 15.6\% \quad (p < 0.001)$ in the septal region, $3.6 \pm 17.1\% \text{ vs } 69.6 \pm 13.9\% \quad (p < 0.001)$ in the anterolateral region, $20.5 \pm 17.6\% \text{ vs } 77.1 \pm 19.5\% \quad (p < 0.001)$ in the diaphragmatic region, and $26.4 \pm 16.7\% \text{ vs } 68.0 \pm 14.6\% \quad (p < 0.001)$ in the posterolateral region.

**Relationship Between Characteristics of Endocardial Electrograms and Regional Wall Motion Abnormalities at MI Sites**

Significant correlations were observed between electrographic amplitude and %LVG area change ($r = -0.3999, \ p < 0.001$) (Fig. 5), electrographic duration and %LVG area change ($r = -0.2319, \ p < 0.02$) (Fig. 6), and amplitude/duration ratio and %LVG area change ($r = 0.4312, \ p < 0.001$) (Fig. 7).

**DISCUSSION**

Both left ventricular dysfunction and LPs

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are prognostic predictors after MI. Numerous reports have discussed the relationship between LPs and left ventricular dysfunction in MIs. Pollak et al and Gomes et al reported that there was no relationship between LPs and either LVEF or regional wall motion abnormalities. They concluded that LPs was an independent predictor of global or regional left ventricular function. However, a significant relationship has been reported to exist between the incidence of LPs and left ventricular dysfunction. Breithardt et al reported that LPs occurred more frequently in patients with ventricular akinesia or aneurysm than in those without those conditions. Chillou et al reported a good correlation between the incidence of LPs and impaired LVEF or coronary artery occlusion. Thus, the relationship between LPs and left ventricular dysfunction is controversial.

SA-ECG is a noninvasive technique for detecting abnormal endocardial electrograms. Abnormal electrograms, such as fragmented electrograms and delayed electrograms, indicate slow conduction that is required for the formation of the re-entry circuit. LPs on SA-ECG have been considered to originate with abnormal endocardial electrograms. Simson reported that abnormal electrograms, such as fragmented electrograms, were all recorded from MI sites. Although the relationship between LPs and left ventricular dysfunction has been studied in detail, that between abnormal endocardial electrograms and regional wall motion abnormalities has been studied only rarely. Directly evaluating the relationship between endocardial electrograms and regional wall motion abnormalities at the MI site should help to clarify the relationship between LPs and left ventricular dysfunction.

Characteristics of Left Ventricular Endocardial Electrograms

We estimated the relationships between characteristics of endocardial electrograms and regional wall motion recorded in septal, anterolateral, diaphragmatic and posterolateral sites, as shown in Fig. 1. Since Josepshon et al recorded fragmentary activity from the endocardium in patients with VT, there have been many reports about ventricular catheter mapping. Cassidy et al compared electrograms of sites at which VT originated with those of sites at which VT did not originate in 52 patients with OMIs. They reported that electrograms from the site of VT origin were of significantly lower amplitude and longer duration, but did not comment about any differences between MI sites and NMI sites. In the present study, comparison of the characteristics of electrograms from MI sites and NMI sites revealed significant differences in amplitude, duration and the amplitude/duration ratio (Table II). In electrograms of normal left ventricles, Cassidy et al reported an amplitude of $6.7 \pm 3.4 \text{ mV}$, a duration of $54 \pm 13 \text{ msec}$, and an amplitude/duration ratio of $0.133 \pm 0.073$.

**Relationship Between Characteristics of Endocardial Electrograms and Regional Wall Motion Abnormalities at MI Sites**

In this study, LVEF was greater in LPs-negative patients than in LPs-positive patients, but this difference was not significant (Fig. 4). This finding agrees with those of Gomes et al. We evaluated the relationship between characteristics of endocardial electrograms and regional wall motion abnormalities at MI sites. There were significant relationships between three characteristics of endocardial electrograms and %LVG area change (Figs. 5–7), which suggests that endocardial electrograms were influenced by regional wall motion abnormalities at MI sites. As regional wall motion was reduced, the electrographic amplitude became smaller and the electrographic duration became longer. Boinneau and Cox reported in dogs that desynchronized long-lasting electrical activity was recorded mainly in the center of the ischemic area. The myocardium in this area was shown to be the most damaged by histochemical staining. The prolongation of electrographic duration was considered a marker of conduction delay. However, this relationship between endocardial electrograms and regional wall motion abnormalities was not linked to that between LPs and LVEF. Mehra et al studied isochronal maps of ventricular activation during ventricular arrhythmias induced by programmed premature stimulation in dogs. They reported that both the zone of the conduction delay and

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the arc of the conduction block were localized within the visible epicardial border of the infarction. The prolongation of electrographic duration recorded at MI sites does not seem to have a direct connection with the formation of the re-entry circuit. We believe that this can explain why the relationship between endocardial electrograms and regional wall motion abnormalities at MI sites is not linked to that between LPs and LVEF.

Limitations

This was a retrospective study and the analysis was performed on a selected population. Another limitation involves the technique of catheter mapping. As Cassidy noted, precise positioning and stabilization of the catheter is sometimes difficult. The reproducibility of endocardial electrograms can also be uncertain. A third limitation centers on the definition of MI sites and NMI sites. We used the septal and anterolateral walls as MI sites in anterior MI and the diaphragmatic and posterolateral walls as MI sites in inferior MI. However, the range of infarction varied in each case. Thus, it was sometimes unclear whether endocardial electrograms recorded in a particular region actually reflected the actual region of the infarction.

Acknowledgements

I wish to thank Professor Seiichi Furuta of Shinshu University School of Medicine and Dr. Yasuyuki Sasaki for their important and useful advice.

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