RELATIONSHIP BETWEEN LATE POTENTIALS AND LEFT VENTRICULAR FUNCTION IN PATIENTS WITH CORONARY ARTERY DISEASE

TSUYOSHI KONTA, M.D., KOZUE IKEDA, M.D., ISAO KUBOTA, M.D.
KAI TSUIKI, M.D., AND SHOJI YASUI, M.D.

We examined the relationship between late potentials and left ventricular function from a hemodynamic point of view in 50 patients with prior myocardial infarction. Late potentials were found in 15 (30%) of 50 patients. A left ventricular aneurysm was found in 28 patients. Late potentials were detected in 14 (50%) of 28 patients with the aneurysm but in 1 (5%) of 22 patients without it (p < 0.01). In the 50 patients, a hemodynamic data from the late potential positive group (n = 15) were compared to those from the late potential negative group (n = 35). The late potential positive group had a significantly lower ejection fraction, cardiac index and stroke volume than the late potential negative group. We further studied the 28 patients with left ventricular aneurysm in a similar way. The cardiac index and stroke volume were also significantly lower in the late potential positive group. The ejection fraction tended to be lower in the late potential positive group. These results suggest that left ventricular function and left ventricular aneurysm are among the factors that influence the development of late potentials.

Signal-averaged electrocardiography was developed as a technique for noninvasive measurement of late and slow activation which is associated with reentrant ventricular arrhythmias. Fractionated electrocardiograms have been recorded during endocardial or epicardial mapping in patients with ventricular tachycardia and ischemic heart disease. These fractionated electrocardiograms have been correlated with the body surface late potentials on signal-averaged electrocardiograms. Recently, in patients with coronary artery disease or dilated cardiomyopathy, the presence of abnormalities of the signal-averaged electrocardiogram has successfully identified patients at high risk of sustained ventricular tachycardia or sudden death. On the other hand, few studies have examined the correlation between late potentials and left ventricular function. Several studies have suggested a relationship between late potentials and left ventricular aneurysm. However, Pollak et al. reported that late potentials were independent of global or regional left ventricular function. These reports did not include hemodynamic studies, although hemodynamic data are very important when discussing ventricular function. In this study, we detected late potentials using a microprocessor-augmented ECG cart and examined the relation between late potentials and left ventricular function from a hemodynamic point of view.

METHODS

Subjects
There were 50 patients with previous myocardial infarction (42 men and 8 women) who

Key words:
Late potentials
Left ventricular function

(Received May 21, 1987; accepted August 22, 1987)
The First Department of Internal Medicine, Yamagata University School of Medicine, Yamagata, Japan
Mailing address: Tsuyoshi Konta, M.D., The First Department of Internal Medicine, Yamagata University School of Medicine, Zao-Iida, Yamagata 990-23, Japan

Japanese Circulation Journal Vol. 52, February 1988 105
underwent left ventriculography and selective coronary arteriography in Yamagata University Hospital from March 1981 to July 1986. The patients satisfied all the following criteria: (1) a clinical diagnosis of myocardial infarction established by typical chest pain and serum enzyme changes; (2) significant stenosis of 70% or more in at least one major coronary artery; (3) absence of other forms of heart disease such as congenital heart disease or myocardial disease; and (4) no conduction disturbance such as right bundle branch block, left bundle branch block or Wolff-Parkinson-White syndrome. The mean age was 56.8 years (range 35–73 years). All the patients entered the study more than 3 months (average 15 months) after the onset of acute myocardial infarction. All the patients gave informed consent before the study.

*Signal-averaged electrocardiography*

All subjects had signal-averaged electrocardiography in sinus rhythm. The electrocardiographic recording technique included the placement of four silver/silver chloride ECG electrodes (Marquette Electronics) in the following locations: the routine V1, V5 and V6R position. A fourth neutral electrode was positioned in the manubrium sterni region. Signals were recorded and processed using a microprocessor-augmented ECG cart, MAC 1 (Marquette Electronics).

Output filters consisted of a three-pole Butterworth low-pass filter at 300 Hz and high-pass filters at 25, 50, 100 and 200 Hz. For illustrative purposes all recordings on this paper were selected with a 100 Hz cut off. The trigger or fiducial point was based on the earliest onset of QRS activity. Paper speed was 200 mm/s. Data output consisted of six simultaneous leads (V5 - (V1 + V6R)/2, V5 - V1, (V6R + V5)/2 - V1, V6R - V1, V6R - (V1 + V5)/2 and V6R - V5).

The hard copy data format consisted of an ink printout of the six leads, each having the averaged signal represented at three different gains and four different frequency bands. In each patient, 512 or 1024 cycles were averaged.

The duration of late potentials was measured as the time from the end of the QRS complex to the point of the voltage exceeding twice the noise level. Late potentials were defined as waveforms with a duration over 20 ms after the end of the QRS complex. A duration of low-amplitude activity in normal subjects was shown to be less than 10 ms and the waveforms with a duration of over 20 ms after the end of the QRS complex were taken to be late potential positive\(^2\). The end of the QRS complex was determined by two or more electrocardiologists.

*Left ventriculography and coronary arteriography*

Biplane left ventriculograms in the 30° right anterior oblique and 60° left anterior oblique projections were recorded on 35 mm film taken at 50 frames/s with the Toshiba 9 inch image amplifier system (Angiorex/U-arm). The left ventricle was made opaque by the injection of a contrast medium (Meglumine diatrizoate) into the left ventricle at a rate of 13 ml/s for 3 s. The left ventricular ejection fraction and left ventricular end-diastolic volume were calculated from biplane left ventriculograms according to the area length method of Dodge et al\(^19\). Cardiac output and stroke volume were determined by the dye-dilution technique. Mean aortic, left ventricular and pulmonary capillary wedge pressures were measured through fluid-filled catheters. Left ventricular aneurysm was defined as an aneurysmal bulging with sharply defined margins of ventricular segment\(^20\). Selective

TABLE I  RELATIONSHIP BETWEEN LATE POTENTIALS AND HEMODYNAMIC DATA IN ALL PATIENTS

<table>
<thead>
<tr>
<th></th>
<th>LP (+) n = 15</th>
<th>LP (-) n = 35</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>EF (%)</td>
<td>36.3 ± 11.0</td>
<td>49.9 ± 11.7</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>CI (l min⁻¹ m⁻²)</td>
<td>2.5 ± 0.5</td>
<td>3.1 ± 1.0</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>LVEDV (ml)</td>
<td>289.3 ± 143.3</td>
<td>224.8 ± 65.0</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>SV (ml)</td>
<td>53.3 ± 19.1</td>
<td>72.8 ± 20.1</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>LVEDP (mmHg)</td>
<td>16.5 ± 8.9</td>
<td>14.6 ± 5.0</td>
<td>NS</td>
</tr>
<tr>
<td>PCWP (mmHg)</td>
<td>10.3 ± 8.1</td>
<td>7.9 ± 3.9</td>
<td>NS</td>
</tr>
<tr>
<td>mAoP (mmHg)</td>
<td>96.3 ± 8.2</td>
<td>97.4 ± 12.5</td>
<td>NS</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>76.3 ± 14.5</td>
<td>69.7 ± 13.8</td>
<td>NS</td>
</tr>
</tbody>
</table>

Abbreviations: LP = late potentials; EF = ejection fraction; CI = cardiac index; LVEDV = left ventricular end-diastolic volume; SV = stroke volume; LVEDP = left ventricular end-diastolic pressure; PCWP = pulmonary capillary wedge pressure; mAoP = mean aortic pressure; HR = heart rate

TABLE II  RELATIONSHIP BETWEEN LATE POTENTIALS AND HEMODYNAMIC DATA IN PATIENTS WITH LEFT VENTRICULAR ANEURYSM

<table>
<thead>
<tr>
<th></th>
<th>LP (+) n = 14</th>
<th>LP (-) n = 14</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>EF (%)</td>
<td>35.3 ± 10.7</td>
<td>44.3 ± 13.7</td>
<td>NS</td>
</tr>
<tr>
<td>CI (l min⁻¹ m⁻²)</td>
<td>2.5 ± 0.5</td>
<td>3.0 ± 0.8</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>LVEDV (ml)</td>
<td>294.3 ± 147.1</td>
<td>240.8 ± 88.6</td>
<td>NS</td>
</tr>
<tr>
<td>SV (ml)</td>
<td>52.3 ± 19.4</td>
<td>70.0 ± 21.8</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>LVEDP (mmHg)</td>
<td>17.0 ± 9.0</td>
<td>15.7 ± 5.7</td>
<td>NS</td>
</tr>
<tr>
<td>PCWP (mmHg)</td>
<td>10.6 ± 8.3</td>
<td>8.5 ± 5.3</td>
<td>NS</td>
</tr>
<tr>
<td>mAoP (mmHg)</td>
<td>96.0 ± 8.5</td>
<td>95.1 ± 9.3</td>
<td>NS</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>76.7 ± 14.9</td>
<td>69.0 ± 10.8</td>
<td>NS</td>
</tr>
</tbody>
</table>

Abbreviations: See footnote to Table I.

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.

Ambulatory electrocardiography

Holter monitoring was done in all patients within a month before or after the signal-averaged electrocardiography. Twenty-four hour ambulatory electrocardiograms 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 Cardiogem) for the presence of ventricular premature contractions. A nonsustained ventricular tachycardia was defined as three or more repetitive ventricular contractions lasting up to 30 s. A sustained ventricular tachycardia was defined as one that required countershock due to loss of consciousness, or that lasted longer than 30 s.

Statistical analysis

Group mean data were expressed as mean ± SD. Statistical difference was examined by the unpaired student t-test or the $\chi^2$-test; $p <$ 0.05 was considered significant.

RESULTS

Late potentials were found in 15 (30%) of the 50 patients with previous myocardial infarction. Fig. 1 shows signal averaged electrocardiograms from a representative patient (Fig. 1).

Sustained ventricular tachycardia was detected in 3 patients. Nonsustained ventricular tachycardia was detected in 5 patients. The mean duration of late potentials in patients without ventricular tachycardia was 12.0 ± 10.0 ms. It was significantly longer in patients with sustained ventricular tachycardia (118.7 ± 18.6 ms) than those with nonsustained ventricular tachycardia (12.8 ± 10.1 ms) or without ventricular

Japanese Circulation Journal Vol. 52, February 1988
tachycardia (12.0 ± 10.0 ms). Of the 50 patients with previous myocardial infarction, 28 patients had left ventricular aneurysm. Late potentials were detected in 14 of the patients (50%) with aneurysm but only in 1 of the patients (5%) without it. Late potentials were detected in 5 (83%) of 6 patients who had left ventricular aneurysm and ventricular tachycardia, and in 9 (41%) of 22 patients with left ventricular aneurysm and without ventricular tachycardia, and the difference was not statistically significant.

In the 50 patients with previous myocardial infarction, hemodynamic data of the late potential positive group (n = 15) were compared to those of the late potential negative group (n = 35) (Table I). The ejection fraction in the late potential positive group was significantly lower than the late potential negative group (p < 0.001). The cardiac index and stroke volume were also lower in the late potential positive group than the late potential negative group (p < 0.05, p < 0.01). Left ventricular end-diastolic volume in the late potential positive group was greater than the late potential negative group (p < 0.05). There were no significant differences between the two groups as regards left ventricular end-diastolic pressure, pulmonary capillary wedge pressure, mean aortic pressure and heart rate.

Among the patients with previous myocardial infarction and left ventricular aneurysm, we compared the hemodynamic data of the late potential positive group (n = 14) with those of the late potential negative group (n = 14) (Table II). Cardiac index and stroke volume were significantly lower in the late potential positive group than the late potential negative group (p < 0.05, p < 0.05). Ejection fraction tended to be lower in the late potential positive group compared with the late potential negative group. There were no significant differences between the two groups as regards left ventricular end-diastolic volume and pressure, pulmonary capillary wedge pressure, mean aortic pressure, and heart rate.

DISCUSSION

The fractionated electrograms have been correlated with the body surface late potentials. Additionally, fractionated electrograms were recorded in regions in which infarct healing had caused wide separation of individual myocardial fibers while distorting their orientation. Therefore, the presence of late potentials is considered to indicate the existence of damaged ventricular myocardium. Several studies of the relationship between late potentials and left ventricular function have been performed. In these previous studies, however, the only hemodynamic variable which has been considered is ejection fraction.

Rozanski et al suggested that the presence of late potentials was specific to patients with left ventricular aneurysm and chronic recurrent ventricular tachycardia. Breithardt et al reported that the prevalence of late potentials was higher in patients with left ventricular akinesia or aneurysm than in patients without it. Abbound et al concluded that the late potentials could be detected in patients with left ventricular aneurysm or right ventricular dysplasia, regardless of whether recurrent ventricular tachycardia was present. However, in patients with left ventricular aneurysm but without ventricular tachycardia, the prevalence of late potentials was found to be only in 1 of 12 in a study by Rozanski et al and 3 of 27 in a study by Breithardt et al. Conversely, Pollak et al reported that the late potentials were independent of ejection fraction, wall motion score, or presence of dyskinesia but associated with ventricular tachycardia. These previous studies were not in agreement.

In this study, the late potential positive group had a significantly lower ejection fraction, stroke volume, and cardiac index than the late potential negative group (Table I). There were no significant differences between the two groups as regards left ventricular end-diastolic, pulmonary capillary wedge, and mean aortic pressures which reflect the preload or afterload. These data demonstrated that the patients with late potentials had lower left ventricular contractility than the patients without. It became clear that in patients with prior myocardial infarction, those with late potentials had more severe left ventricular dysfunction than those without. It is possible that the difference in the left ventricular function between the two groups is due to the presence of left ventricular aneurysm. So we further studied the 28 patients with left ventricular aneurysm in a similar way. There was also a significant difference in hemodynamic data between the late potential positive group (n = 14) and the late potential negative group (n = 14) (Table II); the stroke volume and cardiac index were significantly lower in the late potential.

*Japanese Circulation Journal Vol. 52, February 1988*
positive group. The ejection fraction tended to be lower in the late potential positive group. Even in patients with previous myocardial infarction and left ventricular aneurysm, we found that the late potential positive group had more severe left ventricular dysfunction.

The prevalence of late potentials was significantly higher in patients with left ventricular aneurysm than those without. Among the patients with left ventricular aneurysm, there was no statistical difference in the prevalence of late potentials between the patients with ventricular tachycardia and those without. These results indicate that the late potentials had a greater prevalence in patients with ventricular aneurysm, irrespective of ventricular tachycardia. The three patients with sustained ventricular tachycardia had left ventricular aneurysm and longer duration of late potentials in comparison with the others, so late potentials of long duration may have a close relation to sustained ventricular tachycardia.

In our study, it became clear that the late potential positive group had more severe left ventricular dysfunction than the negative group. Left ventricular function and left ventricular aneurysm are among the factors that influence the prevalence of late potentials.

REFERENCES

20. AUSTEN WG, EDWARDS JE, FRYE RL, GENSINI GG, GOTT VL, GRIFFITH LSC, McGOON DC,

Japanese Circulation Journal Vol. 52, February 1988