BODY SURFACE POTENTIAL MAPPING IN ANTERIOR MYOCARDIAL INFARCTION

—A Longitudinal Study in Acute, Convalescent and Chronic Phases—

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Body surface potential mapping (BSPM) was performed to evaluate the infarct size and the viability of myocardium in the infarct area in 20 patients with anterior myocardial infarction (MI). BSPM was performed at the early acute phase, 1 week, 1 month and 2 months after onset of the symptoms. The departure areas were obtained according to the potential distribution below the mean normal range and were compared with the value for creatine phosphokinase (CPK), hemodynamic parameters, ejection fraction measured by radionuclide ventriculography, extent score (ES) and severity score (SS) of thallium-201 single photon emission computed tomogram. Two months after the infarction, the ergometer exercise was performed and departure areas before and after exercise were compared. With the departure map technique, the departure areas in all cases were found in the anterior region of the thorax; From 1 week to 2 months after MI, the departure areas were significantly reduced. One week after MI, the departure areas had a positive significant relation with peak CPK and ΣCPK. One month after MI the departure areas also had a positive relation with ES or SS. One week and 1 month after MI, the departure areas had a negative relation with the left ventricular stroke work index or the left ventricular ejection fraction. After exercise test in the chronic phase, the departure areas were significantly enlarged. In conclusion, the departure map is useful in evaluating the location, sequential changes of size of anterior MI including the ischemic area around the infarct site and the left ventricular function. It is suggested that the enlarged departure areas after exercise might be the ischemic areas provoked by exercise.

BODY surface potential mapping (BSPM) is useful for the determination of the location of myocardial infarction\textsuperscript{1}–\textsuperscript{12} but there have been only a few reports about BSPM in evaluating the infarct size and viability of the myocardium in the infarct area. We performed a longitudinal study in acute, convalescent and chronic phases of myocardial infarction in order to evaluate the infarct size and the viability of the myocardium in the infarct area. We used the departure map technique which detects the potential distribution out of the normal range, and then compared

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our findings with those of the thallium-201 single photon emission computed tomogram (TI-201 SPECT), technetium-99m multiple ECG-gated ventriculogram and hemodynamic parameters in patients with anterior myocardial infarction.

MATERIALS AND METHODS

Patients
The study involved 20 patients with acute anterior myocardial infarction, 18 men and 2 women, ages 31–76 years (mean 57.4), who fulfilled the following criteria:

a. Typical chest pain and changes in serum enzyme creatine phosphokinase level (CPK) 2 times above the normal range.

b. The standard 12 lead ECG showed the characteristic ST elevation and developed abnormal Q at least in lead V1, V2, V3.

c. TI-201 SPECT showed a decreased uptake in the anterior wall.

d. No conduction disturbance was found (right bundle branch block, left bundle branch block, hemiblock, Wolff-Parkinson-White syndrome etc.).

Normal control
Eleven normal volunteers, 10 men and 1 woman (age 30–73, mean 38) were examined and BSPM were recorded. None of the volunteers had a history of cardiac disorder or systemic arterial hypertension. All of them had normal physical and standard 12 lead electrocardiographic findings.

Body surface potential map
Body surface potential mapping was performed using a body surface potential mapping system, HPM-6500 unit (Fukuda Denshi Company), recorded simultaneously at 87 lead points as described previously by Yamada et al. In myocardial infarction there is a loss of potential in the infarct site. Thus, a departure map was obtained according to the potential distribution below the mean normal range which is made by averaging the potential of normal controls in each lead point on body surface. Departure index was calculated by the following formula:

\[ DI = \frac{QRS_i - \text{mean normal } QRS_i}{\text{S.D. of normal } QRS_i} \]

\[ DI = \text{departure index} \]

Fig.1. The study protocol of the ergometer exercise test. Body surface potential mapping (BSPM) was performed before and immediately after exercise. Radionuclide ventriculography was performed before and during stress, thallium-201 single photon emission computed tomogram (TI-201 SPECT) was performed immediately after and 3 hours after exercise.

\[ i = \text{instantaneous time} \]

\[ SD = \text{standard deviation} \]

Because potential was small in the back and lower part of the chest, the difference of potential in each lead point was divided by the standard deviation in order to get better values.

\[ \text{Departure area} = \text{area of departure index} \leq -2 \]

Departure maps were made every 4 milliseconds from the onset of QRS complex, and the largest of the departure areas was considered the infarct area.

The distance between 2 leads was assumed to be 5 cm and the area was measured by computer system (Lulex 500).

Study protocol
Body surface potential mapping was performed 2–4 days (early acute phase), 1 week, and 1 month after the onset of myocardial infarction.

Bicycle ergometer exercise testing was performed 2 months after the onset of myocardial infarction, with a workload starting at 25 watts and increased by 25 watts every 3 min up to 75 watts or until the patient was unable to undertake further exercise due to exhaustion. Body surface potential maps were recorded before and immediately after the exercise (Fig. 1).

Serum creatine phosphokinase
The serum creatine phosphokinase (CPK) level was measured immediately after the admission of
Fig. 2. The departure area in anterior myocardial infarction was found in the anterior region of the thorax. The departure area in this case was measured at 32 msec after the QRS onset.

the patient, and every 3 hours until it returned to the normal range. The highest CPK level was defined as peak CPK, and total CPK released was defined as \( \Sigma \text{CPK} \), and was calculated by the following formula.

\[
\int_0^1 f(t) \, dt = \int_0^1 (dE/\,dt - KdE) \, dt \quad \ldots (13)
\]

\[
\int_0^1 f(t) \, dt = \Sigma \text{CPK} \quad \text{Total amount of CPK released.}
\]

\( f(t) = \) CPK appearance function.

\( E = \) Serum CPK activity at any given instant.

\( Kd = \) Fractional CPK disappearance rate from serum.

\( t = \) Time after onset of myocardial infarction.

**Thallium-201 SPECT**

Thallium-201 SPECT was performed by Gammascinticamera (ZLC 7500 Siemens) and was reconstructed by computer (Scintipac 2400 Shimazu) after an intravenous injection of 2–3 mCi thallium-201. Images were obtained from 3 different views: short axis, long axis, apical 4 chambers. The size of the defects and low uptake area were calculated as extent score (ES) and severity score (SS) by the Bull’s eye method,\(^{14,15}\) as shown in the following formulas.

\[
\text{ES} = \frac{\text{number of points in infarcted myocardial area}}{\text{total number of points in myocardial area}}
\]

\[
\text{SS} = \frac{\text{sum of the difference between counts of infarcted myocardial area and noninfarcted area}}{\text{total number of points in myocardial area}}
\]

Fig. 3. Sequential changes in departure areas from early acute phase to 2 months after myocardial infarction. The departure areas were significantly reduced from 1 week to 2 months after myocardial infarction.

A TI-201 SPECT was performed within 1 week and 1 month after the onset of myocardial infarction.

**Technetium-99m multiple ECG gated ventriculography**

Twenty mCi technetium-99m was injected intravenously and the difference in radioisotope counts between end-diastolic phase and end-systolic phase in the left ventricular area was calculated as the left ventricular ejection fraction (LVEF). Radionuclide ventriculogram was performed within 1 week and 1 month after the onset of infarction.

**Hemodynamic data**

Hemodynamic data were obtained from right heart catheterization using a Swan-Ganz catheter in the early acute phase and 1 month after the onset of myocardial infarction.

**Statistical analysis**

Statistical analysis was made using paired or unpaired Student’s t test, and p values less than 0.05 were considered to be significant.

**RESULTS**

**Site of departure area**

The departure areas in all cases were found in the anterior region of the thorax, compatible with the findings of the TI-201 SPECT (Fig. 2). The departure areas appeared from 4 msec–68 msec after the onset of QRS complex. The largest areas which were defined as infarct areas were measured from 30 msec–60 msec after the onset of QRS complex.

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Serial Departure Maps in Myocardial Infarction

Fig. 4. The departure areas after exercise in the chronic phase were significantly enlarged as compared to those before the exercise.

Fig. 5. The departure areas in the early acute phase had a significant positive relation with peak CPK or ΣCPK measured in the acute phase.

Fig. 6. The departure areas calculated from the maps 1 week and 1 month after infarction had a significant negative relation with the left ventricular stroke work index (LVSWI) measured in each phase.

Fig. 7. The departure areas calculated from the maps 1 week and 1 month after infarction, had a significant negative relation with the left ventricular ejection fraction (LVEF) measured in each phase.

Serial changes in departure areas

The departure areas tended to increase from the early acute phase to 1 week, and were then significantly reduced from 1 week to 2 months after the onset of infarction (p < 0.05) (Fig. 3).

Changes in departure area by exercise in chronic phase

The departure areas after exercise (2 months after the onset of myocardial infarction) were significantly enlarged as compared with those before the exercise (p < 0.05) (Fig. 4).

Relation between departure areas with other clinical findings

The departure areas calculated from the map 1 week after the onset of myocardial infarction had a significant relation with peak CPK and ΣCPK measured in the acute phase (peak CPK: \( r = 0.570, p < 0.05 \); ΣCPK: \( r = 0.573, p < 0.05 \)) (Fig. 5).

The departure areas calculated from the map 1 week and 1 month after the onset of infarction had a significant negative relation with left ventricular stroke work index (LVSWI) measured in each phase (1 week after: \( r = -0.572, p < 0.05 \); 1 month after: \( r = -0.607, p < 0.05 \)) (Fig. 6).

The departure areas 1 week and 1 month after the onset of infarction had a significant negative relation with LVEF in each phase (1 week after: \( r = -0.474, p < 0.05 \); 1 month after: \( r = -0.722, p < 0.001 \)) (Fig. 7).

The departure areas 1 month after the onset of infarction had a positive relation with extent score and severity score in the same phase (ES: \( r = 0.691, p < 0.01 \); SS: \( r = 0.658, p < 0.01 \)) (Fig. 8).

Patients with anterior infarction whose TI-201 SPECT showed the defect areas only in the anterior region had a significantly shorter duration which the departure areas can be observed.
Fig. 8. The departure areas calculated from the maps 1 month after infarction had a significant positive relation with the extent score (ES) at the same phase, but at the early acute phase, there was no significant relation between departure areas with ES (A). The same findings were also observed between departure areas with the severity score (SS) (B).

Fig. 9. The duration which the departure areas can be observed in patients with anterior and inferior wall defect (A), was significantly longer than that in patients with only anterior wall defect (Al).

(1 week: 34.4 ± 23.77 msec, 1 month: 36 ± 23.32 msec), than patients whose defect areas were in the anterior and inferior regions (1 week: 54.86 ± 8.86 msec, 1 month: 54.86 ± 11.48 msec) (p < 0.005, p < 0.01) (Fig. 9).

DISCUSSION

Body surface potential map is undoubtedly useful in predicting the location of myocardial infarction. In our study, all of the twenty cases with anterior myocardial infarction had departure areas located in the anterior region of the thorax. The infarct areas in departure maps were measured from 30 msec to 60 msec after onset of the QRS complex. Nakano et al. reported that the infarct areas in departure maps in anterior myocardial infarction were measured about 20 msec after onset of the QRS complex.

Durrer et al. reported that 5 msec after impulse stimulation in the isolated human heart, left ventricular endocardium in the anteroseptal region was excited, and the excitation was completed in about 40 msec in the anteroseptal region, 50 msec in the inferior region, and the lateral region was somewhat later. Our findings coincided with, but were somewhat later than the findings of Durrer et al. This delay might be a conduction delay due to the myocardial infarction. The duration which the departure areas can be observed in patients with anterior infarction were significantly shorter than those in patients with anterior infarction which extended to the inferior region (Fig. 9). These findings suggest that the infarct size could be determined not only by the extent of the departure area, but also by the duration which the departure areas can be observed.

The value of CPK in myocardial infarction had a good relationship with the extent of the infarction. In our study, there was a good relation between departure areas measured at the acute phase with peak CPK and ΣCPK. The left ventricular function was postulated to reflect the extent of infarct size in myocardial infarction. In this study the left ventricular function evaluated by LVSWI and LVEF had a significant negative relation with the departure areas, so that the infarct size could be reflected by the extent and the duration which the departure areas can be observed. Patients with departure area above 300 cm² tended to have LVEF below 40%.

Recently, TI-201 SPECT has been considered to be the most useful method of detecting the location and size of the myocardial infarct.
area.\textsuperscript{14,15,20} In this study there was a good positive relation between the departure area and the defect area measured by Ti-201 SPECT. A patient with a large departure area had a large perfusion defect area, as also measured by the Bull's eye method. It is suggested that the extent of the departure area is useful in determining the infarct size. Moreover, BSPM can be performed repeatedly because it is very simple, noninvasive, and low in cost.

All of the cases were compared to 11 normal controls, 10 men and 1 woman, aged from 30–73 years old with mean of 38 years old. There was a variation in their body surface potential, even though they had no any history of cardiac disorder or systemic arterial hypertension. In this study the normal controls are appropriate and can be used for the evaluation of myocardial infarction because there were significant relation between the departure area and Ti-201 SPECT, radionuclide ventriculogram.

In this study, 2 cases were excluded from the statistical analysis, because of the extensive anterior infarction which extended to the inferior wall, and showed not only the anterior infarction type, but also the inferior infarction type in the departure map. Both of the infarction types have very different characteristics in the formation of the departure area. The infarct site which is presented by the loss of the electrical forces, in the anterior infarction type, was directly projected to the anterior region of the thorax, which is very close to the anterior myocardial wall, making the departure area extent little different from the infarct site. In the inferior infarction type, the loss of the electrical forces of the inferior myocardial wall was spread to the lower portion of the whole thorax, causing the extent of the departure areas to be much greater than the infarct site. In these 2 patients, we were unable to choose either the anterior infarction type or the inferior infarction type for statistical analysis.

From the early acute phase to 1 week after infarction, the departure area seemed to increase; it then decreased as seen at 1 month and 2 months after infarction. The same findings were also presented by another report\textsuperscript{16} The departure areas significantly decreased from 1 week to 2 months. Two months after infarction, the departure areas were smaller than those in the early acute phase, but the difference was not significant. Fishbein et al\textsuperscript{21} reported that necrosis and acute inflammation predominate during the first week, chronic inflammation reaches its peak during the second week, and then proliferation of connective tissue dominates from the third week until healing is complete. The time required for complete healing of myocardial infarction varies considerably from about 36 to 90 days after onset.

It is very difficult to evaluate the viability of myocardium in the infarct site, as no effective method is known. In acute myocardial infarction, the myocardial tissue in the infarct site consists of necrotic and profoundly ischemic tissues with electrical inertia and quiescence. This phenomenon has been called "electrical stunning". These ischemic tissues are expected to be viable after the renewed perfusion, which can be observed in body surface, classically as the reversibility of Q wave in the 12 lead ECG. The serial changes of departure areas mentioned above, suggest that some portion of the myocardium in the infarct area might be viable, or that the ischemic area might be decreased because blood supply is reaching the infarct site by collateral circulation or other reasons. But it is very difficult to evaluate this viability using only one instantaneous BSPM. At any rate, it is very important to manage the patients with acute myocardial infarction carefully for 2 months, since the healing process is not complete until this time.

The departure area was significantly enlarged after ergometer exercise as compared with that before exercise in the chronic phase. The difference is considered to be due to an ischemic area that was provoked by exercise. This finding also indicates that the departure area consists of both necrotic and ischemic myocardium. Of the 20 cases studied, 4 had reduced departure areas, and 1 had no change in departure area after the ergometer exercise test. There were no clinical differences between those cases, such as presence of collateral vessels, exercise workload and other clinical parameters.

The finding of the enlarged departure area is similar to the finding by Ti-201 SPECT; again, the defect area increased after exercise stress test, as compared to the resting stage. Exercise increased the ischemic area around the infarct site which was presented as an increase in the departure area on the departure map. Other mechanisms like Brody-effect, wall motion abnormality, and increased lung conductivity due to pulmonary edema had also to be considered. Further evaluation is necessary in order to
CONCLUSION

Body surface potential mapping is a useful way of determining the location and sequential changes in size of the infarct. Further improvement in the departure map technique are necessary in order to evaluate the viability of the myocardium in the infarct area.

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