The Reappraisal of ST Segment Elevation as an Index of Infarct Size in Patients with Acute Anterior Myocardial Infarction

MICHITOSHI INOUE, M.D. AND MASATAKA FUKUNAMI, M.D.

The clinical meaning of precordial ST segment mapping for assessment of infarct size was studied in 12 patients with acute anterior myocardial infarction. ΣST_{39} (the sum of ST segment elevations in 39 precordial leads), ΣST_{V1-6} (the sum of ST segment elevations in the standard chest leads) and ST_{MAX} (the maximum of ST segment elevation in 39 precordial leads) were obtained serially after the onset from precordial electrocardiographic mapping records. The infarct size and the time (Tf) when the evolution of myocardial infarction finished were calculated by the total creatine phosphokinase released (ΣCPK) and serial changes in CPK releasing rate, respectively. ΣST_{V1-6} at Tf, as closely as ΣST_{39} ($r = 0.82$, $p < 0.01$), correlated with ΣCPK ($r = 0.79$, $p < 0.01$). Even ST_{MAX} at the time of Tf significantly correlated with ΣCPK ($r = 0.60$, $p < 0.05$), although the correlation coefficient was not so well as the former two.

These results showed that ΣST_{V1-6} reflected infarct size as satisfactorily as ΣST_{39} and could be useful for measuring infarct size.

MUCH recent research¹⁻⁶ has been focussed on clinical estimation of myocardial infarct size, because it is one of the most important determinants of the prognosis of patients with myocardial infarction.^{5,7,8} Precordial ST segment mapping technique has been introduced by Maroko, PR et al² to assess the extent and severity of the ischemic injury. However, there is controversy as to whether it is possible to estimate infarct size by precordial ST segment mapping.^{9,10} This controversy results from the fact that ST segment deviation may be influenced by many factors such as the location of infarct or the stage of infarct progression. In the

previous paper,¹ therefore, we emphasized that not only infarct site but also the time after the onset must be taken into account for assessing infarct size by precordial ST segment mapping.

On the other hand, it is time-consuming and laborious to record a precordial ST segment map in a patient suffering from acute myocardial infarction. Therefore, it is desired to simplify the precordial mapping lead system as much as possible.

In this study, we attempted to elucidate the effect of the number of precordial leads on reliability to estimate infarct size by employing three indices of ST segment elevation derived from 39, six and one precordial lead system.

Key Words:

Acute myocardial infarction
Precordial ST segment mapping
Infarct size
Creatine phosphokinase
Solid angle theory

MATERIALS AND METHODS

Twelve patients who had sustained an acute anterior transmural myocardial infarction within

First Dept. of Med. Osaka Univ. Medical School

Address for reprints: Michitoshi Inoue, M.D., The First Department of Medicine, Osaka University Medical School, 1-50 Fukushima 1, Fukushima-ku, Osaka 553, Japan

Fig.1. The positions of 39 precordial leads. Twenty-one points on the anterior chest were at the intersections of three imaginary vertical lines (right, left sternal border and left mid-clavicular line) and seven horizontal lines (second to sixth intercostal space near the sternum and 2.5, 5 cm below the fifth point). Eighteen points on the left lateral chest were at the intersections of three vertical lines (anterior, middle and posterior axillary line) and six horizontal lines (the same levels as second to seventh points on the mid-clavicular line).

the previous 12 hours were studied. All had a typical history of prolonged chest pain and characteristic electrocardiographical changes indicative of infarction including Q wave appearance or ST-T wave alterations in any of the standard chest leads and diagnostic rise and fall in serum enzyme of creatine phosphokinase (CPK). None of our series had evidence of complications such as pericarditis or cerebrovascular accident which might affect ST segment level. No patients who had a history of previous infarction were included.

1) Precordial ST segment mapping technique

Electrocardiograms were recorded through unipolar lead ('V' lead) from 39 points on the

Fig.2. Relationships between three indices on ST segment elevation (ΣST_{39} in the upper panel, ΣST_{V1-6} in the middle panel and ST_{MAX} in the lower panel) and infarct size estimated enzymatically (ΣCPK). These correlation coefficients were 0.82 ($p < 0.01$), 0.79 ($p < 0.01$) and 0.60 ($p < 0.05$), respectively.

anterior to lateral chest wall (Fig. 1), using a thermal pen recorder on standard electrocardiographic paper running at 25 mm/sec with a standardization of 10 mm for 1 mV on admission, 12, 24,

Fig.3. Time courses of mean values of ΣST_{39} (upper panel), ΣST_{V1-6} (middle panel) and ST_{MAX} (lower panel) after the onset. The number in the parenthesis (lower panel) indicates the sample number. Each vertical bar shows its standard error.

36, 48, 72 hours, one week and two weeks following the onset of the illness. These points of leads were maintained throughout patients' hospitalization by marking corresponding precordial surface.

The magnitude of ST segment elevation in each lead was measured at the J point. In each recording, the sums of ST segment elevations from all mapping leads (ΣST_{39}) and the standard precordial leads (ΣST_{V1-6}) were calculated. And the maximal ST segment elevation (ST_{MAX}) in 39 precordial leads was also obtained.

2) Enzymatical assessment of the evolution process and the infarct size

Venous blood samples for creatine phosphokinase (CPK) determination were obtained at four-hour intervals from the time of admission until serum enzyme level returned to normal. The appearance function of CPK released from the infarcted myocardium was calculated by the method of Sobel, BE et al⁵ as modified by Norris, RM et al⁶ and used as a guide in assessing the time course of evolution of infarct.¹¹ Total CPK released (ΣCPK) was obtained by integrating this appearance function and was used as an index of infarct size. Though the evolution of the infarct was considered to cease at the time

Fig.4. Relationships between ΣST_{39} and ΣST_{V1-6} (left panel) with correlation coefficients of 0.90, 0.84, respectively. These indices were obtained at the same time 3 hours to 2 weeks after the onset.

when the appearance function of CPK released became zero IU/ml per hour, the exact determination of this time was difficult because it could be influenced substantially by subtle alteration in the disappearance rate of serum CPK activity. Therefore, in this study the time when the appearance function decreased to 5 IU/ml per hour was regarded as the end point of evolution (Tf); this time is much less influenced by the alteration of the disappearance rate of serum CPK activity. In practice, the sum of ST segment elevation at the end of evolution were obtained as an index of infarct size by linear interpolation of the two values which were measured at the closest points immediately before and after the time when the appearance function became 5 IU/ml per hour or less.

RESULTS

1) Relationships between ΣST_{39} , ΣST_{V1-6} or ST_{MAX} and infarct size

ΣST_{39} , ΣST_{V1-6} and ST_{MAX} at the end of evolution (Tf; 32.6 ± 3.3 hours) correlated with the total CPK released (ΣCPK) with correlation coefficients of 0.82 ($p < 0.01$), 0.79 ($p < 0.01$) and 0.60 ($p < 0.05$), respectively (Fig. 2). In spite of reduction in the number of the precordial leads, the coefficient for ΣST_{V1-6} was almost equal to that for ΣST_{39} , although a little decrease in the value of the coefficient was observed in the case of ST_{MAX} .

2) Serial changes in ΣST_{39} , ΣST_{V1-6} and ST_{MAX}

It is necessary to examine whether alteration in ΣST_{V1-6} or ST_{MAX} is parallel to that in ΣST_{39} or not, in order to assess infarct size by more reduced information as above cited. Figure 3 shows the time courses of ΣST_{V1-6} and ST_{MAX} in comparison with that of ΣST_{39} . This result indicates that serial changes in these indices were similar to each other. Figure 4 demonstrates the relationships between ΣST_{V1-6} and/or ST_{MAX} and ΣST_{39} at the same time. There were close correlations between ΣST_{V1-6} and/or ST_{MAX} and ΣST_{39} with correlation coefficients of 0.90 and 0.84, respectively, which were highly significant ($p < 0.001$).

DISCUSSION

Renewed attention¹² has been paid in recent years to the accuracy and limitations of precor-

dial ST segment mapping as a means of estimating the size of ischemic injury and ultimate necrosis, in view of necessity to possess a method to evaluate the effect of therapeutic interventions for reducing the resultant infarct size. In the previous investigation, we carried out to evaluate the usefulness and limitations of precordial ST segment mapping as a clinical tool to assess infarct size of anterior wall as well as of inferior wall. The results in that paper might strongly suggest that ΣST (the sum of ST segment elevations in 39 precordial leads) and nST (the number of leads showing ST segment elevation) at the end of evolution of infarction or 48 hours after the onset could be useful indices for assessing infarct size, provided that infarct site would be strictly classified. In this paper, we also dealt with the data of ST segment elevation at the end of evolution when the CPK had ceased to release. And we attempted to evaluate the usefulness and limitations of the standard precordial leads or only one chest lead to estimate infarct size in comparison with 39 precordial lead system.

ΣST_{V1-6} correlated with the infarct size estimated enzymatically (ΣCPK) ($r = 0.79$, $p < 0.01$) as closely as ΣST_{39} . Furthermore, even ST_{MAX} significantly correlated with ΣCPK ($r = 0.60$, $p < 0.05$), although the coefficient was not enough to estimate infarct size. These results suggested that any index of the three concerning ST segment elevation might reflect infarct size although the accuracy for assessment of infarct size by only one chest lead was not considered to be reliable.

Theoretically, these evidences meaning that any lead showing ST segment elevation reflects infarct size can be explained by the solid angle theory¹³ i.e., electrical potential recorded on the chest surface is mainly influenced by solid angle of ischemic zone subtended at a point of electrode. Solid angle (ω) is related to not only the size (S) of ischemic zone but also the site of ischemic zone as following equation.

$$\omega = S \cdot \cos \theta / r^2$$

where θ is the angle of the normal of the surface S and the line binding between the observation point and the center of the surface S, and r is a distance between them. Therefore, the extent of ST segment elevation is also affected by not only the ischemic size but also the ischemic site. Subsequently, the size cannot be determined from the solid angle at one point unless the site was fixed.

However, provided that the location of infarction would be limited to anterior wall as this study, variation of infarct site could be so negligible that ST segment elevation in one lead possibly reflects infarct size as shown in our result ($r = 0.60$). Although increase in the number of relating leads raise the correlation coefficient to the infarct size, there is a limit to reach in improving the value of the coefficient ($r = 0.79$ for six leads, $r = 0.82$ for 39 leads).

In conclusion, although we must take the infarct site and the time after the onset into consideration in order to utilize precordial ST segment mapping technique, the standard chest lead was found to be also of clinical use with limitation to anterior wall infarct. However, estimation of infarct size using by only one chest lead proved to be questionable on the accuracy. In order to use less number of leads for convenience than the number of precordial mapping leads, it is mandatory to search for optimal number and positions of precordial lead corresponding to the infarct site according to the solid angle theory. Therefore, further investigations on such an optimal arrangement of lead system are to be needed theoretically.

REFERENCES

- INOUE M, HORI M, FUKUNAMI M, FUKUSHIMA M, TADA M, ABE H, MINAMINO T, FUKUI S: Evaluation of precordial ST segment mapping as an index of infarct size in patients with acute myocardial infarction. *Br Heart J* 42: 726, 1979
- MAROKO PR, LIBBY P, COVELL JW, SOBEL BE, ROSS JJR, BRAUNWALD E: Precordial S-T segment elevation mapping. An atraumatic method for assessing alterations in the extent of myocardial ischemic injury; the effects of pharmacologic and hemodynamic interventions. *Am J Cardiol* 29: 223, 1972
- SHARPE DN, BOTVINIC EH, SHAMES DM, NORMAN A, CHATTERJEE K, PARMLEY WW: The clinical estimation of acute myocardial infarct size with 99m-Technetium pyrophosphate scintigraphy. *Circulation* 57: 307, 1978
- HENNING H, HARDARSON T, FRANCIS G, O'ROURKE RA, RYAN W, ROSS JJR: Approach to the estimation of myocardial infarct size by analysis of precordial S-T segment and R wave maps. *Am J Cardiol* 41: 1, 1978
- SOBEL BE, BRESNAHAN GH, SHELL WE, YODER RD: Estimation of infarct size in man and its relation to prognosis. *Circulation* 46: 640, 1972
- NORRIS RM, WHITLOCK RML, BARRATT-BOYES C, SMALL CW: Clinical measurement of myocardial infarct size. Modification of a method for the estimation of total creatine phosphokinase release after myocardial infarction. *Circulation* 51: 614, 1975
- PAGE DL, CLAUFIELD JB, KASTOR JA, DESANTIS RW: Myocardial changes associated with cardiogenic shock. *New Eng J Med* 285: 133, 1971
- HORI M, INOUE M, FUKUI S, SHIMAZU T, MISHIMA M, OHGITANI N, MINAMINO T, ABE H: Correlation of ejection fraction and infarct size estimated from the total CK released in patients with acute myocardial infarction. *Br Heart J* 41: 433, 1979
- NORRIS RM, BARRATT-BOYES C, HENG MK, SINGH BN: Failure of ST segment elevation to predict severity of acute myocardial infarction. *Br Heart J* 38: 85, 1976
- THOMPSON PL, KATAVITIS V: Acute myocardial infarction — Evaluation of precordial ST segment mapping. *Br Heart J* 38: 1020, 1976
- INOUE M, HORI M, FUKUI S, ABE H, MINAMINO T, KODAMA K, OHGITANI N: Evaluation of evolution of myocardial infarction by serial determinations of serum creatine kinase activity. *Br Heart J* 39: 485, 1977
- MULLER JE, MAROKO PR, BRAUNWALD E: Precordial electrocardiographic mapping; A technique to assess the efficacy of interventions designed to limit infarct size. *Circulation* 57: 1, 1978
- HOLLAND RP, ARNSDORF MF: Solid angle theory and the electrocardiogram; Physiologic and quantitative interpretations. *Progr Cardiovasc Dis* 19: 431, 1977