New diagnostic evidence on the T wave map indicating involved coronary artery in patients with angina pectoris

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ABSTRACT  To define the clinical significance of T wave map changes in patients with angina at rest, body surface isopotential T distributions were obtained in 48 patients with single-vessel disease (left anterior descending artery, 34; right coronary artery, eight; left circumflex artery, six) documented angiographically and were compared with those in 120 healthy subjects and those in 19 patients with left ventricular overload whose electrocardiograms showed negative T waves accompanied by an increase in R wave amplitude in left precordial leads. The T wave map abnormalities were observed in 24 of 48 patients (50%) with angina and were classified into three types: (1) type I (18 patients, 37.5%) was characterized by a segmental negative potential in the positive area located at the left thorax and the minimum at the peak of T wave positioned in the upper portion of the left anterior chest, (2) type II (three patients, 6.3%) was characterized by a negative potential with a minimum in the inferior thorax and an indentation of negative potential at the lower margin of the positive potential located over the upper thorax, and (3) type III (three patients, 6.3%) was characterized by a negative potential with a minimum at the back through the period of T wave. All patients showing T wave map abnormalities of type I had a significant stenosis of the left anterior descending artery. Likewise, all patients with type II or III had single-vessel disease of the right coronary or left circumflex artery, respectively. All types of T wave map changes observed in patients with angina were different from those in patients with left ventricular overload, whose maps showed the generalized negative potential at the inferior thorax and the left back and the minima clustered at the precordium. In seven patients with lesions of the left anterior descending artery, T wave map abnormalities of type I recovered to normal after successful percutaneous transluminal coronary angioplasty. The behavior of the negative potential and its extrema on the T wave map, which was not available from routine electrocardiography, was indicative of the involved coronary artery and probably of its associated ischemic area in one-half of our patients with angina pectoris.


THE INCREASED information content of body surface depolarization maps, as expressed by the isopotential map,1, 2 Q index,3–5 isoelectrical map6, 7 and departure map,8 compared with standard 12-lead electrocardiograms (ECGs) offers promise that they may be useful in the evaluation of patients with myocardial infarction. With regard to repolarization maps, ST maps may provide more precise information on the extent of the ensuing myocardial infarction9, 10 and the involvement of the right ventricle.6 The variables measured for these purposes, however, should be used with consideration of the normal variations in ST segment potential distribution.11 In addition, the application of ST mapping to the exercise test increases its diagnostic value for myocardial ischemia.12, 13

The flattened or inverted T waves in any lead of the conventional ECG have been used clinically to detect coronary insufficiency.14 On some occasions, however, it is not easy to differentiate the inverted T waves in the precordial leads associated with ischemia from those associated with left ventricular overload. Distinctions between inverted T waves derived from two different situations are of obvious significance. The usefulness of the maps of the T waveform (T wave map) in the assessment of coronary heart disease has not been established.

In this study, T wave maps obtained at rest in patients with angina pectoris were analyzed and their abnor-
malities were correlated with lesions of coronary artery and wall motion abnormalities in the left ventricle as evaluated by coronary angiography (CAG) and left ventriculography (LVG). In addition, T wave maps in patients with left ventricular overload, whose standard ECGs showed inverted T waves accompanied by an increase in R wave amplitude in left precordial leads, were compared with those in patients with angina pectoris.

Materials and methods

Materials. Forty-eight patients with angina pectoris (41 men and seven women, 35 to 73 years old) at Nagoya University Hospital and Okazaki City Hospital participated in this study. All patients fulfilled all of the following criteria. None of them had a history of acute myocardial infarction. Each had typical chest pain on effort and disease that was documented angiographically by at least 75% luminal obstruction in a single coronary artery. Thirty-four patients (71%, LAD group) had single-vessel disease of the left anterior descending artery (LAD), eight (16.5%, RCA group) had single-vessel disease of the right coronary artery (RCA), and six (12.5%, LCX group) had single-vessel disease of the left circumflex artery (LCX). The evaluations by CAG and LVG were according to the reporting system of American Heart Association.\(^{15}\) Percutaneous transluminal coronary angioplasty (PTCA) was performed and was successful in relieving chest pain in seven patients in the LAD group. There were two control groups: (1) 120 healthy subjects, 60 men and 60 women, 18 to 37 years old, and (2) 19 male patients, 19 to 68 years old, with left ventricular overload due to systemic arterial hypertension, valvular heart disease, and hypertrophic cardiomyopathy without a history of chest pain.

Body surface isopotential maps. Eighty-seven-lead body surface isopotential maps were recorded in all patients a few days before and after CAG by the Mapper 6500 (Chunichi Denshi Company). The details of this mapping system have been reported previously.\(^{16}\) Seven patients in the LAD group who underwent successful PTCA of a lesion of the LAD had the second map recorded about 1 to 3 months after PTCA. The T wave maps, as illustrated by isocontour potential, were constructed at 4 msec interval during the period of the T wave and they were displayed by the format illustrated in figure 1. The chest wall was divided into nine columns (A to I) and the back into four columns (J to M). Locations of the midsternal and the right and the left midaxillary lines were shown as E, A, and I, respectively. The four leads in the upper portions of the midaxilla were not obtained by direct measurement of electrocardiographic signals but were constructed by interpolation.

Results

Normal T wave map. All healthy subjects demonstrated similar T potential distributions for the major features of normal repolarization that we observed. The potential distributions are presented for a representative patient.

Figure 1 is a representative T wave map from a 37-year-old man. The early phase of the T wave occurred with the positive potential at the left anterior chest, the maximum located at lead F\(_5\) and the minimum at lead L\(_7\) (232 msec). Toward the peak of the T wave (296 msec), the positive area extended into the lower portion of the thorax and its amplitude was increased. During the downslope of the T wave (360 msec), the amplitude of the positive potential was decreased. The site of the maximum remained stationary in lead F\(_5\) throughout the period of the T wave.

The map pattern of potential distribution showed little variation.

T wave map findings in patients with angina pectoris. Twenty-four of 48 patients (50%) had abnormal findings on the T wave map. The T wave map abnormalities observed in this study were classified into three types. A representative abnormal T wave map of type I (patient 4 in table 1) is shown in figure 2. At the first stage of the T wave (232 msec), the positive area was
located mainly at the left thorax, with the maximum at lead F5. This potential distribution was quite similar to those in normal subjects. At 264 msec, the small island of negative potential, which was never observed in any of the normal subjects, appeared in the precordial positive area. The segmental negative area increased in size toward the peak of the T wave until it fused with the negative potential in the left shoulder area, resulting in the appearance of bay formation by the negative potential in the positive area (296 msec). The outline of the positive area, except for the segmental negative area, was similar to a normal one. The minimum was located at lead G4, which is comparable to lead V4 on the standard 12-lead ECG. Another example (patient 3 in figure 3) was characterized by the segmental negative area, which appeared as an island in the positive area at the left lateral chest throughout the period of the T wave. The segmental negative area gradually increased in size toward the end of T wave, with the minimum stationary at lead H4. Eighteen patients (37.5%) showed T wave map abnormalities of type I and all of them were included in the LAD group. There were some variations in the size of the segmental negative area and the location of minimum (figure 3).

A representative type II abnormality (patient 2 in table 2) is shown in figure 4. During the first half of the T wave (215, 255, and 295 msec in figure 4), the positive potential was located mainly at the right ante-
rior chest, compared with at the left anterior chest in normal subjects, and the negative potential occupied the lower portion of the left anterior chest, with a minimum at the back. Near the peak of the T wave (345 and 395 msec), positive potential shifted leftward to the left upper back and negative potential extended into inferior thorax, yielding the indentation of negative potential at the lower margin of the positive potential located at the upper thorax. The minimum at the peak of the T wave was positioned at lead G₂. Three patients (6.3%) showed T wave map abnormalities of type II and all were included in the RCA group. All of these patients had a segmental negative area characterized by a negative indentation of the lower margin of the positive potential at the inferior torso. There was little variation in map pattern.

A representative type III abnormality (patient 2 in table 3) is shown in figure 5. At the first stage of the T wave (244 msec in figure 5), positive potential was positioned at the anterior chest and negative potential was positioned over the back and the lower part of the right anterior and left lateral chest. The position of the positive potential in this type was intermediate, compared with at the left anterior chest in type I and at the right anterior chest in type II. This pattern of potential distribution, especially the generalized negativity in the back, was stationary until the end of the T wave without leftward shift of the positive potential into the left back as observed in type II. The minimum was located at the lower portion of the left back at the first stage (244 msec) and shifted to lead L₄ in the midback at the peak of T wave. Three patients (6.3%) had T wave map abnormalities of type III and all of them were included in the LCX group. There was little variation in map pattern.

Relationship of type of T wave map abnormality to coronary lesion. The findings of CAG and LVG are sum-

FIGURE 2. An example of T wave map abnormality of type I (patient 4 in table 1) at 232, 264, 280, 296, 312, and 328 msec. At the first stage, the map was of a normal pattern (232 msec). At 264 msec, a small island of negative area appeared in the positive area located at the left thorax (arrow) and thereafter the segmental negative area increased in size and amplitude and divided the positive area into two portions, one over the sternum and the other at the left lateral area and the back (280 and 296 msec). The minimum was positioned at lead G₄. The outline of the positive area, except for the presence of the segmental negative area, was similar to a normal one. The ECG showed a normal QRS complex, but the inversion of T waves in leads V₅-V₆. T wave map and electrocardiographic abnormalities in this patient disappeared after a successful PTCA (see figure 3).

The reference lead is lead II. Increments of isopotential lines on maps are 0.1 mV.
were restored, as shown in figure 3, with a relief of chest pain.

T wave maps in patients with left ventricular overload. A representative T wave map from a 59-year-old man with mitral and aortic regurgitation (type IV) is shown in figure 6. The T wave map showed a generalized negative area in the lower part of the left thorax throughout the period. None of the 19 patients with left ventricular overload had the indentation by the negative potential at the lower rim of the positive area in the upper torso representative of a type II T wave map abnormality. There were minor changes in potential distribution and the locations of the extrema in patients with left ventricular overload.

Location of the minimum at the peak of T wave in normal subjects and patients with T wave map abnormalities. The minima of the normal T wave map at the peak of the T wave were mostly located over the upper part of the chest and the right back, that is, at lead D7 in 65%, lead L2 in 15%, and lead E7 in 13% of all healthy subjects. In patients with type I abnormalities, there were minima clustered from the midsternum to the left midclavicular line beyond the fifth intercostal space. The minima in all patients with type II were located at lead G2, in the lower portion of the left anterior chest. The minima in all patients with type III abnormalities existed at lead L4 in the back. In type IV abnormalities observed in patients with left ventricular overload, the minima were positioned in the precordium (figure 7).

Discussion

The results of this study provided evidence that body surface T potential distribution recorded at rest may permit recognition of the involved coronary artery in 50% of patients with angina pectoris. Improvement of

TABLE 2
Findings of CAG, LVG, and T wave mapping in patients with single-vessel disease of the RCA

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Abbreviations as in table 1.

aNumbers in subheading indicate the segment of the coronary artery or left ventricular wall according to the reporting system of the American Heart Association.
FIGURE 4. An example of T wave map abnormality of type II (patient 2 in table 2). At the first-half of T wave (215, 255, and 295 msec), positive potential was located mainly at the right anterior chest, compared with at the left anterior chest in normal subjects, and negative potential occupied the lower portion of the left anterior chest with a minimum at the back. Around the peak of T wave (345 and 395 msec), positive potential shifted leftward to the left upper back and negative potential extended into the inferior thorax, yielding the indentation at the lower margin of the positive potential (arrow). This local negative area was characteristic in this type of T wave map abnormality and was different from the generalized negativity at the lower half of the anterior chest and the back as observed in left ventricular overload (see figure 6). The minimum at the peak of T wave was positioned at lead G2. The standard ECG shows inverted T waves in leads II, III, and aVF. The reference lead is lead II. Increments of isopotential lines on maps are 0.2 mV.

TABLE 3
Findings of CAG, LVG, and T wave mapping in patients with single-vessel disease of the LCX

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Abbreviations as in table 1.

Numbers in subheading indicate the segment of coronary artery or left ventricular wall according to the reporting system of the American Heart Association.
ischemia after a successful PTCA procedure in patients with lesions of the LAD could also be evaluated by the T wave map. When the patient has negative T waves in precordial leads of a conventional 12-lead ECG, it may be easier to differentiate the inverted T waves due to ischemia from those resulting from left ventricular overload by means of body surface T wave isopotential mapping.

**Clinical significance of T wave map abnormalities.** Body surface isopotential maps have the advantage that they detect localized electrical change such as niche phenomenon suggesting right ventricular breakthrough and fractionated excitation fronts during depolarization phase. This study suggests that body surface isopotential mapping can detect the local electrical change during the repolarization phase derived from the coronary lesion as well.

The normal T wave potential distribution is characterized by a positive potential located at the left hemithorax, which indicates the normal physiologic sequence during repolarization. This pattern of potential distribution varies little from person to person, among different decades, and between the sexes.

Fifty percent of all patients with angina pectoris had abnormal T wave maps, and these were divided into three types. Eighteen of the patients (37.5%) with angina had segmental negative potentials in the positive area at the left thorax and minima located beyond the fifth intercostal space between midsternum and the left midclavicle. All of them had a lesion of the LAD, usually at its proximal or middle portion. In seven in whom there was regression of the obstructive lesion of the LAD due to PTCA the normal T wave potential distribution was restored. This evidence suggests that the presence of the segmental negative area in the positive area at the left anterior chest reflects ischemia of the anterior wall of the left ventricle due to a lesion of the LAD. Three patients (6.3%) had an indentation of the lower margin of the positive potential over the upper thorax accompanied by a minimum at lead G₂ at the peak of T wave. All of them had single-vessel disease of the RCA. Three patients (6.3%) had a negative potential with a minimum at lead L₄ over the back throughout the period of the T wave. All of these had single-vessel disease of the LCX. The T wave map

**FIGURE 5.** An example of T map abnormality of type III (patient 2 in table 3). At the first stage of the T wave (244 msec), positive potential is positioned at the anterior chest and negative potential is positioned at the back and the lower portion of the right anterior and the left lateral chest. The position of the positive potential was intermediate compared with that in the type I (left anterior chest) and in type II (right anterior chest). This pattern of potential distribution was stationary until the end of the T wave, without leftward shift of the positive potential into the left back as observed in type II. The standard ECG showed the inverted T waves in leads II, III, aVF, and V₅₋₆. The reference lead is lead II. Increments of isopotential lines on maps are 0.1 mV.
The coronary artery lesion at the site of a pinpoint ischemia may have resulted from one of three causes: atheromatous plaques, spasm of the coronary artery, or areas of myocardial infarction. Differences in the extent of T wave abnormalities in these patients provide evidence of the contribution of these three factors to the ischemic process. The extent of T wave abnormalities in patients with angina suggests that the extent of the segmental negative area in the positive area may be related to the grade of ischemia in the individual.

The illnesses frequently seen clinically that are associated with negative T waves in precordial leads are those resulting in left ventricular overload, including valvular heart disease, hypertension, and primary myocardial disease. It is occasionally difficult to differentiate the inverted T waves due to ischemia from those due to left ventricular overload. Daoud et al. reported the possible usefulness for differential diagnosis of measurement of the effects of isoproterenol infusion on inverted T waves, but they were unable to clearly distinguish inverted T waves due to ischemia from those associated with left ventricular hypertrophy. We have confirmed that the T wave potential distributions in patients with precordial negative T waves associated with left ventricular hypertrophy do not show the segmental negative potential that is characteristic of T wave map abnormalities of type I and II, which correspond to LAD and RCA lesions, respectively. Hence, the presence and the site of the segmental negative area on the T wave map might be used to differentiate the inverted T wave associated with left ventricular hypertrophy from that due to the involvement of the coronary artery in angina pectoris.

The disappearances of the characteristic T wave map findings associated with lesions of the LAD after successful PTCA suggests that resting T wave mapping may indeed be valuable in indicating important pathophysiologic changes that occur after PTCA.

Electrophysiologic meaning of the segmental negative area. In normal subjects, the positive area occupies the left side of the thorax. With normal T potential distribution there is a longer action potential duration at the endocardium and a shorter one at the epicardial site, and therefore the sequence of repolarization is from the epicardium to the endocardium.

In 37.5% of our patients with angina, there existed a segmental negative potential in the upper portion of the normally positioned positive area at the left thorax during the whole T wave. These T wave map findings are indicative of ischemia at the anterior wall of the left ventricle due to a proximal lesion of the LAD. The presence of a positive area at the left side of the thorax, as observed in normal subjects without a segmental negative area, implies that normal repolarization may be stationary at areas other than the anterior wall of the left ventricle. The presence of a segmental negative potential may suggest that the action potential duration at the anterior wall of the left ventricle is prolonged due...
to ischemia and that the recovery sequence at that site is delayed. Evidence for this is also provided by the report of Mandel et al., 23 who found that the action potential at the chronically ischemic site in dog experiments was prolonged; their theoretical model based on this physiologic finding yielded the inverted T waves that resembled those observed clinically.

The present study did not reveal directly the relationship of the extent of ischemia to the changes in the T wave map at rest. Further studies addressing this question could extend the clinical application of the resting T wave map to the evaluation of the size of the ischemic area and of the effect of several therapeutic interventions, including PTCA, on ischemia diagnosed on electrocardiographic examination.

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