

Results

Exercise variables

Group A

The end point of exercise was leg fatigue in 11 and shortness of breath in 4 patients. Work load was 77 ± 29 W. Heart rate at peak exercise was 120 ± 12 bpm. Systolic blood pressure at peak exercise was 186 ± 24 mmHg.

Group B

The end point of exercise was anginal chest pain in 9, ST depression in 5 and leg fatigue in 3 patients. Work load was 51 ± 19 W ($P < 0.01$ versus group A). Heart rate at peak exercise was 108 ± 17 bpm ($P < 0.01$ versus group A). Systolic blood pressure at peak exercise was 171 ± 19 mmHg.

Thallium-201 myocardial imaging and postexercise 12-lead ECG

Group A

No patients had thallium defect and significant ST change.

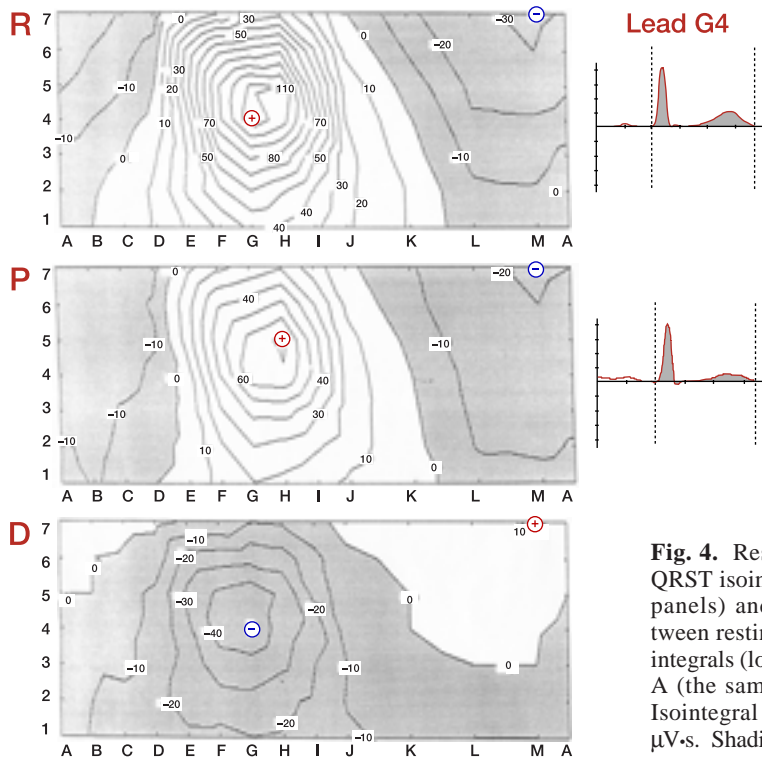
Group B

All patients showed reversible thallium defect. Four patients showed reversible thallium defect in the anterior wall, 7 patients in the posterior wall and 6 patients in both anterior and posterior walls. All patients had significant down-sloping ST depression. Maximal ST depression was -0.21 ± 0.08 mV.

ST-T isointegral maps

Group A

Figure 2 shows resting and postexercise ST-T isointegral maps of a representative patient (60-year-old man). All patients had smooth



dipolar pattern maps both at rest and after exercise with the positive area located over the precordium and the negative area over the right chest and back. This was considered a normal response.

Fig. 4. Resting (**R**) and postexercise (**P**) QRST isointegral maps (upper and middle panels) and the difference (**D**) map between resting and postexercise QRST isointegrals (lower panel) in a patient in group A (the same patient as shown in Fig. 2). Isointegral contours are separated by $10 \mu\text{V}\cdot\text{s}$. Shading indicates negative areas.

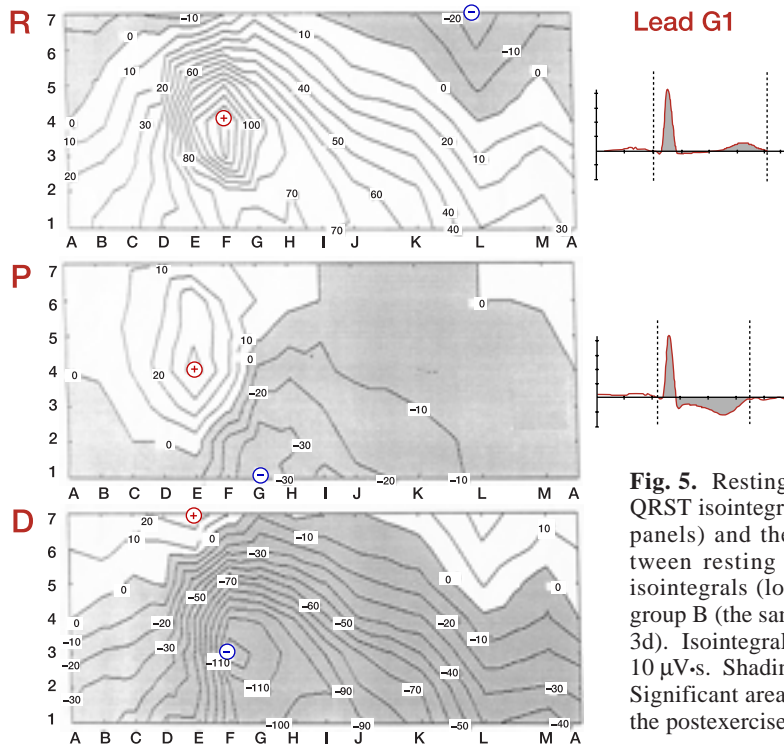


Fig. 5. Resting (**R**) and postexercise (**P**) QRST isointegral maps (upper and middle panels) and the difference (**D**) map between resting and postexercise QRST isointegrals (lower panel) in a patient in group B (the same patient as shown in Fig. 3d). Isointegral contours are separated by $10 \mu\text{V}\cdot\text{s}$. Shading indicates negative areas. Significant area ($< -10 \mu\text{V}\cdot\text{s}$) is present in the postexercise map.

Group B

All patients had significant area in the postexercise map. Seven patients (41%) had significant area in the left-inferior and left-mid regions in the postexercise map, 2 patients (11%) in the left-mid and left-superior regions, 4 patients (24%) in all three of the left regions and 4 patients (24%) in the right-inferior, (right-mid,) left-inferior and left-mid regions. Four types of significant area in the postexercise map are shown in Fig. 3. Significant area in the postexercise map was not correlated with the ischemic area determined by thallium imaging (Table 2).

QRST isointegral maps

Figure 4 shows resting and postexercise QRST isointegral maps and the difference map between resting and postexercise QRST isointegrals in a patient in group A (the same patient as Fig. 2). All three maps show smooth dipolar patterns. Figure 5 shows resting and postexercise QRST isointegral maps and the QRST iso-

integral difference map of a representative patient in group B (the same patient as Fig. 3d). Significant area is observed in the postexercise map. Figures 6a and b show mean and mean -2SD difference maps between resting and postexercise QRST isointegrals in group A. Figure 6c shows the QRST isointegral difference map of a patient in group B (the same patient as Figs. 3d and 5), in which an extensive “ -2SD area” is observed. Figure 7 shows the relationship between resting and postexercise QRST isointegral maps in a patient (group A) with a high correlation coefficient and in a patient (group B) with a low correlation coefficient.

Group A

All patients showed smooth dipolar pattern maps both at rest and after exercise. The positive area covered the precordium with the maximum located in the middle of the left chest and the minimum at the upper region of the right chest or right back. All patients showed a smooth dipolar difference map between resting and postexercise QRST isointegrals. The nega-

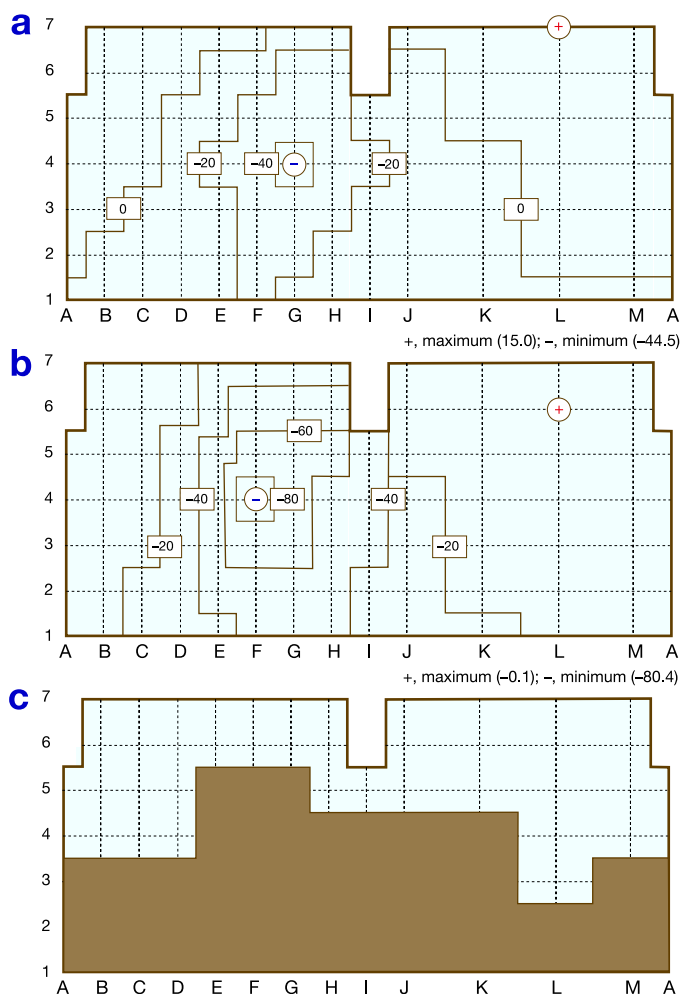


Fig. 6. Difference maps between resting and postexercise QRST isointegrals: Mean map in group A (a), mean -2SD map in group A (b) and a difference map of a patient in group B (the same patient as shown in Figs. 3d and 5) (c). Isointegral contours are separated by 20 $\mu\text{V}\cdot\text{s}$. Shading indicates “-2SD area (less than mean -2SD values in group A)”.

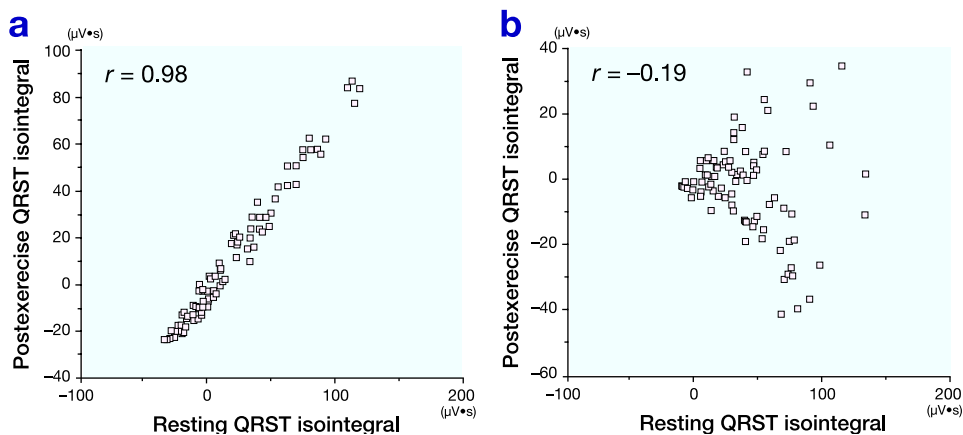


Fig. 7. Relationship between resting and postexercise QRST isointegrals: a patient (group A, the same patient as in Figs. 2 and 4) with a high correlation coefficient (a) and a patient (group B, the same patient as shown in Figs. 3d and 5) with a low correlation coefficient (b).

tive area covered most of the chest with the minimum located in the middle of the left chest and the maximum at the upper region of the right chest or right back. The minimum in mean and mean $-2SD$ difference maps between resting and postexercise QRST isointegrals was located in lead F4 (mean = $-44.5 \mu\text{V}\cdot\text{s}$; mean $-2SD = -80.4 \mu\text{V}\cdot\text{s}$). The correlation coefficient between resting and postexercise QRST isointegrals in 87 lead points was 0.91 ± 0.06 .

Group B

Ten patients (59%) had significant area in the postexercise map. No patients had a non-dipolar pattern map. Fifteen patients (88%) had “ $-2SD$ area” in the difference map between resting and postexercise QRST isointegrals. The correlation coefficient between resting and postexercise QRST isointegrals in 87 lead points in group B (0.28 ± 0.56) was significantly lower than that in group A ($P < 0.001$) (Fig. 8).

Discussion

The present study demonstrates that patients with ischemic ST depression had a greater decrease in the QRST isointegral values in the precordial region than patients without ischemia and ST depression and had low similarities between the resting and postexercise QRST isointegral maps. These findings indicate that ischemic ST depression is related to the dispersion of the exercise-induced changes in repolarization properties.

Body surface ECG mapping offers information about potential distributions around the entire thorax. In patients with coronary artery narrowing, exercise-induced ST depression most often occurs in the left anterior chest leads. According to the data from exercise-stress ST isopotential mapping, predicting the location of ischemic areas is thought to be difficult from the body surface distributions of ST depression (Kubota et al., 1989). Montague and colleagues (1990) studied exercise-stress ST isointegral maps in coronary artery disease and observed that, with exercise-induced angina,

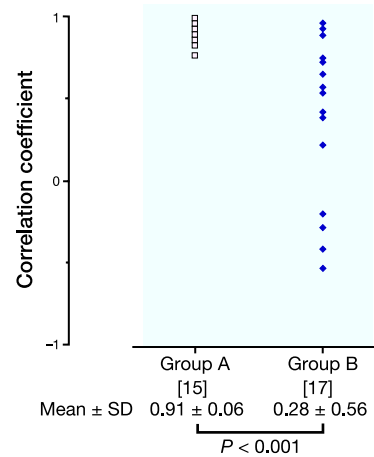


Fig. 8. The correlation coefficient between resting and postexercise QRST isointegral maps for each group, A and B. [], number of patients.

patients with two or three vessel disease had a significantly greater decrease in the ST isointegral values than patients with single vessel disease. There was, however, considerable overlap among individuals. The reason for a lack of correlation between body surface distribution of ST depression and anatomic site of coronary artery disease is unclear. Exercise-induced ST depression is commonly accompanied by a decrease in the height or inversion of the T wave. Nakajima and coworkers (1988) studied exercise-stress ST-T isointegral maps in patients with angina pectoris in the absence of previous myocardial infarction. They observed that postexercise ST-T isointegral maps were divided into 4 types (anterior chest, infero-posterior chest, lateral chest and global) according to the distributions of negative area, which were well correlated with the extent of ischemic area determined by thallium imaging. However, our results demonstrated that significant area in the postexercise ST-T isointegral map was not correlated with the ischemic area. The reason for the discrepancy is not clear. Studies in a large number of patients are needed.

Exercise-induced ST depression is attributed to a current of injury with ischemia in sub-endocardial layers. T wave inversion is thought to be related to the presence of delayed recovery. Exercise-induced ischemia causes not only

ST-T but also QRS changes. R wave amplitude on the surface ECG is decreased in normal subjects while the increase is seen in patients with coronary artery disease (Bonoris et al., 1978a, 1978b). Ikeda and colleagues (1988) reported that intraventricular conduction delay secondary to ischemia plays an important role in the increase in R wave amplitude. ST-T isointegral map reflects ventricular recovery sequence. The recovery sequence is affected by activation sequence and regional recovery properties. The QRST isointegral map has been reported to be useful in investigating recovery properties. Wilson and researchers (1934) reported that the QRST isointegral is independent of the activation sequence and dependent on repolarization properties. They proposed the concept of a ventricular gradient. Based on this concept of the ventricular gradient, Abildskov and workers (1980) introduced the QRST isointegral map. They demonstrated that the QRST isointegral map was independent of the activation sequence and useful in detecting abnormalities in repolarization properties, even in the presence of QRS deflection abnormalities. Although body surface QRST isointegral mapping has been used to assess repolarization abnormalities in a variety of diseases, few reports on exercise-stress QRST isointegral mapping are available. Ohyama and others (1984) studied exercise-induced changes in QRST isointegral map patterns in patients with effort angina in the absence of akinesis or dyskinesis in the left ventricular wall motion. These investigators observed that after exercise the maximum of the map moves far from the resting position and splits into multiple extremes in patients with multivessel coronary artery disease.

In this study, to assess exercise-induced changes in repolarization properties, we performed quantitative analysis of QRST isointegral mapping. Normal responses of QRST isointegral values to exercise have not been determined. We constructed a mean $-2SD$ difference map between resting and postexercise QRST isointegrals in control subjects (group A). When the QRST isointegral value in at least 3 lead points was less than the mean $-2SD$, the decrease was considered significant (" $-2SD$

area"). The correlation coefficient between QRST isointegral maps is independent of the difference in potential magnitudes and indicates the similarities in potential distributions (Hayashi et al., 1989). However, the relationship between resting and postexercise maps has not been analyzed.

Control subjects (group A) showed a decrease in the maximum QRST isointegral value, which may result from shortening of QT interval and a decrease in the R wave and T wave amplitudes in the left precordial leads. These patients showed smooth dipolar ST-T isointegral maps both at rest and after exercise. They also showed smooth dipolar QRST isointegral maps both at rest and after exercise and a high correlation coefficient between the two maps. These findings indicate that control subjects have higher similarities between resting and postexercise repolarization properties.

Patients with ischemic ST depression in the absence of previous myocardial infarction (group B) showed a high incidence of the occurrence of a significant area in the postexercise QRST isointegral map and " $-2SD$ area" in the QRST isointegral difference map. In addition, these patients showed a low correlation coefficient between resting and postexercise QRST isointegral maps. These findings indicate that ischemic ST depression is associated with remarkable changes in ST-T and QRST isointegrals and may be related to the regional abnormalities in repolarization properties.

We conclude that isointegral analysis of body surface ECG mapping has advantages in assessing repolarization properties in the exercise test for the detection of coronary artery disease. Further studies in patients with previous myocardial infarction, intraventricular conduction disturbance or left ventricular hypertrophy are needed.

Acknowledgments: We thank Drs. Takahiro Nawata and Tetsuya Doi and all the members in the First Dept. of Internal Medicine, Faculty of Medicine, Tottori University for their cooperation. We also thank Messrs. Kenichi Endo and Tsuyoshi Nishio, Dept. of Clinical Radiology, Tottori University Hospital for their assistance.

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(Received April 21, Accepted May 26, 1997)