Body Surface Distribution of Abnormally Low QRST Areas in Patients With Left Ventricular Hypertrophy
An Index of Repolarization Abnormalities

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Background. QRST isointegral maps (I-maps) have been useful in detecting repolarization abnormalities. We investigated the body surface distribution of abnormally low QRST areas in patients with left ventricular hypertrophy (LVH) and the relation of the abnormalities in I-map to the severity of LVH as assessed by echocardiography.

Methods and Results. QRST area departure maps were constructed from electrocardiographic (ECG) data recorded in patients with LVH and precordial negative T waves resulting from aortic stenosis (AS) (10 patients), aortic regurgitation (AR) (12 patients), or hypertrophic cardiomyopathy (HCM) with asymmetric septal hypertrophy (22 patients). Fifty normal subjects served as controls. The I-map was constructed from 87 body surface electrocardiograms recorded simultaneously at a sampling interval of 1 msec. The area where the QRST area was smaller than normal limits (mean -2 SD) was designated the "-2 SD area." The echocardiographic left ventricular (LV) mass was calculated by Devereux's method. Patients with large LV masses due to AS or AR had 2 SD areas located over the left anterior chest or the midanterior chest, respectively. The 2 SD area was located over the left shoulder and left anterior chest and had a lingual shape in patients with HCM. The sum of QRST area values less than the normal range (ΣQRST) was significantly correlated with LV mass in patients with AS or AR (r = 0.83 and r = 0.69, p < 0.01 and p < 0.05). However, there was no significant correlation between ΣQRST and the severity of LVH in patients with HCM. ΣQRST divided by the number of electrodes in the 2 SD area was significantly greater in patients with HCM than in those with AS or AR.

Conclusions. These findings suggest that abnormalities in patients with HCM are manifest even in mild LVH and that there is a greater disparity of repolarization in hypertrophied left ventricles due to HCM than in LVH due to aortic valve disease. QRST isointegral departure maps may provide ECG evidence of LV mass of patients with AS or AR and of susceptibility to malignant arrhythmias in patients with HCM. (Circulation 1991;84;1505–1515)

The QRST isointegral map (I-map) is based on the concept of the ventricular gradient reported by Wilson et al.1 Since Abildskov et al2 first introduced I-maps and reported that they are in large part independent of activation sequence and dependent on repolarization properties, I-maps have been found useful in detecting the presence or absence of repolarization abnormalities in the presence of changes in QRS complex.3–9 The I-map has also been demonstrated to be useful in detecting disparity of repolarization that is closely related to susceptibility to malignant arrhythmias.10–16

It has been reported that the T wave change associated with left ventricular hypertrophy (LVH) is mainly secondary to QRS changes such as increases in ventricular activation time and R wave amplitude.17 Analyses of QRST time integral values are largely independent of QRS changes.13,17–19 Therefore, analysis of I-maps of patients with LVH should provide information about repolarization abnormalities that may be obscured in ST-T distributions by

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changes secondary to QRS abnormalities. Igarashi et al\(^a\) showed that QRS values over the lower left chest were significantly smaller in patients with concentric LVH due to essential hypertension than in normal subjects. They did not calculate QRS isointegral departure maps (I-departure maps). The departure map technique introduced by Flowers et al\(^{20}\) is useful in identifying abnormal regions in isopotential\(^{21}\) and isointegral maps.\(^{7-9,22,23}\) However, there have been no reports concerning I-departure maps in patients with various types of LVH.

The purposes of the present study were to determine the body surface distribution of abnormally low QRS areas, an index of repolarization abnormalities, in patients with concentric, eccentric, or asymmetric LVH and to determine the relation of the extent of repolarization abnormalities detected by I-departure maps to the severity of LVH as assessed by echocardiography.

**Methods**

**Study Population**

From consecutive patients whose body surface maps were recorded at the Nagoya University Hospital between January 1986 and April 1990, 44 patients (37 men and seven women; mean age, 49.3 years; age range, 17–72 years) satisfying all of the following criteria were selected for this study (Table 1): diagnosis of aortic valve disease or hypertrophic cardiomyopathy (HCM) confirmed by two-dimensional echocardiography including Doppler echocardiography and/or cardiac catheterization, left precordial lead electrocardiograms showing depressed ST segments and/or negative T waves, no other cardiac disease present such as congenital heart disease, myocardial infarction, or other valvular heart diseases, no conduction disturbances such as bundle branch block or preexcitation syndrome, heart rate between 50 and 90 beats/min, and no serum electrolyte imbalance.

Patients comprised the following three subgroups (Table 1): group A (concentric LVH; 10 patients with aortic stenosis [AS]; seven men and three women; mean age, 52.6 years; age range, 24–72 years), group B (eccentric LVH; 12 patients with aortic regurgitation [AR]; 11 men and one woman; mean age, 49.9 years; age range, 36–65 years), and group C (septal hypertrophy; 22 patients with HCM; 19 men and three women; mean age, 47.8 years; age range, 17–65 years). Asymmetric septal hypertrophy with septoposterior wall thickness ratio exceeding 1.3 was present in the echocardiograms of the patients with HCM.

Patients who received digoxin at a dosage of more than 0.1 mg/day metildigoxin or 0.125 mg/day digoxin were excluded from the study.

Echocardiography was performed in all patients. Cardiac catheterization including coronary arteriography was done in 23 patients within 1 week of body surface electrocardiography.

Control subjects were 50 normal individuals (25 men and 25 women; mean age, 32.7 years; age range, 18–52 years) whose chest radiographs, electrocardiograms, and physical examinations were normal.

Informed consent was given by all subjects before participating in the study.

**Body Surface QRS I-Maps**

**Recording and data analysis.** Body surface electrocardiograms were recorded to construct body surface I-maps using an HPM-6500 or VMC-3000 electrocardiograph (Chunichi Denshi Company Ltd., Nagoya, Japan). Because the details of data acquisition and processing have been reported elsewhere,\(^{24}\) we describe them only briefly. Unipolar electrocardiograms were recorded simultaneously from 87 lead points on the chest surface (59 and 28 lead points on the anterior and posterior chest, respectively) with reference to Wilson's central terminal. Standard 12-lead electrocardiograms and the Frank X, Y, and Z lead electrocardiograms were also recorded simultaneously. These electrocardiographic data were scanned by multiplexers, digitized by analog-digital converters at a rate of 1,000 samples/sec, and stored on floppy disks. Two-point baseline adjustment was performed by choosing the flat portion of the TP segment before the P and after the T deflection of the selected PQRS complex. After baseline adjustment, a root-mean-square voltage-versus-time curve based on the X, Y, and Z leads was plotted to help identify the beginning of the QRS and the end of the T deflections, which were manually selected from this curve. The QRS deflection area was calculated by integrating each lead over the appropriate interval and was expressed in millivolts×msec. QRS isointegral contours were separated by 20 mV·msec. The maximum and minimum were indicated by plus and minus

<table>
<thead>
<tr>
<th>No. of Patients</th>
<th>Age (years)</th>
<th>LV mass (g)</th>
<th>(\Sigma\text{QRS (mV sec)})</th>
<th>(\Sigma\text{QRS/N (mV sec)})</th>
</tr>
</thead>
<tbody>
<tr>
<td>AS: 10 (7/3)</td>
<td>52.6±13.9</td>
<td>389.7±148.1</td>
<td>367.8±407.8</td>
<td>16.4±15.6</td>
</tr>
<tr>
<td>AR: 12 (11/1)</td>
<td>49.9±7.8</td>
<td>403.3±115.7</td>
<td>581.6±425.4</td>
<td>20.2±12.4</td>
</tr>
<tr>
<td>HCM: 22 (19/3)</td>
<td>47.8±12.6</td>
<td>350.0±103.0</td>
<td>642.4±442.5</td>
<td>32.2±17.6</td>
</tr>
</tbody>
</table>

All values expressed as mean ±SD.

M, male; F, female; LV, left ventricular; \(\Sigma\text{QRS}\), sum of the value obtained by subtracting the \(\Sigma\text{QRS}\) value of each point in –2SD area of a given map from the normal mean –2SD value; \(\Sigma\text{QRS/N}\), \(\Sigma\text{QRS}\) divided by number of lead points in –2SD area; AS, aortic stenosis; AR, aortic regurgitation; HCM, hypertrophic cardiomyopathy.

\(*p<0.05.\)
signs, respectively. Data were sampled at the resting expiratory level with the subject in the supine position.

QRST I-departure maps. The mean and SD of the normal QRST at each lead point were calculated from data collected in 50 normal subjects. To estimate the deviation of patient data from the normal value, the departure index (DI) at each lead was calculated on the VCM-3000 as follows: DI = (X−mean)/SD, where X represents the QRST at the corresponding lead for each patient.22 Areas where the DI values were less than 2 on the departure map were designated as “−2 SD areas.” The characteristics of the −2 SD area that were evaluated were n, the number of lead points in each −2 SD area; ΣQRST, the sum of the value obtained by subtracting the QRST value for a given patient at each point in a −2 SD area from the normal mean −2 SD QRST value, and ΣQRST/n (Table 1).

Echocardiographic Data

M-mode echocardiograms were recorded in all patients with the Toshiba SSH 40A (Tokyo, Japan) or Hewlett-Packard 77020AC echocardiograph and a strip-chart recorder. Interventricular septal thickness (ST), posterior wall thickness (PWT), and left ventricular internal dimension (LVID) were simultaneously measured at the R wave peak on the electrocardiogram.25 Echocardiographic left ventricular (LV) mass was calculated with the equation of Devereux et al:26

\[
\text{Echocardiographic LV mass (g) } = 1.04 \times \left[ (LVID + PWT + ST)^3 - LVID^3 \right] - 13.6
\]

Coronary Arteriography

In addition to standard cardiac catheterization, coronary arteriography was performed in 23 patients using the Sones or Judkins technique. Significant obstruction was defined as 75% or greater reduction in the cross-sectional area of the coronary artery. Data were evaluated by two observers who were blinded to study findings, including body surface maps. No patient showed a significant coronary artery obstruction.

Statistical Analysis

Statistical analysis was performed using the Student’s t test, and simple correlations were calculated according to standard statistical methods. A probability value of less than 0.05 was considered significant. Values are expressed as mean±SD.

Results

Mean QRST I-Maps

Normal subjects. As shown in Figure 1A, the positive area of the mean I-map of normal subjects was located over the left chest with the maximum at the site of V_{as} of the standard 12-lead electrocardiogram. The negative area was over the upper chest with the minimum at the upper right anterior chest.

Aortic stenosis. The mean I-map of patients with AS (Figure 1B) showed a negative area over the left anterior chest with the minimum one row below the location of V_{as}. The maximum was located over the upper anterior chest, above and to the right of the minimum.

Aortic regurgitation. The mean I-map of patients with AR (Figure 1C) showed a negative area over the anterior chest, and the minimum was over the lower anterior chest. The maximum was located over the left lateral chest above and to the left of the minimum.

Hypertrophic cardiomyopathy. The mean I-map of patients with HCM (Figure 1D) showed a negative area over the mid and upper left anterior chest with the minimum at the site of V_{as} in the 12-lead electrocardiogram. The maximum was located over the anterior chest above and to the right of the minimum.

Electrocardiograms, I-Maps, and I-Departure Maps

Aortic stenosis. Electrocardiograms of a representative patient with AS (59-year-old man) showed an increase in R voltage (as high as 5.8 mV in V_{as}) and asymmetric T inversions with ST depression (Figure 2A). The echocardiographic mass of this patient was 549 g. The coronary arteriograms showed no significant coronary lesion.

In the I-map (Figure 2B), there was a positive area over the anterior chest with the maximum at the midmsternal line. The negative area was over the left anterior chest and back with the minimum one row below V_{as}. The locations of the negative area and of the minimum differed from the distributions in the mean I-map of normal subjects.

There was a large −2 SD area over the lower left lateral chest in the I-departure map (Figure 2C). A −2 SD area was present in the I-departure maps of eight of the 10 patients with AS. The two patients whose maps did not have a −2 SD area had LV masses of 244 and 336 g, respectively.

Aortic regurgitation. Electrocardiograms of a representative patient with AR (44-year-old man) showed an increase in R voltage (as high as 3.2 mV in V_{as}), asymmetric T inversion, and ST depression (Figure 3A). The echocardiographic LV mass of this patient was 630 g. Coronary arteriograms showed no significant coronary lesion.

In the I-map (Figure 3B), there was a positive area over the left lateral chest and back with the maximum over the left lateral chest. The negative area was over the anterior chest with the minimum over the lower left anterior chest. The locations of the negative area and of the minimum differed from the distributions in the mean I-map of normal subjects. There was a large −2 SD area over the lower anterior chest in the I-departure map (Figure 3C). A −2 SD area was present in the I-departure maps of 11 of the 12 patients with AR. The one patient whose map did not have a −2 SD area had an LV mass of 233 g.

Hypertrophic cardiomyopathy. Electrocardiograms of a representative patient with HCM (56-year-old man) showed an increase in R voltage (as high as 5.2
mV in V4), deep negative T waves, and ST depression (Figure 4A). The echocardiographic LV mass of this patient was 371 g. Coronary arteriograms showed no significant coronary lesion.

In the I-map (Figure 4B), there was a positive area over the right anterior chest with the maximum at the midsternal line. A second maximum was located over the lower left lateral chest. The negative area was over the upper left anterior chest and back with the minimum at the site of lead V4. The locations of the negative area and of the minimum differed from the distributions in the mean I-map of normal subjects. There was a -2 SD area over the upper left lateral chest in the I-departure map (Figure 4C). This abnormal distribution of negative areas over the upper left anterior chest was observed in all patients with HCM.

Relation of parameters derived from I-departure maps to LV mass. The correlation coefficient between ΣQRST and echocardiographically determined LV mass was calculated for each group of patients, and the data are shown in Figure 5. The correlation between ΣQRST and LV mass was 0.83 for patients with AS and 0.69 for patients with AR (Figures 5A and 5B). Both of these r values were significant. For patients with HCM, however, there was no significant correlation between ΣQRST and LV mass (Figure 5C).

**Figure 1.** Mean QRST isointegral maps of normal subjects (panel A) and of patients with aortic stenosis (panel B), aortic regurgitation (panel C), or hypertrophic cardiomyopathy (panel D). Isointegral contours are separated by 20 mV·msec in panels A, C, and D and by 12 mV·msec in panel B. Shading indicates negative areas. Maxima and minima are indicated by plus and minus signs. ● Six precordial lead points of 12-lead electrocardiograms. Plus sign overlaps with one of the six closed circles.
As shown in Figure 6, the correlations between $\Sigma_{QRST}/n$ and LV mass were significant in patients with AS and AR ($r=0.79$ and $r=0.62$) and insignificant in patients with HCM. However, the $\Sigma_{QRST}/n$, which reflects the severity of repolarization abnormalities per recording electrode, was significantly greater in maps of patients with HCM than in maps of patients with AS or AR ($p<0.05$), although there was no significant difference in LV mass of the patients with HCM, AS, or AR (Table 1). The relation of $\Sigma_{QRST}$ to the ratio of the thickness of the interventricular septum to the thickness of the posterior wall and of the relation of $\Sigma_{QRST}$ to the sum of the thicknesses of the interventricular septum and posterior wall were also examined. As shown in Figure 7, there were no significant correlations between these parameters.

**Discussion**

In the present study, we observed that the I-departure map of patients with LVH had characteristic body surface distributions of the $-2$ SD area that depended on the cause of LVH; that $\Sigma_{QRST}$ was highly correlated with the echocardiographic LV mass in patients with AS or AR, but there was no significant correlation between $\Sigma_{QRST}$ and the LV mass of patients with HCM; and that compared with maps of patients with aortic valve diseases, maps of patients with HCM showed greater values of $\Sigma_{QRST}/n$. This finding suggests that repolarization abnormalities may be more severe in HCM patients than in patients with aortic valve disease.

It has been reported that the QRS/T deflection area is largely independent of activation sequence and dependent on repolarization properties. Based
on this concept of the ventricular gradient, Abildskov et al. introduced an I-map. They demonstrated that the I-map is largely independent of the activation sequence and useful in detecting repolarization abnormalities, even in the presence of QRS deflection abnormalities. In addition, Montague et al. reported that an I-map is a useful method for compressing the extensive data obtained from body surface mapping. Since the introduction of the I-map, there have been several reports concerning its usefulness in detecting repolarization abnormalities associated with essential hypertension and myocardial ischemia and infarction and abnormalities associated with the increased vulnerability to arrhythmia associated with acute myocardial infarction and the long QT syndrome. Igarashi et al. found significantly smaller QRST values over the lower left chest of patients with concentric LVH due to essential hypertension than those from maps of normal controls. They attributed the smaller QRST values over the lower left chest to repolarization abnormalities resulting from LVH. Devereux et al. demonstrated a high incidence of negative T waves in 12-lead electrocardiograms in patients with large echocardiographically determined LV masses resulting from hypertension, AR, or AS. These findings are in accordance with previous reports that advanced LVH resulting from either pressure or volume overload causes repolarization abnormalities by subendocardial hypoperfusion or fibrosis.

In the present study, we estimated the severity and distribution of repolarization abnormalities from I-departure maps of patients with concentric and eccentric LVH or HCM. The distributions of positive
and negative areas and of the $-2$ SD area differed in each group of patients. We also found a significant correlation between $\Sigma$QRST and the LV mass in patients with AS ($r=0.83$, $p<0.01$) or AR ($r=0.69$, $p<0.05$). These findings support quantitatively previous reports that greater repolarization abnormalities are associated with more advanced LVH.28-32 On the other hand, there was no significant correlation between $\Sigma$QRST and LV mass in patients with HCM, and large values of $\Sigma$QRST were found in maps of some HCM patients with small LV masses (Figure 5C). This finding indicates that repolarization abnormalities occur in patients with HCM even when LVH is mild. Maron et al33 reported that there is a possibility of sudden death in patients with HCM and normal LV masses. Typical myocardial structural abnormalities indicating HCM have been found in such patients. Spirito and Maron34,35 showed a significantly higher prevalence of severe LVH in patients with HCM who died suddenly, but they also demonstrated that four of 29 patients (14%) who died suddenly showed only mild LVH. We showed that $\Sigma$QRST and $\Sigma$QRST/n are independent of the degree of hypertrophy in patients with HCM. The reasons for this are unclear. However, pathological abnormalities such as disarrangement of the myocardium and small-vessel disease are highly characteristic findings in HCM, even in patients with HCM and normal LV masses, and might play important roles in the independence of $\Sigma$QRST and $\Sigma$QRST/n from the degree of hypertrophy in HCM. The finding of severe repolarization abnormalities in HCM, even in those with mild LVH (Figure 5C), may explain in part their high vulnerability to arrhythmias.
Kubota et al. found a high inverse correlation between ventricular fibrillation threshold and alterations in cardiac surface QRST areas. In their study, the increase in QRST area resulted from a decrease in ventricular repolarization properties that was produced by warming the surface of the heart. They speculated there would be a high positive correlation between fibrillation threshold and QRST alterations because of prolongation of repolarization properties in localized areas. Abildskov et al. used computer simulations and found that small severe lesions produced striking decreases in fibrillation threshold. The greater value of the mean QRST in patients with HCM than in those with aortic valve disease, as shown in this study, may be related to the higher vulnerability to arrhythmias of patients with HCM compared with those with aortic valve disease.

There are some limitations in the present study. First, the study population was small; further study is indicated in a larger population that includes patients with malignant arrhythmias in each group. Second, although the dose was quite small, the administration of digitalis may have affected the I-maps of the 14 patients receiving this drug at the time of electrocardiography. However, that the I-maps recorded from patients taking digitalis did not show high ST areas in the two patients with small LV masses indicates that the effects of digitalis on QRST areas were minimal. The ST depression resulting from effects of the drug should decrease the QRST areas. Therefore, QRST in patients with aortic valve disease may have been overestimated. None of the patients with HCM received digitalis. Accordingly, the actual differences in mean QRST and QRST between the patients with HCM and those with aortic valve disease may be much greater than indicated by the data shown in Table 1. Class II antiarrhythmic agents were administered to some

**Figure 5.** Scatterplots of echocardiographic left ventricular (LV) mass (g) plotted against sum of value obtained by subtracting QRST value of each point in -2 SD area of a given map from normal mean -2 SD QRST value (ΣQRST) (mV·msec). Data are from patients with aortic stenosis (panel A), aortic regurgitation (panel B), or hypertrophic cardiomyopathy (panel C).
patients with HCM, and it is possible that these agents may have affected the QRST areas. However, a previous report has shown that the effect of these agents on QRST areas is minimal.6 Accordingly, it appears unlikely that the agents significantly influenced the QRST areas recorded from patients with HCM. Finally, it is possible that some of the QRST abnormalities seen in these patients were due to ischemic heart disease. However, coronary arteriograms were obtained in 23 patients, and none had significant coronary stenosis.

It is generally accepted that the specificity of T wave changes is rather low despite their high sensitivity.37 The results of the present study indicate that analysis of I-maps and -2 SD areas provides information about the location and severity of repolarization abnormalities that is not available in standard 12-lead electrocardiograms. Furthermore, features of the I-maps correlated with LV mass of patients with AS or AR but not of patients with HCM. This suggests that I-maps could be used to estimate severity of LVH in selected groups of patients. In addition, the occurrence of abnormalities in I-maps of HCM patients with small LV masses suggests that abnormalities of repolarization precede the development of hypertrophy in these patients. Because repolarization abnormalities may be a factor in arrhythmia vulnerability in HCM patients, studies evaluating the prognostic usefulness of I-maps of these patients appear to be indicated.

**Acknowledgments**

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FIGURE 7. Scatterplots of sum of value obtained by subtracting
QRST value of each point in -2 SD area of a given map from
normal mean -2 SD QRS value (ΣQRST) (mV·msec)
plotted against interventricular septal thickness (IVS) over poste-
rior wall thickness (PW) (panel A) and IVS plus PW (panel B)
in patients with hypertrophic cardiomyopathy.

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