Improved Diagnostic Performance on the Severity of Left Ventricular Hypertrophy With Body Surface Mapping

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To improve the diagnostic usefulness of electrocardiography (ECG) in determining the severity of left ventricular hypertrophy (LVH) with body surface mapping, 87 unipolar ECGs were recorded from 57 patients with left ventricular (LV) concentric hypertrophy and 30 with LV dilatation. Body surface ECG features due to LVH were evaluated by increase of QRS voltage and delayed local activation. We measured for each lead R voltage, net area of QRS (AQRS), ventricular activation time (VAT), and departure index (DI) of AQRS and VAT (DI=mean/SD). From these measurements, seven parameters were calculated for each patient: \( R_{\text{max}} \), the maximal R wave voltage; AQRS\(_{\text{max}} \), the maximal AQRS; AQRS-D\(_{\text{max}} \), the maximal AQRS DI; AQRS-D\(_{\text{area}} \), the area size where DIs of AQRS are more than 2; VAT\(_{\text{max}} \), the maximal VAT; VAT-D\(_{\text{max}} \), the maximal VAT DI; and VAT-D\(_{\text{area}} \), the area size where DIs of VAT are more than 2. Among these parameters, the most effective for diagnosis of LVH were selected by stepwise multiple regression analysis. In the concentric hypertrophy group, the combination of VAT-D\(_{\text{area}} \) and R\(_{\text{max}} \) was determined to be the best for estimating wall thickness. The regression equation determined from them correlated well to wall thickness \((r=0.73)\). In the LV dilatation hypertrophy group, only AQRS\(_{\text{max}} \) was selected for estimating LV dilatation. A good correlation between AQRS\(_{\text{max}} \) and LV internal dimension was observed \((r=0.73)\). With the body surface distribution of VAT prolongation, septal hypertrophy was separated from the other LVH. These were superior to the conventional method of 12-lead ECGs. ECG diagnosis of LVH severity improved by incorporating a mapping study. Also, prolongation of VAT and increase in QRS voltage were shown to be important when determining the severity of LVH. (Circulation 1989;79:312–323)

The electrocardiogram (ECG) is a simple, noninvasive tool used for the diagnosis of left ventricular hypertrophy (LVH). However, conventional ECG criteria are currently insufficient when determining the severity of increased wall thickness and dilatation of the left ventricular (LV) chamber.

Previous studies have not shown good correlations of QRS voltages with wall thickness and LV dimension.\(^1,2\) QRS amplitude is presently thought to be insufficient to properly diagnose the level of LVH severity. Up until now, however, the effect of ventricular activation time (VAT) prolongation on the diagnosis of LVH has not been fully studied. In LVH, VAT prolongation (or QRS duration) was shown to enhance the ability to diagnose LVH.\(^3–5\) Therefore, it is believed that prolongation of VAT in addition to QRS voltage could also have diagnostic significance. In the present study, the normal range of VAT in multiple leads was estimated and the prolongation of VAT evaluated quantitatively. Further investigation may well show that VAT prolongation may help to improve LVH diagnostic ability and would serve to complement the present QRS voltage criteria.

Body surface mapping techniques have increased diagnostic usefulness above standard ECG techniques. A large quantity of ECG data obtained from multiple lead systems is expected to increase ability to diagnose the level of LVH severity. Recently, new body surface mapping techniques such as isointegral analysis\(^6–9\) and isochronal analysis\(^10\) have been used to interpret ECG data. With these techniques, it is possible to simplify large quantities of

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mapping data and, therefore, to more easily recognize abnormalities. The AQRS isointegral map is used to detect increases in QRS voltage; the VAT isochrone map, to detect delays of intraventricular conduction. These maps are used when attempting to diagnose the severity of LVH. The purpose of this study was to examine how alteration of ventricular activation sequence occurs in LVH and to improve the ability to diagnose the severity of LVH with the new technique of body surface mapping.

Methods

Study Population

The study group consisted of 87 patients with LVH, ranging in age from 16 to 70 years (mean, 48 years). The patients were divided into three groups: 34 patients with hypertensive heart disease (group HT), 23 patients with hypertrophic cardiomyopathy (group HCM), and 30 patients with aortic regurgitation (group AR). Group HCM was divided into two subgroups based on wall thickness estimated by echocardiogram: group HCM-A (interventricular septum [IVS] thickness [t] ≥13 mm and left ventricular posterior wall [LVPW] t ≥13 mm); and group HCM-B (IVS t ≥13 mm and LVPW t <13 mm). Groups HT and HCM formed the concentric hypertrophy group, and group AR formed the LV dilatation group. These 87 patients were treated as a learning series from which mapping criteria were determined.

The following patients were excluded from the study: 1) patients showing clinical evidence of congestive heart failure, myocardial infarction, or right ventricular overload, such as ventricular septal defect and patent ductus arteriosus; 2) patients having prolonged QRS duration (>100 msec) or other ventricular conduction disturbances such as bundle branch blocks; 3) patients showing either tachycardia (>100 beats/sec) or bradycardia (<50 beats/sec); and 4) patients showing enlargement of right ventricle (RV) dimension (>27 mm) as estimated by M-mode echocardiography.

Diagnostic angiocardiography was performed on all group HCM and group AR patients, excluding those showing high pulmonary artery pressure (mean, >25 mm Hg). In all subjects, echocardiograms were recorded within 2 weeks after the ECG recording to estimate wall thickness and LV and RV internal dimensions and to exclude other heart diseases.

To investigate the diagnostic performance of our criteria, we studied an additional 76 subjects. This group, treated as a test series, comprised 40 clinically normal male volunteers and 36 patients (16 with hypertension, nine with hypertrophic cardiomyopathy, nine with aortic regurgitation, and three with combined valvular disease). All subjects gave their consent to the proceedings before the study commenced.

Body Surface Mapping

Recording. Body surface mapping was performed with a body surface potential mapping system (Model HPM-5100, Chunichi Denshi, Nagoya, Japan). Eighty-seven body surface leads were arranged on each patient’s body in a lattice-like pattern (13×7 matrix) except for the lead points in the midaxillary line. The leads covered the entire thoracic surface (59 leads on the anterior chest and 28 on the back). ECGs from these 87 unipolar leads, with Wilson’s central terminal as reference, standard 12-lead ECGs, and the Frank X, Y, and Z ECGs were sampled simultaneously. The stored signals from each ECG were then displayed on a graphic terminal (Model 4006–1, Tektronix, Beaverton, Oregon). If noise was detected in any of the signals, data sampling was repeated. The flat portion of the PQ segment was chosen to be the baseline. After performing the baseline adjustment, data were recorded on a magnetic cassette tape with a digital format. This system had a resolution of 0.01 mV in the dynamic range ±5 mV, with a sampling frequency of 1,000 samples/sec/channel. The data sampling was performed at the resting expiratory level and in the supine position.

Data analysis. The mapping data were processed offline on a minicomputer (VAX 11/750, Digital Equipment, Maynard, Massachusetts) with the analysis program for further analysis developed by our institution. For this analysis, the QRS onset and offset were determined from the superimposed Frank X, Y, and Z leads and the spatial magnitude.

The following measurements were generated automatically by computer for each lead: R wave amplitude; AQRS (the sum of all positive and negative potentials from QRS onset to QRS offset); and VAT (the duration of time from QRS onset to the time of the most rapid decrease in QRS voltage [dV/dt\text{max}]). The body surface distributions of R voltage, AQRS, and VAT were displayed in the form of a map and called the “R map,” the “AQRS isointegral map,” and the “VAT isochrone map,” respectively.

Departure maps. From the 40 normal volunteers, the mean and the standard deviation (SD) of normal AQRS and VAT at each lead point were determined. To estimate the deviation of patient data from the normal value, the departure index at each lead was calculated as follows:

\[
DI = (X - \text{mean}) / \text{SD},
\]

where X represents the AQRS or VAT at the corresponding lead of each patient. Because we were interested in the increase of AQRS and the prolongation of VAT, the areas where the DI values were more than 2 on the departure map were designated as “+2 SD areas.” The area size on each torso was also calculated.

On the maps, the rectangular area represented the torso surface, with the left half of the maps reflecting the anterior chest and right half reflecting the back. Therefore, both the right and left edges of the maps represented the right midaxillary line.


Mapping parameters. Seven mapping study parameters were calculated for each patient: $R_{max}$, maximal R wave amplitude; $AQRS_{max}$, maximal value of AQRS; $AQRS-D_{max}$, maximal value of departure index for AQRS; $AQRS-D_{area} +2 SD$ area size on body surface in AQRS departure map; $VAT_{max}$, maximal value of VAT; $VAT-D_{max}$, maximal value of departure index for VAT; and $VAT-D_{area} +2 SD$ area size on body surface in VAT departure map. These were presumed to represent the degree of abnormally increased AQRS or abnormally prolonged VAT.

Standard 12-Lead ECG

Standard 12-lead ECGs were recorded at 25 mm/sec and 1 mV/cm. The following ECG criteria for LVH diagnosis were evaluated for comparison with our mapping data: Sokolow and Lyon precordial voltage ($SV_1 + RS_2$ or $V_6$), the point score system of Romhilt and Estes, and the regression equation developed by Casale et al (see Appendix 1).

Echocardiographic Data

M-mode ECGs were recorded from all subjects with a Hitachi EUB-10 echocardiograph (Tokyo, Japan) and strip chart recorder. Patients were examined in the recumbent position with the transducer placed from the third to the fifth intercostal space lateral to the left sternal border. Echoes from the posterior wall of the left ventricle, interventricular septum were recorded immediately below the mitral leaflets. Internal septal thickness (ST), posterior thickness (PWT), LV internal dimension (LVID), and RV internal dimension (RVID) were simultaneously measured at the R wave peak on the ECG as recommended by the American Society of Echocardiography. Sum of the wall thickness (SWT) is expressed as $ST + PWT$ and was used as an index of wall thickness increase; LVID was used as an index of LV dilatation. Echocardiographic LV mass was calculated by the following equation developed by Devereux and Reichek:

$$Echocardiographic\ LV\ mass\ (g) = 1.04 \times ((LVID + PWT + ST)^3 - (LVID)^3) - 13.6$$

Statistical Analysis

Computer analysis was conducted at the Yamagata University Computer Center with SPSSX (Statistical Package for the Social Sciences–X) to perform simple correlation analysis, multiple stepwise regression analysis, and factor analysis (see Appendix 2). When performing multiple stepwise regression analysis, the enter criterion used was 3.84 of the $F$ value, and the removal criterion used was 2.71 of the $F$ value. Standard statistical definitions were used for sensitivity, specificity, and overall accuracy.

Results

Map Patterns

Normal subjects. The body surface distributions of mean AQRS and mean VAT of the 40 normal volunteers are shown in Figure 1. In the mean AQRS map for normal subjects, positive values occur on the left anterior and lower chest, whereas negative values occur on the right upper anterior chest and right upper back. As shown on the VAT map, the shortest VATs occurred on the right upper back. The VAT map shows isochronous lines extending from the right upper back and right upper chest to the left anterior chest, then left to the back. Figure 2 shows a set of maps from a 41-year-old normal subject. The AQRS and VAT maps shown in Figure 2 are similar to mean maps for normal subjects. The +2 SD areas were not found on departure maps for normal subjects.

Concentric hypertrophy group (groups HT and HCM). The group mean AQRS and VAT maps are illustrated in Figure 1. These maps indicate an increase in both AQRS and prolongation of VAT for the left anterior and lateral chest.

Figures 3 and 4 show maps taken from patients in groups HT and HCM-A. In these groups, R voltage and AQRS increased on the left lateral chest, and VATs were prolonged around the region. On AQRS departure maps, +2 SD areas were observed on the left lateral chest. On VAT departure maps, +2 SD areas can be observed on the left upper and left lateral chest.

Figure 5 shows maps taken from a group HCM-B case. In this group, AQRS increases are located on the middle anterior chest, and VAT prolongations are located on the upper anterior chest. The locations of VAT +2 SD areas were especially characteristic in group HCM-B. Figure 6 shows the group mean maps of groups HCM-A and HCM-B. In mean VAT map of the HCM-B group, characteristic prolongation of VAT is observed on the upper anterior chest. These findings for group HCM-B were different from those of groups HT, HCM-A, and AR, whereas the mean maps of group HCM-A were similar to those of groups HT and AR.

Left ventricular dilatation group (group AR). Figure 7 presents maps taken from a group AR patient. AQRS and R voltage increases occurred on the left middle to upper chest. Prolongation of VATs also occurred in the same region. The +2 SD areas were located on the left upper chest region as well. Group mean AQRS and VAT maps (Figure 1) show both increased AQRS and VAT prolongation on the left lateral chest.

Relation of ECG Mapping Parameters to Wall Thickness and Left Ventricular Internal Diameter

Table 1 gives data for echocardiographic measurements taken from each group. SWT in the concentric hypertrophy group was increased, as was LVID in the LV dilatation group.
Simple correlations between mapping parameters, SWTs, and LVIDs are listed in Table 2. In the concentric hypertrophy group, SWTs have been correlated to all mapping parameters \((r=0.33-0.68)\). The VAT-D area had the best correlation coefficient \((r=0.68)\) among all parameters. The VAT-D\(_{\text{max}}\) and AQRS-D\(_{\text{max}}\) parameters yielded the next best correlations (both \(r=0.63\)) with the SWTs; however, these parameters did not correlate significantly with the LVIDs.

In the LV dilatation group, all mapping parameters, with exception of the VAT\(_{\text{max}}\), had significant correlations \((r=0.43-0.73)\) with the LVIDs. AQRS\(_{\text{max}}\) yielded the best correlation coefficient \((r=0.73)\). The AQRS-D area and R\(_{\text{max}}\) yielded the next-best correlations \((r=0.55\) and \(r=0.57\), respectively) with the LVIDs, but these parameters did not correlate significantly with the SWTs.

Stepwise multiple regression analysis, using data from the 87 patients of the learning set, was performed to determine which parameters were most important for estimation of SWT and LVID (Table 3). In the concentric hypertrophy group, the combination of VAT-D area and R\(_{\text{max}}\) was determined to be the best index for determining SWT.

### Table 1. Study Group and Echocardiographic Data

<table>
<thead>
<tr>
<th>Group</th>
<th>Mean age (yr)</th>
<th>SWT</th>
<th>LVID</th>
</tr>
</thead>
<tbody>
<tr>
<td>Concentric hypertrophy</td>
<td>57</td>
<td>27.6±6.4*</td>
<td>47.3±6.3</td>
</tr>
<tr>
<td>Group HT</td>
<td>34</td>
<td>24.8±5.4†</td>
<td>48.6±6.2</td>
</tr>
<tr>
<td>Group HCM</td>
<td>23</td>
<td>31.7±5.6*</td>
<td>45.4±6.9</td>
</tr>
<tr>
<td>LV dilatation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(group AR)</td>
<td>30</td>
<td>21.4±3.9</td>
<td>58.9±10.0§§</td>
</tr>
</tbody>
</table>

SWT, sum of wall thickness (septal thickness and posterior wall thickness); LVID, left ventricular end-diastolic internal dimension.

*\(p<0.001\); †\(p<0.01\) compared with LV dilatation group; ‡\(p<0.001\) compared with LV dilatation group; §\(p<0.001\) compared with group HT; §§\(p<0.001\) compared with group HCM.
With the equation \( y (\text{mm}) = 18.7 + 0.0122 \cdot \text{VAT-Darea} + 2.02 \cdot \text{Rmax} (\text{mV}) \), a good correlation coefficient (multiple \( R \)) of 0.73 was obtained. The standard regression coefficient (\( \beta \)) of VAT-Darea was determined to be 0.58 and of Rmax was determined to be 0.29. This indicated that when determining correlation coefficients, relative importance of VAT-Darea was greater than that of Rmax. When determining LVID, only AQRS-Dmax showed a significant effect, but this effect was weak (\( r = 0.29 \)).

In the LV dilatation group, AQRSmax was exclusively selected as the best index for the estimation of LVID. Inclusion of any other parameters did not significantly increase the regression coefficient.

**TABLE 2. Simple Correlation Between Mapping Parameters and Echocardiographic Data**

<table>
<thead>
<tr>
<th>Map index</th>
<th>Concentric hypertrophy</th>
<th>LV dilatation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SWT</td>
<td>LVID</td>
</tr>
<tr>
<td>Rmax</td>
<td>0.48*</td>
<td>−0.17</td>
</tr>
<tr>
<td>AQRSmax</td>
<td>0.54*</td>
<td>−0.25</td>
</tr>
<tr>
<td>AQRS-Dmax</td>
<td>0.63*</td>
<td>−0.29</td>
</tr>
<tr>
<td>AQRS-Darea</td>
<td>0.54*</td>
<td>−0.23</td>
</tr>
<tr>
<td>VATmax</td>
<td>0.33†</td>
<td>−0.15</td>
</tr>
<tr>
<td>VAT-Dmax</td>
<td>0.63*</td>
<td>−0.26</td>
</tr>
<tr>
<td>VAT-Darea</td>
<td>0.68*</td>
<td>−0.25</td>
</tr>
</tbody>
</table>

*Map index, see text; SWT, sum of wall thickness (septal thickness + posterior wall thickness); LVID, left ventricular end-diastolic internal dimension.

With \( \text{VAT-Darea} \) and \( \text{AQRS-Dmax} \), intervals of contour lines were 1.0 mV on R map, 20 \( \mu \)Vsec on AQRS map, 5 msec on VAT map, and 1 departure index on each departure map. On departure maps, + area indicates a relative increase in AQRS and prolonged VAT. +, Maximum; −, minimum.

**FIGURE 2.** Set of maps from a 41-year-old normal volunteer. Top left panel: R map. Middle left panel: Area of QRS (AQRS) map. Lower left panel: AQRS departure map. Upper right panel: Ventricular activation time (VAT) isochrone map. Lower right panel: VAT departure map; maximum R wave voltage (Rmax), 2.20 mV. The AQRS and VAT maps resemble normal mean maps. Areas of departure index values greater than 2 (+ 2 SD areas) are not found on departure maps. Intervals of contour lines are 1.0 mV on R map, 20 \( \mu \)Vsec on AQRS map, 5 msec on VAT map, and 1 departure index on each departure map. On departure maps, + area indicates a relative increase in AQRS and prolonged VAT. +, Maximum; −, minimum.

**TABLE 3. Stepwise Multiple Regression Analysis for Determination of Wall Thickness and Left Ventricular Dilatation With Mapping Parameters**

<table>
<thead>
<tr>
<th>Class 1</th>
<th>VAT-Darea (0.58)</th>
<th>AQRS-Dmax</th>
<th>. . .</th>
<th>AQRSmax</th>
</tr>
</thead>
<tbody>
<tr>
<td>SWT</td>
<td>LVID</td>
<td>SWT</td>
<td>LVID</td>
<td></td>
</tr>
<tr>
<td>Class 2</td>
<td>Rmax (0.29)</td>
<td>. . .</td>
<td>. . .</td>
<td>—</td>
</tr>
<tr>
<td>Multiple R</td>
<td>0.73</td>
<td>0.29</td>
<td>0.73</td>
<td></td>
</tr>
<tr>
<td>( R^2 )</td>
<td>0.54</td>
<td>0.08</td>
<td>. . .</td>
<td>0.54</td>
</tr>
<tr>
<td>( F )</td>
<td>31.0*</td>
<td>4.9†</td>
<td>. . .</td>
<td>31.5*</td>
</tr>
</tbody>
</table>

SWT, sum of wall thickness (septal thickness and posterior wall thickness); LVID, left ventricular end-diastolic internal dimension. Number in parentheses, standard regression coefficient (\( \beta \)). Map index, see text. \( \text{R} < 0.001, \text{R} < 0.05. \)
Using the equation \( y \ (\text{mm}) = 43.1 + 0.193 \cdot \text{AQRS}_{\text{max}} \ (\mu \text{Vsec}) \), a good correlation coefficient of 0.73 was obtained. For the determination of SWT, no parameters were shown to be effective.

**Factor Analysis of Mapping Parameters**

Because all mapping parameters were mutually dependent, a factor analysis (Table 4) was performed to determine the fundamental factors within these parameters. In the factor analysis, two primary factors were selected. \( R_{\text{max}} \), AQRS\(_{\text{max}}\), AQRS-D\(_{\text{max}}\), and AQRS-D\(_{\text{area}}\) parameters showed a strong relation to factor 1 (0.744–0.875). VAT\(_{\text{max}}\), VAT-D\(_{\text{max}}\), and VAT-D\(_{\text{area}}\) parameters showed a strong relation to factor 2 (0.801–0.865). ECG changes were affected by two different components due to the presence of LVH.

**Comparison With Standard 12-Lead ECG**

The diagnostic ability of the mapping study was compared with that of the standard 12-lead ECG. Table 5 shows the differences between correlation coefficients obtained from estimation with a mapping study and those obtained with a 12-lead ECG study. The estimations of SWT obtained from 12-lead ECGs ranged from 0.44 to 0.59. The regression equation obtained from the mapping study was shown to be superior to those from the standard 12-lead ECG when determining SWT in the concentric hypertrophy group (\( r = 0.73 \)).

**Detection of the Presence of Left Ventricular Hypertrophy**

The diagnostic ability of mapping studies and 12-lead ECG studies on the presence or absence of LVH was examined in the 76 subjects in the test series. Definition of LVH was performed by using two different criteria: echocardiographic LV mass greater than 215 g and either SWT equal to or
FIGURE 4. Set of maps from a patient with hypertrophic cardiomyopathy (HCM-A group, refer to text). Map display is same as in Figure 2. Ventricular activation times (VATs) are prolonged out of normal range on upper left anterior chest (50–55 msec) and left lateral chest (60–70 msec). R voltage and AQRS are also increased in these regions. Areas of departure index values greater than 2 are observed on each departure map.

greater than 26 mm or LVID equal to or greater than 60 mm. We estimated SWT and LVID from the regression equation developed in the present study, and echocardiographic LV mass was calculated from the estimated SWT and LVID. The cutoff value to determine the presence of LVH in the mapping study was estimated LV mass greater than 215 g or either estimated SWT equal to or greater than 26 mm or estimated LVID equal to or greater than 60 mm. Sensitivity, specificity, and predictive accuracy of the regression equation and criteria of the 12-lead ECG are shown in Table 6.

In the first category (echocardiographic LV mass over 215 g), the regression equation from the mapping study correctly diagnosed 88% of the subjects (sensitivity 100%, specificity 78%), whereas in the 12-lead ECG tests, overall test accuracy ranged from 63% to 88%. In the second category (SWT ≥ 26 mm or LVID ≥ 60 mm), the regression equation from the mapping study correctly diagnosed 88% of the subjects (sensitivity 100%, specificity 82%), whereas in the 12-lead ECG tests, overall test accuracy ranged from 71% to 80%.

Discussion

It has been reported that AQRS isointegral maps, constructed from the net area of QRS complex, were useful in evaluating the global changes in QRS

| Table 5. Comparison of Mapping Study With Standard Method of 12-Lead Electrocardiograms: Correlation Coefficients Between Mapping or Electrocardiographic Parameters and Echocardiographic Data |
|---------------------------------|---------------|----------------|----------------|
|                                 | Concentric hypertrophy | LV dilatation |
|                                 | SWT            | LVID           | SWT            | LVID           |
| Regression from map             | 0.73           | 0.29           | . .            | 0.73           |
| 12-Lead electrocardiogram       |                |                |                |                |
| Sokolow-Lyon voltage            | 0.44           | . .            | . .            | 0.57           |
| Romhilt-Estes point score       | 0.59           | . .            | . .            | 0.54           |
| Casale exponent                 | –0.47*         | . .            | . .            | –0.54*         |

SWT, sum of wall thickness (septal thickness and posterior wall thickness); LVID, left ventricular end-diastolic internal dimension; regression from map, multiple regression coefficient obtained from mapping study.

*Except for three patients with atrial fibrillation.
FIGURE 5. Set of maps (upper panel) and 12-lead ECG (lower panel) obtained from a patient with hypertrophic cardiomyopathy (HCM-B group, refer to text). Map display is the same as in Figure 2. Ventricular activation times (VATs) are prolonged on upper anterior chest. Area of QRS (AQRS) is increased on middle anterior chest. The areas of departure index values greater than 2 (+2 SD areas) of both are located in that region. Locations of +2 SD areas are characteristic of group HCM-B patients. In this case, estimated SWT from the regression equation was 28 mm, estimated left ventricular internal dimension was 51 mm. Measured values were 30 and 49 mm, respectively. Increased wall thickness was predicted. Conversely, all 12-lead ECG tests gave false-negative results (Sokolow-Lyon voltage, 30 mm [3.0 mV]; Romhilt-Estes point score, 3; Casale equation, −1.50 [sinus rhythm]).
complex of myocardial infarction\textsuperscript{6–8} and lung disease.\textsuperscript{9} VAT isochrone maps, constructed from the distribution of VATs on the body surface, are also useful tools to detect abnormal activation sequence in myocardial infarction.\textsuperscript{10} Moreover, departure maps reveal the abnormality of the ECG measurements when they exceed normal ranges. In this study, departure maps for both VAT and AQRS have been constructed. Previous studies have described the superiority of departure maps of instantaneous QRS voltage or AQRS to original maps,\textsuperscript{7,8,17,18} With the same methodology, the normal range of VAT for each lead was determined and the VAT prolongation quantitatively evaluated.

Flowers et al.,\textsuperscript{17,18} who first described the departure map, used the value (X–mean–2 SD) as a DI for the detection of abnormal positivity due to the delay of the local activation in myocardial infarction. In our institution, the formula (X–mean–2 SD) was used as a DI.\textsuperscript{7,8} This formula is thought to be superior to the original for the detection of both abnormal negativity and abnormal positivity. Furthermore, because the formula is compatible with the Mahalanobis generalized distance, this index indicates the extent of the deviation from normal subjects. The maximum and the minimum points imply the points at which the most prominent changes occur.

The minimum dV/dt point was chosen as the point for the estimation of VAT in this study. It was thought that the minimum dV/dt point was superior to the peak R point for detection of abnormal VAT prolongation, because the standard deviations in the minimal dV/dt of normal subjects were less than those in the peak R VAT, and therefore, the minimal dV/dt point was thought to be relatively stable in recognition during various states (respiration or position).

**Interpretation of Maps**

Results revealed that VAT prolongation and increase of AQRS were located on the left lateral chest for groups HT, HCM-A, and AR. It was difficult to separate these groups according to the VAT and AQRS +2 SD locations. In the HCM-B (septal hypertrophy) group, however, the location of the +2 SD area of the VAT could be differentiated from the other group. The VAT prolongation out of normal range was shown on the upper anterior chest. This finding allowed the HCM-B group to be differentiated from the other groups.

Localized hypertrophied myocardium of the septal wall was responsible for the characteristic pattern of VAT distributions on a torso. On the other hand, diffuse hypertrophy of the LV wall, as in groups HT, HCM-A, or AR, caused a similar VAT distribution pattern that was observed in diffuse hypertrophy of the LV wall, regardless of differences in etiology of the hypertrophy.

Figure 5 shows a merit of mapping study, capturing localized LVH such as septal hypertrophy. In this case, the map yielded true-positive results, but 12-lead ECG tests gave false-negative results. Mapping study is thought to be beneficial when evaluating localized LV hypertrophy.

**Correlation of Mapping Parameters to Wall Thickness and Left Ventricular Internal Diameter**

The results of the study showed that the combination of VAT-D_{area} and R_{max} yielded the best diagnostic index on severity of wall thickness in concentric hypertrophy and that AQRS_{max} was the best...
index for detection of LV dilatation in eccentric hypertrophy. These diagnostic performances of the mapping technique proved to be better than those of the conventional 12-lead ECG method.

From the factor analysis results, two primary factors were selected. The variation of ECG parameters in LVH was explainable by the interpretation from these two factors. Factor 1 correlated well with \( R_{\text{max}} \), \( \text{AQRS}_{\text{max}} \), \( \text{AQRS-D}_{\text{max}} \), and \( \text{AQRS-D}_{\text{area}} \). Factor 2 correlated well with \( \text{VAT}_{\text{max}} \), \( \text{VAT-D}_{\text{max}} \), and \( \text{VAT-D}_{\text{area}} \). We suppose that factor 1 represents the increase of QRS voltage, and factor 2 represents the prolongation of VAT. ECG changes in LVH were constructed from the variation of the two primary factors, QRS voltage increase and VAT prolongation.

Wall thickness in concentric hypertrophy was determined by both factors, whereas the LV dilation in the LV dilatation was determined by one factor, \( \text{AQRS}_{\text{max}} \). This indicates that VAT prolongation in concentric hypertrophy occurred independently of QRS increase. On the other hand, VAT prolongation in the LV dilatation was dependent on QRS voltage increase. We supposed that VAT prolongation in the concentric hypertrophy group was caused by mechanisms different from those in the LV dilatation group.

Increased QRS voltages are thought to be caused by many factors: increased left ventricular mass, increased left ventricular surface, increased intracavitary blood volume, closer proximity of ventricle to chest. Possible mechanisms caused by VAT prolongation are thought to be conduction delay caused by a damaged or stretched left conduction pathway, increased thickness of the LV wall, and the dilated LV. VAT prolongation is presumed not to be less influenced by extracardiac factors, whereas QRS voltage is influenced by some extracardiac factors such as adipose tissue or muscle on the chest and the distance between chest and heart. Therefore, a VAT prolongation gives important information for diagnosis of LVH. Evaluation of the prolongation was considered meaningful for LVH diagnosis. Integration of these two ECG components changes could improve recognition of concentric hypertrophy.

Detection of the Presence of Left Ventricular Hypertrophy

The regression equation obtained from the mapping studies yielded high overall test accuracy (88% in both categories) for the detection of the LVH, and was equal to or better than the other 12-lead ECG studies. However, because the regression
equation in the present study was developed solely for estimating wall thickness and LV dilatation, this equation may not be the best possible means of detecting the presence or absence of the LVH. To obtain the optimal criteria, further examination with multiple logistic regression analysis or discriminant analysis is required.

It has been reported that difference of the body constitution influences the QRS amplitude. Among the test subjects, the Sokolow-Lyon and the Romhilt-Estes criteria gave more sensitive results than the original study. Differences in body constitution between the Japanese test population described in this paper and test populations in the United States may explain why higher sensitivity in the classic methods occurred.

Clinical Implications

Results from conventional methods were behind those from mapping studies. The major reason for this conclusion was thought to be that relatively little attention was given to VAT prolongation. Presence of VAT prolongation accounts for only one of the 13 points in the Romhilt-Estes point score, and VAT was not evaluated in some voltage criteria (such as the Sokolow-Lyon voltage). VAT prolongation has independent, important information about LVH diagnosis. Increased attention to VAT changes should give more pertinent information concerning diagnosis of the LVH.

Sufficient criteria for diagnosis of LVH severity were obtained with body surface mapping for the evaluation of both QRS voltage increase and VAT prolongation. Mapping studies may be too laborious to be practical for clinical use, but increased knowledge from mapping studies should provide simpler criteria for diagnosis of LVH severity. This technique is becoming increasingly accessible in Japan and in some centers, where it is performed at the same cost as an echocardiogram.

Appendix 1

The equation developed by Casale et al is as follows:

$$\text{Risk} = \frac{1}{1 + e^{-\text{exponent}}}$$

For patients in normal sinus rhythm

$$\text{Exponent} = 4.558 - 0.092 \cdot (\text{RaVL} + \text{SV}_3) - 0.306 \cdot \text{TV}_1 - 0.212 \cdot \text{QRS} - 0.278 \cdot \text{PTFV}_1 - 0.559 \cdot \text{sex}$$

For patients in atrial fibrillation

$$\text{Exponent} = 5.045 - 0.093 \cdot (\text{RaVL} + \text{SV}_3) - 0.312 \cdot \text{TV}_1 - 0.325 \cdot \text{QRS} - 0.602 \cdot \text{sex}$$

Partition value of exponent for detection of LVH in sinus rhythm is $\text{LVH} < -1.55$ and in atrial fibrillation is $\text{LVH} < -1.20$, where units of measurement are voltages of RaVL, SV3, and TV1 in millimeters (1 mm = 0.1 mV); QRS duration (sec x 100, hundredths of a second); P terminal force in lead V1 (PTFV) (mm x sec, based on area); and sex entered as 1.0 for men and 2.0 for women.

Appendix 2

The factor analysis was performed on ECG measurements obtained from the test population. The mathematical procedures adopted are as follows:

In general, the model for the ith standardized variable is written as

$$X_i = A_1 \cdot F_1 + A_2 \cdot F_2 + \ldots + A_k \cdot F_k + U_i$$

where the F is the common factor, U is the unique factor, and the A is the constant used to combine the kth factors. The unique factors are assumed to be uncorrelated with each other and with the common factors.

First, the correlation matrix for all variables is computed. Second, a factor extraction is performed. In this step, the number of factors neces-
sary to present the data is determined. We used principal component analysis for factor extraction (factors with a variance less than 1 are excluded because these factors are no better than a single variable). In the next step, the initial matrix is transformed into one that is easier to interpret. A variety of algorithm is used for the rotation (transformation) to obtain a simple structure. We used the varimax method, the most commonly used orthogonal rotation method. Finally, to interpret the factors, factor loading for the community data and correlation between the factors and variables are calculated. Because we used the orthogonal rotation method, these two scores were equal. Scores are shown in Table 4 as the factor matrix.

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