Body Surface Distribution of Abnormally Low QRST Areas in Patients With Wolff-Parkinson-White Syndrome

Evidence for Continuation of Repolarization Abnormalities Before and After Catheter Ablation

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Background. Whether the Wolff-Parkinson-White syndrome (WPW) is associated with repolarization abnormalities is controversial. The QRST isointegral map (I-map) is theoretically independent of the activation sequence and dependent on repolarization properties. There have been no reports concerning the effects of radiofrequency (RF) catheter ablation of accessory pathway (AP) on repolarization properties analyzed by I-mapping.

Methods and Results. I-maps were constructed from data recorded in 38 patients with WPW to investigate repolarization properties and their body surface distribution in a physiological state, without pharmacological influences, and in 13 ablated patients to elucidate the effects of RF ablation on repolarization properties. Patients were divided into three groups: group A, 15 patients with type A WPW (left-sided AP); group B, 10 patients with type B (right-sided AP); and group C, 13 patients who were successfully ablated. Group C consisted of three subgroups: subgroup CA, 7 patients with type A WPW; subgroup CB, 3 patients with type B WPW; and subgroup CC, 3 patients with concealed WPW. Controls consisted of 608 normals. Although I-maps of WPW were highly ($r=.87$) correlated with the mean normal I-map, the location of the minimum in groups A and B differed significantly from that in normals. The minimum was located over the upper right anterior chest in normal subjects, and the back in 82% of 22 patients with type A WPW (including ablated patients [groups A+CA]), and over the mid to lower right anterior chest in 62% of 13 patients with type B WPW including ablated patients [groups B+CB]). Groups A+CA and B+CB had an abnormally low QRST area (“−2SD area”) over the back and right anterior chest, respectively. The abnormally located minimum and the “−2SD area” were present in 7 of 10 ablated patients with manifest WPW [groups CA+CB]. After RF ablation, the distribution of the minimum, initially abnormal, became normal over a period of days or weeks, and the “−2SD area” disappeared over 1 week in all 7 patients. Correlation coefficients between I-maps and the mean normal I-map increased after RF ablation.

Conclusions. (1) WPW is often associated with abnormalities in repolarization properties. (2) Repolarization abnormalities were located over the back in type A WPW and over the right mid to lower chest in type B WPW. (3) The abnormalities remain immediately after RF ablation and gradually normalize. These findings support the concept that ST-T abnormalities in 12-lead ECGs following RF ablation are attributable to “cardiac memory.” (Circulation. 1993;88:2674-2684.)

Key Words • radiofrequency • catheters • ablation • QRST • Wolff-Parkinson-White syndrome • mapping • repolarization

Catheter ablation of the accessory pathway by radiofrequency (RF) current has proven to be an effective strategy for treating patients with the Wolff-Parkinson-White syndrome (WPW).1-4 It was recently demonstrated that marked ST-T abnormalities in 12-lead ECGs often appear after RF ablation and that such ST-T abnormalities gradually normalize over weeks.2-4 Rosenbaum et al5 reported that the ST-T changes induced by right ventricular pacing persist long after the discontinuance of the right ventricular pacing. Those authors introduced the concept of “cardiac memory,” ie, repolarization properties under a certain activation sequence gradually change when the heart is driven with a different activation sequence. It has been speculated that the ST-T abnormalities after ablation are attributable to cardiac memory but not to the...
myocardial damage produced by either ischemia or ablation procedures. However, there have been few reports concerning repolarization abnormalities in patients with the WPW syndrome. Nicolai et al. showed that the disappearance of delta waves induced by anti-arrhythmic agents such as procaïnamide and ajmaline chloride disclosed the presence of ST-T abnormalities in patients with the manifest WPW syndrome. However, the effects of drugs on repolarization properties could not be completely ruled out. Furthermore, no reports have directly demonstrated repolarization abnormalities in patients with manifest WPW syndrome in an antiarrhythmic drug-free state before RF ablation and the continuity of the repolarization abnormalities before and after RF ablation. The QRS integral map (I-map) has been reported to be theoretically independent of the activation sequence and dependent on repolarization properties. The I-map has been used to assess repolarization abnormalities in various pathological states. The purposes of this study were (1) to elucidate whether the manifest WPW syndrome is associated with abnormalities in repolarization properties, and what part of the body surface, if any, reflects the abnormalities; (2) to evaluate the effects of RF ablation on repolarization properties; and (3) to investigate, using body surface I-maps, whether the ST-T abnormalities following ablation are a continuation of the repolarization abnormalities present before ablation.

Methods

Study Population

We selected for study 38 patients (25 men and 13 women; mean age, 41.7 years; age range, 15 to 72 years) whose body surface maps were recorded at the Nagoya University Hospital between January 1988 and November 1992 and at the Nagoya Daini Red Cross Hospital between March 1992 and February 1993. Participants were required to satisfy the following criteria: manifest WPW syndrome confirmed by 12-lead ECGs or concealed WPW syndrome confirmed by electrophysiological study; no antiarrhythmic drug administered or the drug had been discontinued for at least five elimination half-lives at the time of mapping; absence of other cardiac disease such as congenital heart disease, myocardial infarction, or valvular heart disease; no hypertension; heart rate between 50 to 100 beats per minute; and no imbalance of serum electrolytes.

Patients were divided into three groups: group A (Rosenbaum type A: 11 men and 4 women; mean age, 44.2 years; age range, 15 to 72 years), group B (Rosenbaum type B: 6 men and 4 women; mean age, 33.6 years; age range, 15 to 65 years), and group C (patients successfully ablated: 8 men and 5 women; mean age, 45.1 years; age range, 17 to 65 years). Subjects in group C were subdivided into subgroup C1 (Rosenbaum type A: 3 men and 4 women) and C2 (Rosenbaum type B: 3 men) and C3 (concealed WPW: 2 men and 1 woman).

Control subjects consisted of 608 normal individuals (376 men and 232 women; mean age, 42.3 years; age range, 17 to 81 years) who were registered in the Japanese Circulation Society Task Force Committee on Body Surface Mapping. They were free of cardiovascular disease as determined by routine clinical examination including chest radiographs and ECGs.

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

Body Surface QRS T-Maps

Recording and data analysis. Body surface ECGs were recorded to construct body surface I-maps using a VCM-3000 (Chunichi Denshi Company Ltd, Nagoya, Japan). Because the details of data acquisition and processing have been reported elsewhere, we describe them only briefly. Unipolar ECGs 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 ECGs and the Frank X, Y, and Z ECGs were also recorded simultaneously. These ECG data were scanned by multiplexers, digitized by AD converters at a rate of 1000 samples per second, and stored on floppy disks. Two-point baseline adjustment was performed by choosing the flat portion of the TP segment before P and after T deflection of the selected QRS complex. After baseline adjustment, a root-mean-square (rms) voltage-versus-time curve based on the 87 leads was plotted to help identify the beginning of QRS and the end of T deflection that were manually selected from this curve. The QRS deflection area was calculated by integrating each lead over the appropriate interval and expressed in millivolts × millisecond (mV · ms). QRS integral contours were separated by 20 mV · ms. The maximum and minimum were indicated by + and − signs, respectively. QRS duration was defined as the time from the beginning to the end of QRS deflection selected manually from the rms voltage-versus-time curve based on 87 leads. Data sampling was performed at the resting expiratory level with the subject in the supine position.

QRS T-departure maps. The mean and SD values of the normal QRS at each lead point were calculated from data collected in 608 normal subjects. To estimate the deviation of patient data from the normal value, the departure index (DI) at each lead was calculated with the VCM-3000 as follows: DI=(X−mean)/SD, where X represents the QRS at the corresponding lead for each patient. Because we were interested in the decrease of QRS, areas in which the DI values were less than −2 on the departure map were designated as “−2SD areas.” The following parameters were derived from the “−2SD area”: nQRS, number of lead points in “−2SD area”; and ΣQRS, sum of the value obtained by subtracting the QRS value of a given patient at each point in “−2SD area” from the normal mean−2SD QRS value.

RF Catheter Ablation

RF catheter ablation was successfully performed in 13 patients at the Daini Red Cross Hospital. All antiarrhythmic drugs had been discontinued for at least five elimination half-lives before a map was recorded in these patients. In all 13 patients, maps were recorded 2 to 24 hours before ablation and again at 1 to 2 hours, 1 day, and 1 week after ablation. In 5 of the 13 patients, additional maps were recorded at 4 weeks and again at 3 months after ablation. Ablation procedures were defined as successful if the antegrade and retrograde accessory atrioventricular conductions were completely abolished. The creatine kinase–MB fraction was measured every 4 hours for 24 hours after the procedure. No
patient had an abnormal elevation of creatine kinase-MB fraction after the ablation.

Statistical Analysis
Values are expressed as mean±SD. Statistical analysis was performed using one-way ANOVA (when a significant effect was observed, group comparisons were made using Scheffé's test) and paired t test, and simple correlations were calculated according to standard statistical methods. A value of $P<.05$ was considered statistically significant.

Results
Mean I-Map in Normal Subjects
As shown in Fig 1, the positive area of the mean I-map of normal subjects was located over the left chest with the maximum at the site of V₄ of the standard 12-lead ECGs. The negative area was over the upper chest with the minimum at the upper edge of the frame.

I-Maps in Patients With Type A WPW Syndrome
In the I-map of a representative patient (58-year-old man) in group A (Fig 2B), there was a positive area over the left chest with the maximum over the left mid anterior chest. The negative area was over the back with the minimum over the back. The locations of the negative area and the minimum differed from the distribution in the mean I-map of normal subjects. The minimum was present over the back in 18 (82%) of the 22 patients in groups A+Cₐ (patients with type A WPW including ablated patients). There was a −2SD area over the back in the I-departure map (Fig 2C). A −2SD area was present in the I-departure map of 13 (59%) of the 22 patients in groups A+Cₐ. QRS duration in this patient

![Image of front and back](image-url)
was 120 milliseconds. The ΣQRST and nQRST in this patient were 190 mV · ms and 6, respectively.

I-Maps in Patients With Type B WPW Syndrome

In the I-map of a representative patient (21-year-old woman) in group B (Fig 3B), there was a positive area over the left chest with the maximum over the left mid anterior chest. The negative area was over the right chest with the minimum over the right lower chest. The locations of the negative area and the minimum differed from the distribution in the mean I-map of normal subjects. The minimum was present over the mid chest in 8 of the 13 patients in groups B+Cb (patients with type B WPW including ablated patients). There was a −2SD area over the right lower chest in the I-departure map (Fig 3C). A −2SD area was present in the I-departure maps of 11 of the 13 patients in groups B+Cb. QRS duration in this patient was 138 milliseconds. ΣQRST and nQRST in this patient were 1620 mV · ms and 30, respectively.

Location of Minimum in Normal Subjects, Types A and B WPW Syndrome

The mean location of the minimum was over the right upper chest in normal subjects (Fig 1). The mean location of the minimum in groups A+Cb (22 patients with type A WPW including ablated patients) and B+Cb (13 patients with type B WPW including ablated patients) was over the back and right mid chest, respectively (Fig 4). The mean location of the minimum in groups A+Cb differed significantly (P<.001) from that in normal subjects horizontally, whereas the mean location of the minimum in groups B+Cb differed significantly (P<.001) from that in normal subjects vertically. The mean location of the minimum in groups A+Cb differed significantly from that in groups B+Cb horizontally (P<.001) and vertically (P<.01).

Correlation Between the Mean I-Map of Normal Subjects Versus the I-Maps of Patients

Although the I-maps of patients with the manifest WPW syndrome differed from the mean normal I-map in the location of the minimum and the distribution of part of the negative area, the I-map of each patient was highly correlated with the mean I-map of normal subjects (Table 1). In the successfully ablated patients, the correlation between the I-map of the patient and the mean normal I-map significantly increased 1 week after ablation compared with that before ablation, whereas the correlation showed no significant change 1 day after ablation compared with that before ablation (Table 2).
Fig 4. Mean location of minimum in normal subjects and patients with Wolff-Parkinson-White syndrome. Closed circle, open circle, and open square indicate mean location of minimum in normal subjects (n=608), groups A+C_A (n=22), and groups B+C_B (n=13), respectively. Dots of V_I and V_2 indicate lead points of V_I and V_2 in 12-lead ECGs.

Relationship Between QRS Duration and Parameters of nQRST and ΣQRST

The mean QRS duration calculated from the rrms voltage-versus-time curve of 87 lead points was significantly (P<.05) greater in groups B+C_B (13 patients with type B WPW including ablated patients) than in groups A+C_A (22 patients with type A WPW including ablated patients) (Table 3). The mean QRS duration in patients with the "-2SD area" was significantly (P<.01) greater than in patients without it (Table 3). The ΣQRST and nQRST were significantly (P<.01) greater in groups B+C_B than in groups A+C_A (Table 3).

I-Map and I-Departure Map Before and After RF Catheter Ablation

Fig 5 shows 12-lead ECGs, the I-map, and the I-departure map of a representative patient (43-year-old man) from group C_B before RF catheter ablation, in whom the ablation of the right posterior accessory pathway proved successful. In 12-lead ECGs, there were negative delta waves and QS deflections in leads V_I and V_2 (Fig 5A). The I-map (Fig 5B) showed a positive area over the left chest with the maximum over the left mid anterior chest. The negative area was over the right chest with the minimum over the right mid chest. The location of the negative area and of the minimum differed from the distribution in the mean I-map of normal subjects. There was a "-2SD area" over the right lower chest in the I-departure map (Fig 5C).

Table 1. Correlation Between QRS Isointegral Maps of Each Group and Mean Isointegral Map of Normal Subjects

<table>
<thead>
<tr>
<th>Group</th>
<th>Correlation Coefficient</th>
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<tbody>
<tr>
<td>A (n=15)</td>
<td>.89±.08</td>
</tr>
<tr>
<td>B (n=10)</td>
<td>.85±.12</td>
</tr>
<tr>
<td>C (n=13)</td>
<td>.86±.07</td>
</tr>
<tr>
<td>A+C_A (n=22)</td>
<td>.88±.07</td>
</tr>
<tr>
<td>B+C_B (n=13)</td>
<td>.84±.11</td>
</tr>
<tr>
<td>Total (n=38)</td>
<td>.87±.06</td>
</tr>
</tbody>
</table>

Groups A+C_A, patients with type A Wolff-Parkinson-White syndrome including ablated patients.
Groups B+C_B, patients with type B Wolff-Parkinson-White syndrome including ablated patients.

Table 2. Correlation Between QRS Isointegral Maps of Ablated Patients and Mean Isointegral Map of Normal Subjects

<table>
<thead>
<tr>
<th>Group</th>
<th>Before Ablation</th>
<th>1 Day After Ablation</th>
<th>1 Week After Ablation</th>
</tr>
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<tbody>
<tr>
<td>C_A</td>
<td>.86±.07</td>
<td>.86±.07</td>
<td>.90±.09</td>
</tr>
<tr>
<td>C_B</td>
<td>.83±.09</td>
<td>.85±.09</td>
<td>.93±.03</td>
</tr>
<tr>
<td>Subtotal</td>
<td>(n=10) .85±.07</td>
<td>.86±.07</td>
<td>.91±.07*</td>
</tr>
<tr>
<td>C_C</td>
<td>.90±.05</td>
<td>.92±.06</td>
<td>.94±.01</td>
</tr>
<tr>
<td>Total</td>
<td>(n=13) .86±.07</td>
<td>.88±.07</td>
<td>.92±.07*</td>
</tr>
</tbody>
</table>

*P<.05 vs before ablation.

ΣQRST and nQRST were 1003 mV·ms and 25, respectively. Fig 6 shows 12-lead ECGs, the I-map, and the I-departure map of the same patient 1 day after ablation. In 12-lead ECGs (Fig 6A), the delta waves disappeared, and there were narrower and normally directed QRS deflections with negative T waves in leads II, III, and aVF. Although the configuration of the 12-lead ECGs after ablation differed from those before ablation (Figs 5A and 6A), the I-map (Fig 6B) and the I-departure map (Fig 6C) were similar to those before ablation (Figs 5B and 5C) as to the location of the minimum and the distribution of the negative area and "-2SD area." ΣQRST and nQRST were 997 mV·ms and 25, respectively. Fig 7 shows 12-lead ECGs, the I-map, and the I-departure map 1 week after ablation. In 12-lead ECGs (Fig 7A), T waves in leads II, III, and aVF were shallower compared with those 1 day after ablation. In the I-map, the minimum moved upward to the normal position and the distribution of the negative area resembled that in the mean I-map of normal subjects (Fig 7B). In the I-departure map, "-2SD area" disappeared (Fig 7C). Fig 12 shows 12-lead ECGs, the I-map, and the I-departure map 3 months after ablation. In 12-lead ECGs (Fig 8A), there were negative T waves in inferior leads. In the I-map, the minimum was located at the normal position, and the distribution of the negative area was similar to that in the mean I-map of the normal subjects (Fig 8B). In I-departure map, "-2SD area" was absent (Fig 8C). The correlation between the I-map of this patient and the mean I-map of the normal subjects was 0.72 before ablation, 0.76 1 day after ablation, 0.92 1 week after ablation, and 0.99 3 months after ablation.

Fig 9 shows the I-map and I-departure map of a representative patient (37-year-old woman) from group C_B (Figs 9A and 9B), 1 day (Figs 9C and 9D), 1 week (Fig 9E), and 1 month (Fig 9F), respectively, after RF catheter ablation, in whom the ablation of the left lateral accessory pathway proved successful. In the I-map before ablation (Fig 9A), there was a positive area over the left chest with the maximum over the left mid inferior chest. The negative area was over the upper back with the minimum over the left upper back. The locations of the negative area and the minimum differed from the distribution in the mean I-map of normal subjects. There was a "-2SD area" over the back in the I-departure map (Fig 9B). The I-map and I-departure map 1 day after ablation (Figs 9C and 9D) were similar to those before ablation (Figs 9A and 9B) as to the location of the minimum and the distribution of the negative area and "-2SD area." Although the
Table 3. QRS Duration, nQRS, and ΣQRST in Groups A+CA and Groups B+CB and QRS Duration in Patients With Manifest Wolff-Parkinson-White Syndrome With and Without "−2SD Area"

<table>
<thead>
<tr>
<th>Group</th>
<th>QRS Duration, ms</th>
<th>nQRS</th>
<th>ΣQRST, mV·ms</th>
</tr>
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<tbody>
<tr>
<td>A+CA</td>
<td>118.5±16.9</td>
<td>6.9±8.5</td>
<td>246.6±332.6</td>
</tr>
<tr>
<td>B+CB</td>
<td>131.7±14.8</td>
<td>17.5±13.2</td>
<td>726.3±647.3</td>
</tr>
<tr>
<td>&quot;−2SD area&quot; (+) (n=24)</td>
<td>130.8±15.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&quot;−2SD area&quot; (−) (n=11)</td>
<td>107.3±6.3</td>
<td></td>
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</tr>
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</table>

*P<.05, †P<.01.
Groups A+CA, 22 patients with type A Wolff-Parkinson-White syndrome including ablated patients.
Groups B+CB, 13 patients with type B Wolff-Parkinson-White syndrome including ablated patients.

Minimum remained on the back in I-map 1 week after ablation (Fig 9E), the "−2SD area" disappeared from the I-departure map. In the I-map 1 month after ablation (Fig 9F), the minimum was located at the normal position, and the distribution of the negative area was similar to that in the mean I-map of the normal subjects. The correlation between the I-map of this patient and the mean I-map of the normal subjects was 0.76 before ablation, 0.79 1 day after ablation, 0.79 1 week after ablation, and 0.98 1 month after ablation.

In all three patients with concealed WPW syndrome and successful ablation, the minimum was located over the normal right upper anterior chest, and no "−2SD area" was present either before or after ablation. The correlation between the I-map of the three patients and the mean I-map of the normal subjects was more than 0.90 before and after ablation (Table 2).

Discussion

We observed that patients with the manifest WPW syndrome often exhibit abnormalities in the I-map that indicate repolarization abnormalities, although the I-maps of these patients were closely correlated with the mean I-map of normal subjects. The distribution of the abnormalities in the I-map depended on the location of the accessory atrioventricular connection. The ST-T abnor-

![Fig 5. Twelve-lead ECGs (A), QRS isointegral map (B), and departure map (C) of a successfully ablated patient with type B Wolff-Parkinson-White syndrome before ablation. Shading in departure map indicates "−2SD area." Closed circles indicate lead points of 12-lead ECGs.](image-url)
malities present immediately after the ablation were a continuation of the repolarization abnormalities present before ablation. After RF ablation, the abnormality gradually normalized over days or weeks.

RF ablation of the accessory atrioventricular connection is reportedly one of the most effective approaches to managing arrhythmias due to WPW syndrome.\(^1\)\(^-\)\(^4\) ST-T abnormalities in 12-lead ECGs have been demonstrated immediately after successful ablation in patients with the manifest WPW syndrome but not in patients with the concealed WPW syndrome, regardless of energy sources such as RF and direct current.\(^2\)\(^-\)\(^4\) Kalbfleisch et al\(^2\) evaluated the serial changes in T waves in patients who had undergone successful RF ablation of accessory atrioventricular connection. They found that postablation ST-T abnormalities occurred in 22 of the 26 patients with the manifest WPW syndrome but in none of the 16 patients with the concealed WPW syndrome, that the ST-T abnormalities were transient and disappeared in 3 months in nearly all patients, and that the transient repolarization abnormalities depended on the direction of the delta wave and the degree of the baseline preexcitation. Wood et al\(^3\) reported that postablation T wave abnormalities were present in 7 patients with the manifest WPW syndrome due to right-sided accessory atrioventricular connection but not in 12 patients with left-sided accessory atrioventricular connection. They showed that the repolarization abnormalities following ablation were similar to T wave abnormalities in the absence of preexcitation during atrioventricular reciprocating tachycardia or rapid atrial pacing before ablation.

It has been speculated that the ST-T abnormalities may be attributable to the continuation of the repolarization abnormalities present before ablation or "cardiac memory" and not to either ischemia or the procedure.\(^2\)\(^-\)\(^4\) However, because of difficulties in proving the repolarization abnormalities in the presence of abnormal activation sequence, there have been few reports concerning repolarization abnormalities during preexcitation in patients with the manifest WPW syndrome at normal heart rate. Nicolai et al\(^6\) reviewed the data on 45 patients with the manifest WPW syndrome and found ST-T abnormalities during normal pathway conduction induced by the administration of antiarrhythmic drugs such as procainamide and ajmaline chloride or the eyeball pressure maneuver in 39 (87%) patients with the manifest WPW syndrome. However, effects related to pharmacologic or autonomic nervous effects on repolarization properties could not be completely ruled out. Wood et al\(^3\) demonstrated repolarization abnormalities during normal pathway conduction before ablation only.
at an unusually high heart rate during atrioventricular reciprocating tachycardia or during rapid atrial pacing in patients with the manifest WPW syndrome. No reports have directly demonstrated repolarization abnormalities during preexcitation in a physiological state without pharmacologic influences before RF ablation and the continuation of the repolarization abnormalities before and after RF ablation in patients with manifest WPW syndrome. In the present study, we showed that patients with manifest WPW syndrome often show associated repolarization abnormalities indicated by the abnormal location of the minimum and the presence of the "−2SD area" in the I-map recorded during preexcitation in a physiological state without pharmacologic influence. The I-maps obtained before and immediately after the ablation were similar, suggesting that the repolarization properties before ablation continued after it.

Greater repolarization abnormalities after ablation have been reported in patients with a greater degree of preexcitation before ablation.2,3 Kalbfleisch et al2 demonstrated ST-T abnormalities after ablation in patients with either a right-sided or a left-sided accessory atrioventricular connection. However, Wood et al3 found repolarization abnormalities only in those patients with a right-sided accessory atrioventricular connection. Wood et al3 thought this might be explained by lesser degrees of preexcitation due to a later activation of the left-sided accessory connections compared with that of right-sided ones.2,3 In the present study, the mean QRS duration in patients with type B WPW syndrome was significantly greater (P<.01) than that in type A WPW syndrome. The mean nQRST and ΣQRST in type B WPW syndrome were significantly greater (P<.01) compared with those in type A WPW syndrome, suggesting that greater repolarization abnormalities tend to occur in patients with right-sided accessory connections than in those with left-sided accessory connections. The mean QRS duration in patients with the "−2SD area" was significantly greater (P<.001) than that in patients without that finding. This observation agreed with previous reports showing that greater repolarization abnormalities after ablation occurred in patients with a greater degree of preexcitation before ablation.2,3 In the present study, we focused on the presence, location, and disappearance of "−2SD area." However, we showed that "+2SD area" usually coexists with "−2SD area" in a reciprocal manner.11 Although changes in location of the positive area and the maximum were smaller compared with those of the negative area and the minimum, changes in "+2SD area" may provide additional useful information on recovery properties.
ST-T abnormalities after intermittent left bundle branch block and right ventricular pacing have been reported to persist, leading to an erroneous diagnosis.\textsuperscript{5,24,25} Rosenbaum et al\textsuperscript{5} demonstrated that the ST-T wave abnormalities induced by intermittent left bundle branch block or right ventricular pacing persisted for days and weeks after the restoration of the normal activation sequence and that the ST-T abnormalities could be inexplicable by secondary ST-T wave changes and attributed to electrotonic modulation. They introduced the concept of “cardiac memory,” i.e., that when a change in the activation sequence provokes ST-T wave changes, such changes gradually increase and reach a plateau only after several days or weeks; their dissipation appears to require much more time. However, while electrotonic modulation of repolarization properties was proposed as one of the mechanisms of “cardiac memory,” the causes of cardiac memory are still controversial. Costard-Jäckle et al\textsuperscript{26} examined how ventricular repolarization was modulated by prolonged changes in activation sequence by assessing the effects of ectopic pacing on the distribution of action potential durations in isolated rabbit hearts. They concluded that the sequence of ventricular activation modulates the sequence of ventricular repolarization by an as-yet-unidentified process with very slow onset and offset characteristics. Recently, del Balzo and Rosen\textsuperscript{27} reported that cardiac memory might be based on a physiological property of the myocardium that is related to specific K\textsuperscript{+} channels. In the present study, we found an abnormal location of the minimum in the I-map and the “–2SD area” during preexcitation before ablation and the similarities in I-maps during preexcitation before and during the normal activation sequence immediately after ablation. The abnormalities in I-maps before and immediately after ablation gradually normalized over the course of 1 week with an increase in the correlation with the mean I-map of normal subjects. Because the I-map abnormalities did not worsen, no abnormal elevations of creatine kinase–MB fraction were observed, and since no left ventricular wall motion abnormalities were detected on echocardiography after the ablation, we attribute the repolarization abnormalities following ablation to “cardiac memory,” not to ischemia or the ablation procedure.

Wilson et al\textsuperscript{28} reported that the QRST deflection area is largely independent of the activation sequence and dependent on repolarization properties, and they proposed the concept of a ventricular gradient. Plonsey\textsuperscript{1} showed that the QRST deflection area is theoretically independent of the activation sequence and dependent on repolarization sequence on condition that repolar-
repolarization properties are not influenced by activation sequences. Based on this concept of the ventricular gradient, Abildskov et al. introduced the I-map. They demonstrated that the I-map is largely, but not exclusively, independent of the activation sequence and useful in detecting repolarization abnormalities, even in the presence of QRS deflection abnormalities. Burgess et al. found that the I-map indicates the extent and severity of repolarization abnormalities relatively independent of the activation sequence. Thus, the I-map has been reported to be useful in investigating recovery abnormalities. However, the I-map is not so conventional clinical use.

Recently, small but statistically significant changes in the QRS deflection area have been reported in altered activation sequence. These changes have been attributed in part to altered repolarization properties due to the electrotonic interaction resulting from alteration of the activation sequence. Costard-Jäckle et al. assessed the effects of ectopic pacing on the distribution of action potential duration by measuring the monophasic action potential of isolated rabbit hearts. They observed an inverse relation between the activation time and the action potential duration. Although the process of slowly modulated repolarization properties were not yet identified, the changes in action potential duration were in accordance with the action potential changes explicable by electrotonic interaction induced by the ectopic pacing. Thus, an altered activation sequence has been reported to produce changes in repolarization properties. Although subtle, the changes in I-maps found after altering the activation sequence were due to a sensitive reflection of the changes in repolarization properties.

In the present study, we observed abnormalities in the I-map recorded before and immediately after ablation. We believe that these abnormalities are attributable to changes in repolarization properties resulting from preexcitation in patients with the manifest WPW syndrome. The abnormal minimum and "+2SD area" located in the area reflecting preexcited site may be explained by the prolonged duration of action potential of the preexcited epicardium where the accessory atrioventricular pathway usually connects. Although a prolongation of the action potential duration in the preexcited epicardium in patients with the WPW syndrome remains valid, its likelihood is supported by the concept of electrotonic interaction and the experimental evidence of Costard-Jäckle et al. Nadeau et al. reported a similarity of the I-maps before and after surgical ablation of the accessory atrioventricular connection, supporting I-map's dependence on repolarization properties and independence of activation sequence. In the present study, we demonstrated abnormalities in the I-map before and immediately after ablation compared with the mean I-map of controls. However, we also showed that the preablation I-maps were highly (r=0.87) correlated with the mean I-maps obtained in normal subjects. This finding was in accordance with the report by Nadeau et al. In addition, we demonstrated an improved correlation between the I-maps of patients and the mean normal I-map after ablation in conjunction with normalization of the I-maps. These findings support the concept of the ventricular gradient—that QRS deflection areas are largely independent of activation sequence and dependent on repolarization properties—as long as we admit that the subtle modulation of repolarization properties results from an altered activation sequence.

**Study Limitations**

There are some limitations in the present study. Although we showed that abnormalities in the I-map indi-
cate the presence of repolarization abnormalities, we did not measure actual action potentials. However, it is not possible to record action potentials in the epicardium by catheter in closed-chest patients. It is also difficult to record action potentials from the epicardium repeatedly before and after ablation. Further studies in a larger number of patients and direct evidence of changes in action potential, such as serial changes in monophasic action potential, are needed. Several published studies support the concept of cardiac memory,2-4,26 the mechanism of which remains to be verified.

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