The role of initial minimum potentials on body surface maps in predicting the site of accessory pathways in patients with Wolff-Parkinson-White syndrome

SHIRO KAMAKURA, M.D., KATSURO SHIMOMURA, M.D., TOHRU OHE, M.D., MOKUO MATSUHISA, M.D., AND HIDEAKI TOYOSIMA, M.D.

ABSTRACT Forty-one patients (23 men and 18 women, ages 20 to 66 years) with Wolff-Parkinson-White syndrome were studied with isopotential body surface maps during sinus rhythm to find the most reliable index for predicting the sites of single accessory pathways. The sites predicted by surface maps were compared with those confirmed by multicatheter electrophysiologic study or in the course of surgical operation. Location of the initial minimum by a time criterion, 40 msec after onset of the QRS complex, was not reliable enough for prediction in patients with the small delta wave on their electrocardiograms, because ventricular activation via the normal conduction pathway significantly influenced the location of the minimum. Location of the minimum by an amplitude criterion, −0.15 mV or slightly deeper, was influenced minimally by fusion of ventricular activation, the patient’s body size, or age and corresponded well to the site of the accessory pathway in 36 of 41 patients. Those minima appeared on circumscribed areas of the map in accordance with the anatomic subdivisions of the atrioventricular ring. Thus location of the minimum by the amplitude criterion was an excellent index for predicting the site of the accessory pathway, regardless of the degree of ventricular fusion. These amplitude-based map features suggest that nonstandard electrocardiograms recorded from selected positions on the body surface can be used as accurate predictors of the sites of accessory pathways.


BODY SURFACE MAPPING has recently come into use as a noninvasive diagnostic procedure for locating the site of the accessory pathway in patients with Wolff-Parkinson-White (WPW) syndrome.1-4 De Ambroggi et al.2 and Iwa et al.3 reported the location of maxima and minima during the first 20 to 40 msec of the QRS complex to be a useful index. Significant fusion of the ventricular activation fronts, both from the normal conduction system and the accessory pathway, is known to affect potential distribution during the QRS complex and to complicate the interpretation of location of the pathway.4-6 Benson et al.4 concluded that the potential distribution at 40 msec of the QRS complex and that of the ST segment could be excellent indexes of the site of pathway only in patients with marked preexcitation during sinus rhythm or in patients with preexcitation maximized by atrial pacing. According to their time-based analytic method, the site could not be predicted accurately without the aid of invasive techniques in many patients with WPW syndrome with the small delta wave.

We studied the relationship between the potential distribution on the early QRS surface maps and sites of pathways confirmed at surgical operations or by electrophysiologic studies. This report demonstrates (1) the value of amplitude-based map analysis over the time-based analysis in predicting the site of single accessory pathways, independently of the presence of ventricular fusion, and (2) the exact locations of the initial minima on a clearly defined electrode grid, which may improve the diagnostic utility of standard or nonstandard electrocardiograms (ECGs).
Materials and methods

We obtained standard 12-lead ECGs, body surface maps, and electrophysiologic recordings from 41 patients with WPW syndrome (23 men and 18 women, 20 to 66 years). All had delta waves on their ECGs, and there was no associated cardiac disease except Ebstein's anomaly in two patients. Patients with short P-delta intervals of less than 0.06 sec were excluded from this study because the potential caused by atrial activation might have significantly influenced the low-level potential distribution of early ventricular activation.

Body surface mapping. This procedure was performed with a Heart Potential Mapper (Model 5100A, Chunichi Denshi Ko-gyosho Co., Ltd.). The method used for recording ECGs and constructing maps has been described in detail. Briefly, the apparatus was equipped with 96 buffer AC amplifiers, sample and hold circuits, analog-to-digital converters, multiplexers, and a microcomputer. The AC amplifier had a frequency response between 0.03 and 860 Hz and a gain of 60 dB. The maximal sampling rate was 1 kHz. In this study, all the electrocardiographic data for mapping were sampled simultaneously at intervals of 2 or 4 msec during sinus rhythm. Electrodes for mapping were placed at 87 points (59 anterior, 28 posterior) on the chest surface (figure 1). For electrode positioning, the second row from the top (row 6) in the maps was set at the level of the second intercostal space on the midternal line, and the middle row (row 4) was set at the level of the fifth intercostal space on the midclavicular line. Electrode columns A and I correspond to the right and left midaxillary lines, respectively; the left half of the map shows the anterior chest surface and the right half the posterior surface. All ECG waveforms of 87 points and isopotential maps were displayed or printed out after the data were processed.

The onset of the QRS segment corresponds to the time at which the successively increasing potential of a maximum or minimum exceeded ± 0.1 mV; the end of the QRS complex or onset of the ST segment corresponds to the time at which the distribution of maximum and minimum inverted after successive decreases in potential. Patients were separated into two groups in relation to QRS duration at the time of recording. Patients with a QRS duration of more than 120 msec were included in the large delta group (group 1); those with a QRS duration of less than 120 msec were included in the small delta group (group 2). Isopotential lines were drawn at intervals of 0.20 or 0.40 mV.

Electrophysiologic study. Three bipolar electrode catheters with an interpolar distance of 10 mm were inserted into the femoral veins and positioned against the right atrium, right ventricle, and across the tricuspid valve. An additional quadri-
ed by preoperative electrophysiologic studies were consistent with those confirmed by operative mapping studies. The number of patients for each site was as follows: left lateral, five; left posterior, one; posterior septum, two; right anterior, two; right lateral, two; right posterior, three.

**QRS maps: group 1 vs group 2.** QRS duration was more than 120 msec in 23 patients (group 1, large delta) and less than 120 msec in 18 patients (group 2, small delta). Eight of 24 patients with a left-sided pathway, three of five with a posterior septal pathway, and all 12 with a right-sided pathway had the large delta wave.

The maps of each patient were carefully evaluated in relation to QRS duration to confirm that the minimum showed a characteristic movement during early QRS. That is, the minimum tended to move around while at a low level potential of less than \(-0.10\) mV; however, it intensified rapidly and became stable in all patients when the potential exceeded \(-0.15\) mV before 40 msec of the QRS complex. The mean time for attaining \(-0.15\) mV was 19.4 \(\pm\) 10.4 msec. This prominent minimum was stationary at 40 msec of the QRS complex in 22 of 23 group 1 patients (95.7%) and in 13 of 18 group 2 patients (72.2%). In five group 2 patients, the minimum shifted to other sites before 40 msec. The stationary duration of the minima after the onset of the QRS complex was 87.8 \(\pm\) 31.6 msec in group 1 and 50.9 \(\pm\) 16.7 msec in group 2 (p < .01) (table 1).

**Figure 3** illustrates map sequences during the QRS complex in two patients with a left lateral accessory pathway. QRS duration was 148 msec in the patient whose data are shown in panel A and 80 msec in the patient whose data are shown in panel B. In panel A, one of the minima was stable at the upper middle back for more than 80 msec after the onset of QRS. In panel B, the minimum that remained at the middle back at onset (figure 3, B, 1) and 8 msec of QRS (figure 3, B, 2) shifted to the upper back area at 40 msec of QRS because of the influence of an extending negative potential produced by ventricular activation, via a normal conduction pathway (figure 3, B, 3). As shown in these cases, the locations of minima at 40 msec differed even in patients with the same preexcitation site, but the locations of minima with voltages between \(-0.15\) mV and \(-0.20\) mV were the same for both. Accordingly, we adopted the amplitude criterion (\(-0.15\) mV potential) instead of the criterion of time after onset of ventricular activation as an index for predicting the site of the accessory pathway because the minimum intensified rapidly and became stable when its potential was deeper than \(-0.15\) mV and

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**Table 1**

**Surface mapping data**

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of patients</th>
<th>Age (yr) ± SD</th>
<th>QRS duration (msec)</th>
<th>Time of minimum potential attaining (-0.15) mV at early QRS (msec)</th>
<th>No. of patients with stationary minimum at 40 msec of QRS</th>
<th>Stationary duration of minimum (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Large delta</td>
<td>23</td>
<td>42.5 ± 13.7</td>
<td>137.0 ± 14.5</td>
<td>21.6 ± 12.7</td>
<td>22 (95.7%)</td>
<td>87.8 ± 31.6</td>
</tr>
<tr>
<td>Small delta</td>
<td>18</td>
<td>44.8 ± 12.8</td>
<td>94.4 ± 11.3</td>
<td>16.6 ± 5.4</td>
<td>13 (72.2%)</td>
<td>50.9 ± 16.7</td>
</tr>
<tr>
<td>Total</td>
<td>41</td>
<td>43.5 ± 13.2</td>
<td>118.3 ± 25.1</td>
<td>19.4 ± 10.4</td>
<td>35 (85.4%)</td>
<td>71.6 ± 31.8</td>
</tr>
</tbody>
</table>

*Plus or minus values are mean ± SD.*

*a*Length of time that the minimum in the circumscribed area according to anatomic subdivision of the atrioventricular ring after onset of QRS.

*bp < .01 group 1 vs group 2.*
never moved to other areas until attaining $-0.20 \text{ mV}$, even in the patient with the smallest delta wave. In this patient the area where the minimum was initially located kept negative potential until the end of the QRS complex.

**Value of amplitude-based maps.** Figure 4 is a map illustrating the location of minima with a $-0.15 \text{ mV}$ or slightly deeper potential in all patients. These minima were located on the upper back in four patients with a left anterior pathway and in two patients with a left lateral pathway, on the middle back in 13 patients with a left lateral pathway, on the lower back in five patients with a left posterior pathway and in one patient with a posterior septal pathway, and on the right lower lateral chest in three patients with a posterior septal pathway. In contrast, minima were located on the upper anterior chest in five patients with a right anterior pathway, on the middle anterior chest in four patients with a right lateral pathway and in one patient with a right posterior pathway, and on the right lower anterior chest in two patients with a right posterior pathway and in one patient with a posterior septal pathway. In five patients indicated by asterisks, the minimum appeared where the minima due to preexcitation through a pathway of the adjacent site were expected to appear. Thus the location of minima with a $-0.15 \text{ mV}$ or slightly deeper potential corresponded well with the site of the accessory pathway as classified into seven anatomic sections, with some overlap. The influence of the patient’s body size, age, or sex was considered to be slight.

On the basis of these results, we constructed a diagram showing the approximate relationship between the site of the accessory pathway and the area on the body where the resulting minimum is expected to appear (figure 5). Table 2 shows the assignment of electrode positions for predicting the site of the accessory pathway. Figure 5 and table 2 indicate that the minima appear on a limited body surface zone, comprising a band running obliquely from the upper anterior chest...
The preexcitation shown we patients, than to down of 23 of means we tried patients in the duration was misjudged was complex was evaluated for the index exceeded for the area of the incision and the site of preexcitation shown in table 2, the accessory pathways were misjudged in 11 of 41 patients. Because QRS duration was less than 120 msec in seven of these 11 patients, we could not correctly predict the sites in seven of 18 group 2 patients (38.9%) in contrast to four of 23 group 1 patients (17.4%).

The location of the maximum was not a reliable index for predicting the site of an accessory pathway, either at 40 msec or when the minimum potential exceeded -0.15 mV.

ST segment maps. The usefulness of ST segment maps was also evaluated by comparing locations of maxima and minima at 40 msec after the junction of the ST segment with sites of accessory pathways in group 1. We could find no consistent relationship between them when ST segment maps were used independently of QRS maps. Significant variability was noticed, not only in the location of minima and maxima but also in the distribution of low-level potentials. These results are contrary to those of Benson et al.

Correlation of the ECG with the accessory pathway site. The ability of the standard 12-lead ECG in localizing the site of the accessory pathway was studied. The site predicted by the classification of Gallagher et al. based on delta wave polarity was compared with those confirmed by electrophysiologic or operative mapping studies. They were consistent in only 27 of 41 patients (66%). Standard ECGs had obvious limitations in predicting the sites of accessory pathways in group 2 patients (9/18, 50%) and in patients with a left-sided pathway (54%), in contrast to their accuracy in group 1 patients (83%) and in patients with a right-sided path-
way (92%). Another classification or method with standard or nonstandard ECGs was required for more accurate localization, particularly in group 2 patients.

The exact location of the minimum on the clearly defined electrode grid (Table 2) might be of value in exploring suitable lead positions of nonstandard ECGs. We selected six electrode positions, including A2, B3, C4, D5, K4, and L3, from the area where initial minima were located and compared their ECGs, which were recorded simultaneously in the course of surface mapping. When the ECG waveform both with the biggest Q wave and no initial r wave was considered to be related to the site of pathway — for example, if the ECG of L3 had the biggest Q wave, then the pathway site was assumed to be the left posterior — we could localize the site correctly in 34 of 41 patients (83%). This method of prediction using lead positions of nonstandard ECGs was expected to improve accuracy in locating the site of the accessory pathway.

**Discussion**

ECGs or vectorcardiograms have been used to predict the site of accessory pathways in patients with WPW syndrome. Recently, surface maps have been introduced as a more accurate noninvasive diagnostic method. Yamada et al. classified surface maps into three types by potential distribution during the QRS complex. De Ambroggi et al. identified six types of surface maps, according to the location of the maximum and minimum during the delta wave. Iwa et al. studied the reliability of minima appearing on the body surface at 40 msec of the QRS complex in predicting the sites of accessory pathways. They indicated the difficulty of pinpointing the sites in some cases because the minima from the same site of preexcitation did not always appear in the same specific circumscribed area and the area was not distinctly demarcated from other areas where minima from different sites appeared. Benson et al. reported that potential distributions in the first 40 msec of the QRS complex were reliable, although the sites of accessory pathways could not be localized correctly in patients without marked preexcitation. In previous reports, they observed only the potential distribution at a given time after the onset of ventricular activation and did not pay attention to the potential amplitude. Therefore, when the duration of the delta wave varied from patient to patient, the prediction, based on potential distribution at a critical instant, was affected significantly by the time selected.

**FIGURE 6.** Relationship between site of accessory pathway and location of the minimum at 40 msec of the QRS complex. Asterisks identify patients whose minima appeared where those due to accessory pathways in other sites should appear.
Our study showed that the preexcitation sites predicted by the location of minima at 40 msec of the QRS complex did not correspond with the true sites in 11 of all 41 patients or in seven of 18 with the small delta wave (figure 6). Our results were in agreement with theirs in that the sites of accessory pathways were not correctly predicted in many group 2 patients (small delta wave). This was probably due to the influence on the body surface potential distribution of ventricular activation initiated via the normal conduction pathway. To avoid this influence, Benson et al. indicated the significant variability of potential distributions during the first 10 to 40 msec of the QRS complex in one-third of their patients with marked preexcitation. They concluded that early QRS maps before 40 msec were unreliable in predicting sites of pathways. Our results showed they were still reliable in most cases, as long as the minimum potential became deeper than $-0.15 \text{ mV}$ before 40 msec of the QRS complex. The mean time for attaining a minimum potential of $-0.15 \text{ mV}$ was 19.4 ± 10.4 msec in all cases (table 1); 40 msec of the QRS complex was too late for prediction in five patients with the small delta wave.

Instead of unreliable early QRS maps, Benson et al. also stressed the usefulness of ST segment maps in the prediction of preexcitation sites. However, we could not find good correlation between ST segment maps and sites of accessory pathways. The reason for this is not known, but differences in electrode array, recording system, or degree of preexcitation in their study and ours may have brought about the discrepancy in results.

In five of our 41 patients, the accessory pathway was misjudged as located in an adjacent site by our index (figure 4). This may not represent a true error, but rather an overlap of adjacent zones that may be caused by continuity of anatomic pathway loci and orientation or effects of the torso shape and inhomogeneity. Explanations for this include the possibilities that (1) the epicardial breakthrough may have appeared at a site remote from the ventricular end of the accessory pathway, (2) cardiac size or rotation may have influenced the potential distribution on the body surface, (3) electrode arrangement may have been too sparse for detailed evaluation, (4) the terminal phase of atrial depolarization or repolarization may have influenced the low-level potential distribution of the early QRS complex in patients with a short P-delta interval, and (5) multiple accessory pathways may have been present in spite of extensive electrophysiologic evaluation. It is necessary to take these possibilities into consideration when predicting pathway sites.

Standard ECGs were not as reliable as surface maps, as Gallagher et al. pointed out, particularly in patients with the small delta wave. We could localize the site of pathway only in 50% of them when we used the delta wave polarity as a predicting index. For this reason,
standard ECG leads may not be adequate for diagnosis of WPW syndrome. We have shown by the preliminary data based on our map analysis that ECGs recorded from six preselected points on the body surface could localize the site of pathways in 83% of the patients. This result indicates that the nonstandard V-lead ECG may be a powerful predictor of the sites of accessory pathways. Identification of the most adequate ECG loci and most effective method for prediction by routine ECG equipment are problems for future investigation.

This study has shown the value of amplitude-based map analysis over time-based analysis in predicting single accessory pathway sites, particularly in patients with the small delta wave, and the exact locations of minima on a clearly defined electrode grid, which might be of value in the application of standard ECG electrodes. We conclude that the location of minima with a potential of $-0.15 \text{ mV}$ in the early QRS complex is a simple and accurate index of the site of the accessory pathway in most patients, regardless of the degree of ventricular fusion. These results are expected not only to improve the diagnostic accuracy of surface maps but also to contribute to the expansion of the diagnostic utility of standard or nonstandard ECGs.

References

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