Reproducibility of Local Activation Times During Intraoperative Epicardial Mapping

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SUMMARY The reproducibility of the measurement of local epicardial activation times using intraoperative epicardial mapping was studied in 10 patients. Two multipolar electrodes were used, one as a reference located on the anterior surface of the heart, the other as a hand-held probe for depicting local activation times. During atrial pacing at 100 beats/min, activation times at various sites of the heart were measured consecutively using grid systems that defined 75 points on the surface of the heart. The surface of the heart was divided into three areas: the anterior wall of the right ventricle, the left lateral wall and the posterior wall of both ventricles. The measurements were repeated twice at the same paced atrial rate. From these data the mean absolute differences between the first and second measurements were calculated for each point.

The earliest onset of excitation was depicted on the surface of the anterior wall of the right ventricle. The mean difference between the first and second measurement at identical paced rates was 8 msec on the anterior wall, 6 msec on the left lateral wall, and 7 msec on the posterior wall. At individual points, the differences were much greater (40 msec maximum). These greater differences were mainly found in the area of epicardial fat tissue in the atrioventricular groove or the sulcus interventricularis.

Using a visual coordinate system, it is possible to achieve a good reproducibility of local activation times, above all with respect to the large differences that may be found in patients with accessory pathways or with ventricular tachycardia.

SURGICAL PROCEDURES have become increasingly important in treating intractable ventricular tachyarrhythmias in patients with or without the preexcitation syndrome. The operative approach is aimed at the interruption of reentry circuits or the excision of foci considered responsible for the genesis of these arrhythmias. The success of surgical intervention depends on exact localization of these sites by mapping the sequence of epicardial and endocardial depolarizations. Although several studies on this subject have been published, no thorough information is available on the reproducibility of epicardial signals during intraoperative mapping in man. However, this is an important prerequisite for later surgical procedures.

To provide information on the reproducibility of measurements of the epicardial activation sequence, duplicate recordings of epicardial potentials were obtained from patients with coronary heart disease or valvular or septal defects who were undergoing open heart surgery.

Patients and Methods

Ten patients were studied during open heart surgery. The clinical diagnoses and the electrocardiographic findings are presented in table 1. All patients gave informed consent for epicardial mapping as an additional procedure.

The measurements were done intraoperatively after median sternotomy and exposure of the epicardium before cardiopulmonary bypass. A multipolar reference electrode was sutured to the anterior wall of the right ventricle. The measurement of epicardial activation times at various sites in the heart was done by means of a tripolar, hand-held probe with an electrode distance of 1.5 mm. All signals were immediately amplified at the operation desk to a standardized level of 1.4 V using specially designed amplifiers (impedance 10 kΩ). No special filter settings were used (cut-off frequency 1.6–700 Hz). Throughout the study the degree of amplification was kept constant to avoid changing the slew rate of the initial deflection of epicardial signals.

The shape and amplitude of bipolar signals from closely adjacent electrodes depends largely on the direction of the wave of excitation relative to the localization of the electrodes. To minimize bias that might be introduced by this factor, three triangularly arranged electrodes were used, yielding three bipolar signals. All signals of this hand-held probe were then algebraically summed. The reference and probe signals (a fixed time signal) and two surface ECG leads were transferred to the registration room outside the operating theater. Recordings were obtained using an eight-channel, direct-writing, ink-jet recorder (Siemens Elema, Mingraph) with a paper speed of 100 mm/sec. The upper limit of measurement accuracy was ± 5 msec. At higher speeds, the onset of the signal is more difficult to define, thus introducing greater inaccuracies. The onset of a bipolar epicardial signal was taken when the upstroke from the baseline reached a degree of 45°, as suggested by Scherlag and co-workers.

The epicardial sites used to measure local activation were defined according to anatomic landmarks. The main line for orientation was the ramus interventricularis anterior to the left coronary artery. The heart was divided into an anterior, left lateral and...
TABLE 2. Clinical Diagnosis and Electrocardiographic Findings

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Diagnosis</th>
<th>P (msec)</th>
<th>PQ (msec)</th>
<th>QRS (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>59</td>
<td>M</td>
<td>CAD</td>
<td>110</td>
<td>160</td>
<td>100</td>
</tr>
<tr>
<td>2</td>
<td>8</td>
<td>F</td>
<td>PS + VSD</td>
<td>90</td>
<td>150</td>
<td>80</td>
</tr>
<tr>
<td>3</td>
<td>63</td>
<td>F</td>
<td>AS</td>
<td>110</td>
<td>190</td>
<td>100</td>
</tr>
<tr>
<td>4</td>
<td>50</td>
<td>M</td>
<td>CAD</td>
<td>80</td>
<td>190</td>
<td>90</td>
</tr>
<tr>
<td>5</td>
<td>58</td>
<td>M</td>
<td>CAD</td>
<td>100</td>
<td>190</td>
<td>100</td>
</tr>
<tr>
<td>6</td>
<td>54</td>
<td>M</td>
<td>CAD</td>
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<td>160</td>
<td>100</td>
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<td>7</td>
<td>59</td>
<td>F</td>
<td>AS</td>
<td>100</td>
<td>180</td>
<td>80</td>
</tr>
<tr>
<td>8</td>
<td>66</td>
<td>F</td>
<td>KHK</td>
<td>110</td>
<td>190</td>
<td>80</td>
</tr>
<tr>
<td>9</td>
<td>15</td>
<td>F</td>
<td>ASD</td>
<td>100</td>
<td>180</td>
<td>90</td>
</tr>
<tr>
<td>10</td>
<td>21</td>
<td>F</td>
<td>ASD</td>
<td>100</td>
<td>180</td>
<td>90</td>
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<td>Mean</td>
<td>45.3</td>
<td></td>
<td></td>
<td>99</td>
<td>177</td>
<td>91</td>
</tr>
</tbody>
</table>

Abbreviations: CAD = coronary artery disease; PS = pulmonic stenosis; VSD = ventricular septal defect; AS = aortic stenosis; ASD = atrial septal defect; KHK =

posterior wall. These major areas were again subdivided using a special grid system (fig. 1). The crossing points of this grid system were labeled by letters and digits (A1, A2, B1, B2, etc.). Local epicardial activation times were measured from these crossing points as the difference between the reference signal and the probe signal. The site of earliest epicardial activation — breakthrough — was considered point zero. All epicardial data were referred to this point. Mean values at each site were calculated from at least five consecutive cycles.

In each patient, epicardial activation times were measured twice (first and second measurements). During each study the interval between the reference and the summation signal was measured for each point of the grid system, as indicated above.

Then, in each patient the mean differences between the mean values of the first and second measurements were calculated for each point. From these data, the mean absolute differences, the standard deviation and the range of the differences between measurements were calculated for the anterior, left lateral and posterior wall of the heart for all 10 patients. In three cases the intraindividual accuracy of measurement was estimated by repeating the measurement after 2 months. The mean absolute deviation between both measurements was 3.0 ± 2.0 msec [SD]. The intraindividual accuracy was 5.0 ± 3.0 msec.

Results

Figure 2 is a representative example of the local activation times in one patient. The earliest epicardial signals occurred on the surface of the anterior wall of the right ventricle (C3, D3), 15–25 msec after the onset of the QRS. This delay is due to the spread of excitation from endocardium to epicardium. The latest activation times were depicted at about 40 msec on the posterior wall of the left ventricle (O1, P1, Q1). At these sites the differences were more pronounced (up to 18 msec) due to low-amplitude signals of which the onset was more difficult to define. These areas were commonly covered with increased epicardial fat tissue. The anterior wall was activated in 18 msec, the left lateral in 21 msec and the posterior wall in 24 msec during the first measurement; during the second measurement, the mean activation times were 17 msec, 19 msec and 20 msec, respectively. The mean difference (fig. 2) between measurements was 4 msec (range 0–9 msec) in the anterior wall area, 6 msec (range 2–13 msec) in the left lateral wall and 6 msec (range 0–18 msec) in the posterior wall.

Table 2 gives the results in all patients. The mean difference between the first and second measurement was 8 msec (range 4–12 msec) in the anterior wall, 6 msec (range 3–11 msec) in the left lateral wall and 7 msec (range 5–15 sec) in the posterior wall.

Discussion

The normal epicardial activation of the human heart has been described.\textsuperscript{12, 14} In the revived human heart the earliest epicardial breakthrough is observed in the pretrabecular area.\textsuperscript{15} Then, the activation spreads radially to the apex and the base of the heart. The latest activation times can be recorded from the

![Diagram](http://circ.ahajournals.org/)

**Figure 1. Epicardial grid for mapping.**

RA = right atrium; LA = left atrium; PA = pulmonary artery.
Reproducibility of mean local activation times in a representative patient. (A) Each pair of numbers represents the mean values obtained during the first and second measurements (msec). (B) Mean absolute differences of local epicardial activation times (msec) during repeated measurements. RA = right atrium; LA = left atrium; PA = pulmonary artery.

Figure 2. Reproducibility of mean local activation times in a representative patient. (A) Each pair of numbers represents the mean values obtained during the first and second measurements (msec). (B) Mean absolute differences of local epicardial activation times (msec) during repeated measurements. RA = right atrium; LA = left atrium; PA = pulmonary artery.

Posterobasal area of the left ventricle. During intraoperative mapping, the earliest onset of epicardial activation in the anterior wall of the right ventricle occurs at about 18–25 msec; the latest activation times in the right ventricular posterobasal area occur about 70–80 msec after the onset of the surface QRS. The earliest epicardial potentials of the left ventricle could be registered from the posterobasal and posteromedial paraseptal area, the latest from the left ventricular posterobasal paraseptal area. Our investigations are in agreement with the results of these studies concerning the sequence and timing of the epicardial activation of the human heart.

Intraoperative epicardial mapping has mainly been used to localize the origin of ventricular tachycardia of accessory pathways. In most studies a visual grid system has been used to identify the various sites of epicardial activation. This grid system is defined by typical landmarks. Recently, attempts have been made to register multiple epicardial signals simultaneously. However, this is technically difficult and requires on-line computer measurement of the
Table 2. Local Epicardial Activation Times During Repeated Measurements in 10 Patients

<table>
<thead>
<tr>
<th>Location</th>
<th>Anterior wall</th>
<th></th>
<th>Left lateral wall</th>
<th></th>
<th>Posterior wall</th>
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<tbody>
<tr>
<td>Mean</td>
<td>A1 2 3 4 5</td>
<td>B1 2 3 4 5</td>
<td>C1 2 3 4 5</td>
<td>D1 2 3 4 5</td>
<td>E1 2 3 4 5</td>
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<tr>
<td>difference</td>
<td>4 7 7 12</td>
<td>12 7 5 9 7</td>
<td>7 9 7 3 5</td>
<td>11 9 8 5 5</td>
<td>2 3 4 5 6</td>
</tr>
<tr>
<td>sd</td>
<td>3 5 4 5 7</td>
<td>12 6 5 9 5</td>
<td>7 3 6 2 4</td>
<td>9 11 6 3 4</td>
<td>4 13 5 9 6</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Location</th>
<th>J1 2 3 4 5</th>
<th>K1 2 3 4 5</th>
<th>L1 2 3 4 5</th>
<th>M1 2 3 4 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>8 5 3 9 5</td>
<td>11 7 8 9 6</td>
<td>8 3 3 7 5</td>
<td>5 8 9 11 8</td>
</tr>
<tr>
<td>sd</td>
<td>10 5 3 4 3</td>
<td>10 5 5 5 5</td>
<td>10 4 3 8 4</td>
<td>5 8 10 8</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Location</th>
<th>N1 2 3 4 5</th>
<th>O1 2 3 4 5</th>
<th>P1 2 3 4 5</th>
<th>Q1 2 3 4 5</th>
<th>G1 2 3 4 5</th>
<th>F1 2 3 4 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>9 9 6 15 7</td>
<td>12 14 7 8 11</td>
<td>12 8 9 8 15</td>
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<td>11 11 5 5 4</td>
<td>9 10 5 9 9</td>
<td>5 9 6 5 5</td>
<td>5 5 5 8 6</td>
<td>4 7 4 7 7</td>
</tr>
</tbody>
</table>

Values are in milliseconds.

signals. This procedure, therefore, will not be available for all cardiac centers, and the visual grid system will probably remain of practical importance.

The reproducibility of epicardial signals using this visual grid system has not yet been clearly defined in humans. Spielman and co-workers, using automatic devices for measurement of epicardial signals in animals, reported that the reproducibility at various sites was about 2 msec. These data were related to the reproducibility of measuring the same signal but not to the retrieval of the same site of activation. Recently Wyndham et al. gave a more detailed report on the normal activation of the human heart during epicardial mapping, with only limited data on the reproducibility of their technique.

Therefore, we examined 10 patients during intraoperative epicardial mapping. The mapping was performed twice during identical supraventricular paced rhythms. The mean difference between the first and second measurements in all patients irrespective of the site of measurement was about 8 msec. However, at single points the maximal deviation was up to 40 msec.

There are three possible reasons for the differences between the first and second measurements. First, there is some inevitable error in measuring intracardiac and epicardial signals due to accuracy of measurement and recording speed. Second, in some areas on the surface of the heart, the onset of the signal was difficult to define because of low amplitudes and slow rates. This occurred predominantly in regions with a large amount of epicardial fat tissue. Third, reproducibility of identical sites of the grid system may be difficult, especially in the posterior wall area. Our data, for example, showed worse reproducibility in the posterior wall area than in the anterior wall area.

In patients with ventricular tachycardia or reentrant tachycardias due to accessory pathways, the deviation of the activation times from normal may range up to 100 msec or more after the onset of the reference signal. Therefore, the relatively small difference between the first and second measurements is not clinically significant for detecting areas with low conduction or an accessory pathway.

In conclusion, our results clearly show that the use of a visual grid system allows good reproducibility of epicardial activation patterns. This is an important prerequisite for localizing the sites of reentry or accessory pathways.

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