Signal-Averaged Electrocardiogram

Improved Identification of Patients With Ventricular Tachycardia Using a 28-Lead Optimal Array

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Background. Although the signal-averaged ECG (SAECG) is currently the best noninvasive test to identify patients with ventricular tachycardia (VT) following myocardial infarction (MI), it still is a relatively insensitive test. Body surface mapping has improved the sensitivity of ECG in detecting various cardiac diseases. This study applied body surface mapping to the SAECG in the form of a clinically practical, 28-lead optimal array and compared its sensitivity and specificity with those of an orthogonal array.

Methods and Results. Two hundred twenty-three patients with previous MI (82 with inducible VT) underwent SAECG using 28 surface electrodes from which were obtained a three-lead orthogonal array and a 28-lead optimal array (optimal). From the orthogonal array, two QRS durations (QRSd) were obtained using the combined vector magnitude method (CVM) and the earliest onset to latest offset of the three individually filtered leads (individual). From the optimal array, 28 QRSd were obtained, each defined as the duration from the earliest onset of any of the 28 leads to the offset of each individually filtered lead. QRSd >120 msec in ≥3 leads was considered abnormal. For CVM and individual, QRSd of >120 msec were considered abnormal. While the specificity of each method was comparable (84%, 86%, and 84% for CVM, individual, and optimal, respectively), the sensitivity of optimal (70%) was significantly greater than the sensitivity of CVM (54%) (p = 0.001) or individual (59%) (p = 0.004). The magnitude of improvement in sensitivity, 16% and 15%, respectively, was equal for anterior (n = 120) and inferior (n = 103) infarctions.

Conclusions. Body surface mapping using the 28-lead optimal array significantly improved the sensitivity of the SAECG without loss of specificity. The increased sensitivity was of equal magnitude for inferior and anterior infarctions. The superiority and practicality of the 28-lead optimal array make it worth pursuing as an option for further refinement in SAECG. (Circulation 1993;87:857–865)

Key Words • mapping, body surface • ventricular tachycardia • electrocardiography • late potentials

Spontaneous ventricular tachycardia (VT) is a common cause of sudden death following myocardial infarction. The etiology is thought to be due to viable myocardial fibers interdigitating with fibrous tissue at the margin of an infarct acting as substrate for reentry.1-5 Slow conduction in these tortuous strands of viable muscle fibers is thought to generate late potentials. Detection of late potentials using the signal-averaged ECG (SAECG) is one of the best noninvasive tests for predicting patients prone to VT and sudden death following myocardial infarction.6-10 Techniques of detecting late potentials have been refined over the past decade.11 Noise reduction by signal averaging several hundred cardiac cycles to improve the signal-to-noise ratio is crucial to the detection of these low-amplitude signals, which are of the order of 5–20 μV.12,13 However, the SAECG in its current form still has a relatively poor positive predictive accuracy of 17% to 27%.6,8,14-17 The test often fails to detect late potentials in patients with VT following anterior myocardial infarctions compared with those with inferior infarctions.9 Further improvement in sensitivity without loss in specificity is desirable, particularly for patients with anterior infarctions.

The amplitude of a late potential at the body surface depends on the media and distance through which the signal conducts from the site of delayed conduction in the heart to the recording electrode on the body surface. The vector direction of the late potential itself is also important as weak signals will only be recorded by a lead whose vector direction lies more or less in the same direction as that of the late potential. By using a larger number of electrodes strategically located over a large area on the torso, the detection of late potentials should be enhanced.

Extensive body surface potential mapping has been shown to improve the detection of cardiac diseases such
as myocardial infarction, left ventricular hypertrophy, right ventricular hypertrophy, and ischemia during exercise stress testing.\textsuperscript{18-21} Body surface mapping of late potentials has also been used to correlate with site of origin of VT.\textsuperscript{22-26} However, the application of body surface potential mapping to improve the sensitivity of SAECG in predicting patients with VT following myocardial infarction has not been described.

Extensive body surface mapping using 64–200 electrodes is cumbersome to apply and is therefore seldom used clinically. A practical, limited-lead set, termed optimal array, has been described for more than a decade.\textsuperscript{27-29} By using 20–30 properly located electrodes, the optimal array permits accurate estimation of body surface potential distribution by reducing the redundant information obtained from nearby electrodes of a more extensive array.

The aims of this study were to determine if extensive body surface potential mapping in the form of a clinically practical, 28-lead optimal array could improve the sensitivity of the SAECG in identifying patients with VT without loss of specificity and to determine if any improvement in late potential detection by body surface mapping is specific to the site of infarction.

\textbf{Methods}

Two hundred twenty-three patients with previous myocardial infarction undergoing electrophysiological studies were entered into the study. All patients underwent programmed ventricular stimulation for either management of documented sustained VT or out-of-hospital cardiac arrest or risk stratification after myocardial infarction. Patients with bundle branch block were excluded. “Cases” (n=82) were patients with documented sustained VT or out-of-hospital cardiac arrest who also had VT induced during programmed ventricular stimulation. Coronary angiography and left ventriculography was performed in all “cases” to document prior myocardial infarction. “Controls” (n=141) were those who did not have inducible VT during programmed ventricular stimulation for risk stratification at approximately 1–2 weeks after acute myocardial infarction. Thus, all patients had previous myocardial infarction at least 7 days before the time of study. All patients had been off antiarrhythmic drugs, including \(\beta\)-blockers, for at least 5 days. Patients on amiodarone were excluded from the study. Details of the programmed stimulation protocol have been described previously.\textsuperscript{30} In brief, one, two, three, and four extrastimuli were introduced during pacing at a cycle length of close to 600 msec at the right ventricular apex, using a rectangular pulse of 2 msec and twice-diastolic threshold. A negative test was defined as ventricular fibrillation, ventricular flutter, or fast VT of cycle length less than 230 msec or no arrhythmia inducible with four extrastimuli. A positive test was defined as inducible monomorphic VT and cycle length of 230 msec or more and lasting more than 10 seconds. Patient characteristics are shown in Table 1. Sites of infarction, as determined from ECGs, wall motion abnormalities on gated heart blood pool scans, and/or ventriculography during cardiac catheterization were broadly categorized into anterior and inferior. Of the 26 patients with a history of more than one infarction, 16 had infarctions involving territories from both categories. For these 16 patients (four with inducible VT), the most recent site of infarction was used for the classification.

\textbf{SAECG}

Each patient underwent one single SAECG in the supine position in an unshielded room on the same day as the electrophysiological study. Silver/silver chloride electrodes (3M Pediatric Monitoring Electrodes No. 2248, Ontario) were applied according to the grid shown in Figure 1 after the skin surface was cleansed with alcohol. In male patients, the anterior chest wall was shaved if necessary.

\textbf{Optimal Array}

An optimal 28-lead array described by Lux\textsuperscript{29} was used (Figure 1). This is a practical, limited lead set derived from a 192-lead body surface mapping system. By using 28 properly located electrodes, it allows accurate estimation of total body surface potential distribution, offering a practical solution to the cumbersome task of

\begin{table}
\centering
\caption{Patient Characteristics}
\begin{tabular}{|l|l|l|}
\hline
& Cases & Control \\
(n=82) & (n=141) \\
\hline
Inducible VT & Yes & No \\
Age (±SD years) & 60±10 & 56±10 \\
Male:female & 76:6 & 117:24 \\
LVEF (%) & 32±13 & 47±15 \\
MI (ant:inf) & 44:38 & 76:65 \\
\hline
\end{tabular}
\end{table}

LVEF, left ventricular ejection fraction; MI, myocardial infarction; Ant, anterior; Inf, inferior.

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure1.png}
\caption{Position of the 28 electrodes on the torso. The thick upper and lower lines correspond to horizontal levels just below the sternal notch and just above the umbilicus, respectively. The thick center line runs down the center of the chest anteriorly and the thick lines on the left and right correspond to the midaxillary lines. X, Y, and Z leads are a subset of the 28 leads.}
\end{figure}
a more extensive body surface mapping system. Each lead was referenced to the Wilson central terminal.

Orthogonal Array

The X, Y, and Z leads were a subset of the 28-lead system (Figure 1). The X lead was at the left midaxillary line at the fourth intercostal space. The Y lead was to the left of the umbilicus, and the Z lead was at the V$_2$ position. Each lead was referenced to the Wilson central terminal.

ECGs from the 28 leads were acquired simultaneously, amplified (5,000 gain), and filtered (0.05–500 Hz). Data were concurrently sampled at 1 kHz/channel, digitized at 12-bit resolution, and recorded continuously for a fixed duration of 250 seconds. Between 250 and 400 beats were usually acquired during this interval. The recordings from the X, Y, and Z leads were then recalled in 2-second segments so that ventricular premature beats and noisy segments of recording could be marked manually for exclusion from averaging. The two sinus beats following each ventricular premature beat were also excluded. Averaging was performed on a DEC PDP 11/73 computer after an iterative cross-correlation procedure had been used to optimize QRS alignment.$^{31}$

Each averaged complex from the 28 unipolar leads was filtered bidirectionally with a 25-Hz high-pass filter (four-pole Butterworth). The areas to commence forward search for QRS onset and backward search for QRS offset were manually selected. Simson’s computer algorithm was used to find the QRS offset for each lead. Thus, the offset is the midpoint of a 5-msec segment where the average value exceeded the mean +3 SDs of a 40-msec noise sample. To determine the QRS onset, the mean of the absolute differences between successive values over a 25-msec stationary segment within a relatively flat PR interval was compared with each subsequent change in value between two successive points. The onset point was defined as the first point of a 3-msec segment where all three successive changes in value exceeded the mean of the 25-msec stationary segment by threefold or more. For the combined vector magnitude, Simson’s computer algorithms for onset and offset search were used to find the QRS end points.

Noise for each of the 28 leads was measured as the root-mean-square voltage value of the filtered signal over a 40-msec segment immediately after the QRS offset for that lead. The mean noise achieved for each lead from a subset of 92 patients (60 consecutive patients with and 32 consecutive patients without inducible VT) is shown in Table 2. For the combined vector magnitude, noise was measured in the same manner and was less than 1 $\mu$V (mean, 0.5±0.2 $\mu$V).

For the optimal array, QRS duration for each lead was referenced to the onset point of the lead with the earliest QRS onset. QRS duration for each lead was defined as the duration from the earliest QRS onset of any of the 28 leads to the QRS offset for that lead. Thus, 28 QRS durations were obtained for the optimal array for each patient.

The conventional SAECG combines three orthogonal leads into a combined vector magnitude before analyzing for late potentials. This method can potentially attenuate the fidelity and amplitude of late potentials. Since each lead was analyzed individually in the optimal array, to allow a fair comparison between the orthogonal array and the optimal array, the orthogonal leads were analyzed individually as well as in the form of a combined vector magnitude.

Thus, two QRS durations were obtained for the orthogonal array. The first QRS duration was obtained from the combined vector magnitude of the X, Y, and Z leads (CVM). The second QRS duration was defined as the duration from the earliest QRS onset in any of the three individual X, Y, and Z leads to the latest QRS offset of any of the three individual X, Y, and Z leads (individual).

For both the CVM and individual, the cut point for defining late potentials was tested in steps of 5 msec from a QRS duration of 100 msec to a QRS duration of 140 msec except from 105–120 msec, where steps of 3 msec were used. Root-mean-square voltage for the last 40 msec of the combined vector magnitude was calculated (V40). Presence of low-voltage signals using two commonly used criteria: V40, <25 $\mu$V, and V40, <20 $\mu$V were also studied.$^{11,32,33}$

For the optimal array, two variables, i.e., the QRS duration and the number of leads exceeding this duration (nx), were studied. QRS duration was tested in steps of 5 msec from 100 msec to 140 msec while nx was tested in steps of 1 lead from nx ± 1 to nx ± 6.

Statistical Analysis

Data were expressed as mean±SD. Sensitivity for each system was the percentage of “cases” with an

<table>
<thead>
<tr>
<th>TABLE 2.</th>
<th>Mean Noise for Each Lead*</th>
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<tbody>
<tr>
<td>Lead</td>
<td>Mean (±SD) noise ($\mu$V)</td>
</tr>
<tr>
<td>1</td>
<td>0.3±0.2</td>
</tr>
<tr>
<td>2</td>
<td>0.4±0.2</td>
</tr>
<tr>
<td>3</td>
<td>0.3±0.2</td>
</tr>
<tr>
<td>4</td>
<td>0.4±0.3</td>
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<tr>
<td>5</td>
<td>0.3±0.2</td>
</tr>
<tr>
<td>6</td>
<td>0.4±0.3</td>
</tr>
<tr>
<td>7</td>
<td>0.4±0.3</td>
</tr>
<tr>
<td>8</td>
<td>0.4±0.2</td>
</tr>
<tr>
<td>9</td>
<td>0.4±0.2</td>
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<tr>
<td>10</td>
<td>0.4±0.2</td>
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<tr>
<td>11</td>
<td>0.4±0.3</td>
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<tr>
<td>12</td>
<td>0.4±0.2</td>
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<tr>
<td>13</td>
<td>0.4±0.3</td>
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<tr>
<td>14</td>
<td>0.4±0.3</td>
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<tr>
<td>15</td>
<td>0.4±0.3</td>
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<tr>
<td>16</td>
<td>0.3±0.2</td>
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<tr>
<td>17</td>
<td>0.4±0.2</td>
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<tr>
<td>18</td>
<td>0.4±0.3</td>
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<td>19</td>
<td>0.4±0.3</td>
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<tr>
<td>20</td>
<td>0.4±0.4</td>
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<tr>
<td>21</td>
<td>0.4±0.3</td>
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<tr>
<td>22</td>
<td>0.4±0.3</td>
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<tr>
<td>23</td>
<td>0.4±0.4</td>
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<tr>
<td>24</td>
<td>0.4±0.2</td>
</tr>
<tr>
<td>25</td>
<td>0.3±0.2</td>
</tr>
<tr>
<td>26 (=$X$)</td>
<td>0.4±0.3</td>
</tr>
<tr>
<td>27 (=$Y$)</td>
<td>0.4±0.2</td>
</tr>
<tr>
<td>28 (=$Z$)</td>
<td>0.4±0.2</td>
</tr>
</tbody>
</table>

*Obtained from 92 patients (60 with inducible VT).
Orthogonal
Optimal
versus
for that
pare
124±28
was
abnormal SAECG using the criteria for late potentials for that system. Specificity for each system was the percentage of “controls” with a normal SAECG using the criteria for late potentials for that system. McNemar’s test was used for comparing sensitivities among the different systems. Wilcoxon’s test was used to compare QRS durations obtained by the different systems.

**Results**

The mean QRS durations for the different methods are shown in Table 3. For all three methods, QRS durations were significantly longer in patients with VT than in those without VT. For the orthogonal array, this was 124±28 msec versus 103±17 msec using the combined vector magnitude (p<0.0001) and 129±25 msec versus 103±15 msec using the individual lead analysis method (p<0.0001). For the optimal array, using the longest QRS duration from the 28 leads, this was 139±26 msec versus 111±17 msec (p<0.0001). When the study population was considered as a whole (Table 4), both the CVM (111±24 msec, p<0.0001) and individual (113±23 msec, p<0.0001) underestimated QRS durations when compared with the optimal array (121±25 msec). There was a tendency for the combined vector magnitude to underestimate QRS duration compared to the individual lead analysis method (111±24 msec versus 113±23 msec, p=0.06).

Sensitivities and specificities for some of the tested criteria for the combined vector magnitude method and the optimal array are shown in Table 5. In general, when a shorter QRS duration was used to define late potentials, sensitivity rose while specificity fell. For the optimal array, when the number of leads (nx) required to exceed a defined QRS duration was increased, sensitivity decreased while specificity increased. Thus, setting a QRS duration criterion at 120 msec, if nx criterion was changed from nx≥1 to nx≥4, the sensitivity would decrease from 74% to 67% while the corresponding specificity increase from 75% to 84%. Figure 2 is a plot of sensitivity against specificity achieved by the two arrays. For the same specificity achieved by each array, the optimal array achieved a higher sensitivity compared to the orthogonal array. Also, for any sensitivity, the optimal array achieved a higher specificity than the orthogonal array.

The effect of combining QRS duration with V40 in defining late potentials is shown in Table 3 and Figure 3. For each combination of QRS duration with V40, there was an identical result achievable by using only a single QRS duration as the criterion for defining late potentials. Since 120 msec is the commonly used cut point in the literature for defining late potentials and since this cut point was associated with the best predictive accuracy in both methods of analysis in the present study, a QRS duration of 120 msec will be used hereafter as the cut point for both the combined vector magnitude and the individual lead analysis methods. For the optimal array, a late potential will be defined as QRS duration of more than 120 msec in three or more leads.

Using the above criteria, of the 141 patients with no VT, the orthogonal array correctly classified 119 and 121 patients using the combined vector magnitude and individual lead analysis methods, respectively, yielding specificities of 84% and 86%, respectively (Table 6). This was similar to the specificity of 84% achieved by the optimal array. Of the 82 patients with VT, the orthogonal array correctly classified 44 and 48 patients using the combined vector magnitude and individual lead analysis methods, yielding sensitivities of 54% and 59%, respectively. The optimal array achieved a sensitivity of 70%, significantly more sensitive when compared with the combined vector magnitude (p=0.001) or the individual lead analysis method (p=0.004). For the orthogonal array, there was a nonsignificant trend toward the individual lead analysis method being more sensitive and more specific than the combined vector magnitude method.

Of the 223 patients studied, there were 120 patients with prior anterior infarctions and 103 patients with prior inferior infarctions. Of the 120 patients with anterior infarctions, there were 44 patients with and 76 patients without VT (Table 7). Of the 103 patients with inferior infarctions, there were 38 patients with and 65 patients without VT. Compared with the method of combined vector magnitude, the optimal array was significantly more sensitive in both the anterior infarction group and the inferior infarction group, allowing an additional seven patients and six patients in the two groups, respectively, to be identified. There was a nonsignificant trend for the optimal array to be more sensitive than the individual lead analysis method of the orthogonal array, correctly classifying an additional five patients in the anterior infarction group (p=0.06) and four patients in the inferior infarction group (p=0.1). Within the orthogonal array, there was a nonsignificant trend for the individual lead analysis method to be more sensitive than the combined vector magnitude method (p=0.7). While the optimal array tended to be more sensitive in identifying VT patients with inferior infarctions compared with anterior infarctions (76% versus 64%), it tended to be less specific in identifying non-VT patients with inferior infarctions compared with anterior infarctions (80% versus 87%), so that there was no overall difference in the predictive accuracies (p=0.95).

The absolute magnitude of increase in sensitivity obtained by the optimal array was 16% for the anterior infarction group and 15% for the inferior infarction

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**Table 3. QRS Durations Between Cases and Controls**

<table>
<thead>
<tr>
<th>Cases (n=82)</th>
<th>Controls (n=141)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Orthogonal array (msec)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Combined vector magnitude</td>
<td>124±28</td>
<td>103±17</td>
</tr>
<tr>
<td>Individual lead analysis</td>
<td>129±25</td>
<td>103±15</td>
</tr>
<tr>
<td>Optimal array (msec)</td>
<td>139±26</td>
<td>111±17</td>
</tr>
</tbody>
</table>

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**Table 4. QRS Durations by Different SAECG Methods**

<table>
<thead>
<tr>
<th>Patients (n=223)</th>
<th>QRS durations</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Orthogonal array (msec)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Combined vector magnitude</td>
<td>111±24</td>
<td>&lt;0.001*, 0.06†</td>
</tr>
<tr>
<td>Individual lead analysis</td>
<td>113±23</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Optimal array (msec)</td>
<td>121±25</td>
<td></td>
</tr>
</tbody>
</table>

*Compared with optimal array.
†Compared with individual lead analysis.
group; this represented improvements over the combined vector magnitude method of 33% and 25%, respectively.

**Discussion**

Current methods of SAECG have a relatively low positive-predictive value of approximately 17-27% in post-myocardial infarction patients.\(^6,8,14-17\) Further improvements in sensitivity without loss in specificity are required. Continued methodological refinements may include the use of different lead systems, refined methods of signal processing, and novel analyses. In this study, we compared the sensitivity and specificity of two different lead systems, using the same hardware for data acquisition, processing, and analysis in the same group of patients. By using a limited lead set of 28 strategically positioned electrodes,\(^29\) termed optimal array, there was an improvement of 15% in sensitivity compared with an orthogonal array without any loss in specificity. This is represented by a shift to the right in the sensitivity/specificity curve in Figure 2. The refinement was at least as valuable in patients with anterior infarctions as in those with inferior infarctions. The absolute magnitude of increase in sensitivity obtained by the optimal array was 16% for the anterior infarction group and 15% for the inferior infarction group.

Existing methods of detecting late potentials suffer several limitations. 1) The usual practice of combining three orthogonal leads into a single vector magnitude assumes that the combined complex can provide a “total view” of the heart. This method can potentially attenuate the fidelity and amplitude of late potentials. The X, Y, and Z leads may not be truly orthogonal or balanced due to electrical inhomogeneities within the body, variability in posture, torso shape, and anatomic location of the heart. 2) Even if the X, Y, and Z leads were truly orthogonal for a particular patient or specific heart-to-torso relation and were analyzed individually, if the signal-to-noise ratio was unfavorable (< three times the noise level) or if late potentials were not dipolar, late potentials may be missed. Qualitative assessment of filtered or unfiltered individual leads may help to identify late potentials in these situations, but this is highly subject to interobserver and intraobserver variability. 3) The anterior and septal regions of the heart are depolarized early in the QRS complex during sinus rhythm. As a result, the SAECG is less sensitive and has a lower predictive accuracy for predicting VT patients with anterior or anteroseptal myocardial infarctions than for identifying VT patients with inferior or inferoposterior myocardial infarctions.

### Table 5. Results of Some Tested Criteria to Define Late Potentials

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Sens (%)</th>
<th>Spec (%)</th>
<th>PA (%)</th>
</tr>
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<tbody>
<tr>
<td>Combined vector magnitude (msec)</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>QRSd&gt;105</td>
<td>82</td>
<td>55</td>
<td>65</td>
</tr>
<tr>
<td>QRSd&gt;108</td>
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<td>62</td>
<td>68</td>
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<tr>
<td>QRSd&gt;111</td>
<td>72</td>
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<tr>
<td>QRSd&gt;114</td>
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<td>71</td>
</tr>
<tr>
<td>QRSd&gt;117</td>
<td>61</td>
<td>79</td>
<td>72</td>
</tr>
<tr>
<td>QRSd&gt;120</td>
<td>54</td>
<td>84</td>
<td>73</td>
</tr>
<tr>
<td>QRSd&gt;125</td>
<td>42</td>
<td>89</td>
<td>72</td>
</tr>
<tr>
<td>QRSd&gt;110 (or V40&lt;25 μV)</td>
<td>77</td>
<td>60</td>
<td>66</td>
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<td>QRSd&gt;115 (or V40&lt;25 μV)</td>
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<td>QRSd&gt;115 (or V40&lt;20 μV)</td>
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<td>QRSd&gt;125 (or V40&lt;25 μV)</td>
<td>64</td>
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<td>70</td>
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<td>QRSd&gt;125 (or V40&lt;20 μV)</td>
<td>61</td>
<td>76</td>
<td>70</td>
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<table>
<thead>
<tr>
<th>Optimal array (msec)</th>
<th></th>
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<tbody>
<tr>
<td>QRSd&gt;115 (nx=1)</td>
<td>85</td>
<td>62</td>
<td>70</td>
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<tr>
<td>QRSd&gt;115 (nx=2)</td>
<td>78</td>
<td>69</td>
<td>72</td>
</tr>
<tr>
<td>QRSd&gt;115 (nx=3)</td>
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<tr>
<td>QRSd&gt;115 (nx=4)</td>
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<td>QRSd&gt;120 (nx=1)</td>
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<td>QRSd&gt;120 (nx=2)</td>
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<td>QRSd&gt;120 (nx=3)</td>
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<td>QRSd&gt;120 (nx=4)</td>
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<td>61</td>
<td>87</td>
<td>77</td>
</tr>
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Sens, sensitivity; Spec, specificity; PA, predictive accuracy; QRSd, QRS duration; V40, root-mean-square voltage of the last 40 msec; nx, number of leads with a QRSd exceeding the stated QRSd criteria.
have improved signal-to-noise ratios. 3) Anterior and septal sites of late potentials may have been better detected because the spatial distribution of the array gave a higher density of electrodes (“views”) over the precordium.

Since delayed conduction may exist in several sites in one patient, late potentials may not necessarily be dipolar, and body surface mapping might be expected to be superior to the orthogonal leads. Faugère et al.22 studied the number of extrema in body surface maps of the end of the QRS complex and concluded that late potentials were dipolar and could be well detected with three orthogonal leads. Multiple extrema were described in one patient in their study. The opposite conclusions arrived at between Faugère et al.’s study and ours may be due to several reasons. In contrast to our study, their data were sampled at 500 Hz and digitized with a resolution of 2.5 μV and recorded for only 52 seconds (compared with 1,000 Hz, 1 μV, and 250 seconds, respectively, for our study). Noise was not reported in their study but was reflected in the relatively short mean QRS duration (118±20 msec) and high V40 (43±41 μV) reported for their 21 VT patients. Thus, only 48% of their VT patients had a QRS duration of >120 msec and only 48% of their VT patients had a V40 of <25 μV. In their study, 63 uniformly distributed thoracic leads were used compared with the 28-lead optimal array used in our study. No direct comparison can be drawn between these two arrays. However, in Lux’s study, a uniform 5×6 (30 leads) array was found to be no better than the nine leads used in standard ECG.29

Another attempt to use ECG information from more extensive sites on the torso to identify patients with prior anterior myocardial infarction and VT was reported recently.34 Spectral analysis was performed on the entire QRS complex comprising the root-mean-square of 87 SAECGs recorded from 87 uniformly distributed sites. A reduction in the 40–80 Hz frequency components was suggested to be a marker for VT. Sensitivity and specificity were not quoted, and only four patients had sustained VT among their VT group of 10 patients. No remarkable differences in the patterns of QRS area maps were found between those with and without VT.

Previously reported sensitivity of the SAECG in identifying patients with sustained VT and ischemic heart disease has ranged from 50% to 92%, while specificity has ranged from 62% to 93%.32,33–41 The variation is due to different criteria for late potentials, differences in the VT population as well as control groups, and different definitions of VT. In general, when a shorter QRS duration is used to define late potentials, the sensitivity improves but at a cost of lower specificity. Thus, although the sensitivity achieved by the vector magnitude method in this study may appear low (54%), it was achieved with a relatively high specificity (84%). This is consistent with previous studies where sensitivities of 50–60% were achieved with specificities of around 80–90% while studies describing sensitivities of 80–90% were achieved with specificities of 60–70%.36–42 The control group in our study were postinfarction patients studied just prior to hospital discharge. Moreover, because of physicians’ preferences and referral bias for electrophysiology study for risk

FIGURE 3. Sensitivity plotted against specificity: Combination parameters (QRS duration combined with V40 as the criterion to define late potentials) versus single parameter (QRS duration only). For the combination parameter, the variables V40<20 μV and V40<25 μV were each combined with a single QRS duration (tested in steps of 5 msec from 110 to 125 msec). For the single parameter, QRS duration cut points were identical to those in Figure 2. For each combination of QRS duration with V40, there was an identical result achievable by using only a single QRS duration as the criterion for defining late potentials. QRSd, QRS duration; V40, root-mean-square voltage of the terminal 40 msec.
stratification, there was a tendency for this group to have larger infarcts and higher-grade ventricular ectopy than might otherwise have been the case. This was, therefore, a population in which there was a higher incidence of false-positive SAECGs resulting in a lower specificity than would otherwise have been the case. To obtain the relatively high specificity in our overall study population, there was a compromise in the sensitivity. However, since the postinfarction group before hospital discharge is the target population for whom the SAECG will be of most use, we felt that such a control group was an appropriate "training set."

The cut points in this study were deliberately derived solely from computer results with no overreading so that time-consuming qualitative assessment of all 28 leads individually in each patient would not be required in future prospective studies. Since no overreading or qualitative assessment was applied, it is possible that certain leads would wrongly detect artefacts as late potentials. Moreover, since onsets and offsets were identified by computer algorithms based solely on mean and SD values of the noise levels in the ST segment, it is possible that one out of the 28 leads would be abnormal in a patient with no VT. The criteria for defining late potentials using the optimal array were based on the number of leads where QRS duration exceeded 120 msec. If the number of leads (nx) required to exceed this duration were reduced, the sensitivity would improve and specificity would drop. The arbitrary criterion in this study of three or more leads exceeding a QRS duration of 120 msec to define abnormality appeared to provide a reasonable balance between sensitivity and specificity.

An interesting observation from this study was that the two commonly used criteria to define late potentials—QRS duration and V40—were interdependent. Thus, for each combination of QRS duration with V40, there was an identical result achievable by using only a single QRS duration as the criterion for defining late potentials. This is consistent with the results from recent studies showing that the duration of the filtered QRS complex is the most significant independent predictor of arrhythmic events postmyocardial infarction.9

**Study Limitation**

Conventionally, bipolar leads have been used for the orthogonal array. In this study, all the leads used, including the orthogonal array, were unipolar leads referenced to the Wilson central. In over 200 patients undergoing a single SAECG using simultaneously both the Frank XYZ leads and unipolar XYZ leads referenced to the Wilson central, we found no difference in the total QRS durations obtained from the vector magnitudes of the two lead sets for patients with and without inducible VT, whether a 25-Hz or a 40-Hz bidirectional high-pass filter was used (Ho and Richards, unpublished data). The use of the Frank lead system in SAECG had been described by investigators from our unit and others.31,43–45 Hammill et al44 concluded that diagnostic criteria for SAECG with use of Frank XYZ leads (with a spectral filter in their report) produced results similar to those reported for use of bipolar XYZ leads. Pietersen and Gymoese45 also found no clinical difference between the original Frank leads and two other bipolar orthogonal lead systems. The use of unipolar recordings in the present study is therefore unlikely to have produced significantly different results than would have been obtained if bipolar recordings had been used.

**Conclusions**

Detection of late potentials can be improved by applying body surface mapping in the form of a clinically practical, 28-lead optimal array to the SAECG. This results in a 15% improvement in sensitivity without any loss in specificity. The increased sensitivity was of equal magnitude for patients with prior inferior as well as those with prior anterior infarctions. The superiority and practicality of the 28-lead optimal array make it worth pursuing as an option for further refinement in SAECG.

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