Quantitative analysis of the high-frequency components of the signal-averaged QRS complex in patients with acute myocardial infarction: a prospective study

JOSEPH A. GOMES, M.D., RAHUL MEHRA, PH.D., PHILIP BARRECA, B.S., NABIL EL-SHERIF, M.D., ROBERT HARIMAN, M.D., AND RONALD HOLTZMAN, M.S.

ABSTRACT We performed a prospective study of the high-frequency components of the terminal portion of the QRS complex in 50 patients with acute myocardial infarction (AMI) (mean age 63 ± 10 years) within 3.25 ± 2.45 days of the acute event. Signal averaging (400 beats) at a filter setting of 80 to 300 Hz was performed and the duration of the low-amplitude signals of less than 40 μV in the terminal portion of the QRS, the root-mean-square (RMS) voltage of the terminal 40 msec of the QRS complex, and the total duration of the signal-averaged QRS vector complex were measured. The low-amplitude signals were abnormally prolonged in 22 of 50 patients (44%); the RMS-V was abnormal (<20 μV) in 21 of 50 patients (58%), and the signal-averaged vector complex was abnormal (>120 msec) in 15 of 46 patients (33%) without bundle branch block. There was no significant correlation between any of the signal-averaged parameters and site of AMI or total creatine kinase (CK) and CK-MB values. On the basis of the occurrence of spontaneous ventricular tachycardia in the acute and postcoronary care phase of AMI, the patients were divided into two groups. Group I consisted of 31 patients (62%) who had no documented ventricular tachycardia and group II consisted of 19 patients (38%) who had one or more runs of ventricular tachycardia. Fourteen of the 19 patients in group II (73.6%) had nonsustained ventricular tachycardia and five patients (26.3%) suffered sustained ventricular tachycardia/ventricular fibrillation or sudden death. The low-amplitude signals and the signal-averaged QRS complex duration were significantly longer in group II than group I. The RMS voltage was significantly lower in group II than group I. In addition, in five of the patients (10%) who had sustained ventricular tachycardia/fibrillation and/or sudden death the low-amplitude signals were 50 msec or greater and the RMS voltage was 13 μV or less; the signal-averaged QRS vector complex was greater than 120 msec in four of these patients. We conclude the following: (1) Abnormal signal-averaged parameters are seen in 33% to 58% of patients with AMI. (2) There is no correlation between any of the signal-averaged parameters and site of AMI or CK and CK-MB values. (3) Low-amplitude signals and the duration of the signal-averaged QRS complex are significantly longer and the RMS voltage significantly lower in patients with AMI who have spontaneous ventricular tachycardia in the acute and postcoronary care phase of AMI. (4) Signal-averaged parameters may be valuable in predicting arrhythmic events in patients with AMI.

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EXPERIMENTAL AND CLINICAL STUDIES In recent years have demonstrated that reentry plays a major role in the genesis of most ventricular arrhythmias.1-11 The conditions that favor the occurrence of some reentry are slow conduction, unidirectional block, and recovery of excitability. The demonstration of delayed fractionated electrical activity during diastole in regions of experimental myocardial infarction and in patients with ventricular tachycardia and left ventricular aneurysm during endocardial catheterization and intraoperative mapping has provided evidence for slow conduction in the ventricular myocardium. Furthermore, delayed fractionated electrical activity in diastole has been closely correlated with the genesis of reentrant ventricular arrhythmias.2, 3, 6, 10 Recently, several investigators using high-gain amplification and signal-averaging techniques demonstrated diastolic...
electrical activity in experimental myocardial infarction and in patients with recurrent ventricular tachycardia and left ventricular aneurysm. Furthermore, Simson et al., using quantitative analysis, have demonstrated low-amplitude signals in the terminal portion of the QRS complex in patients with recurrent sustained ventricular tachycardia.

Most previous signal-averaging studies have been done in patients with recurrent ventricular tachycardia with or without ventricular aneurysm and in dogs with experimental acute myocardial infarction. The purpose of this prospective study was (1) to assess the occurrence of low-amplitude signals in the terminal portion of the QRS complex from body surface recordings in patients with an acute myocardial infarction (AMI), (2) to determine whether the occurrence of low-amplitude signals is related to the size and location of the infarct, and (3) to attempt to correlate the occurrence of low-amplitude signals with the occurrence of spontaneous ventricular tachyarrhythmias in the early phase of myocardial infarction.

Materials and methods
A total of 50 male patients with AMI were studied. In all patients the diagnosis of AMI was confirmed by (1) clinical, (2) electrocardiographic, and (3) total creatinine kinase (CK) and CK-MB values. Signal averaging was not attempted in patients in pulmonary edema or cardiogenic shock. In addition a total of 20 normal male subjects underwent signal averaging of the body surface QRS complex for determination of normal signal-averaged parameters.

Signal-averaging technique. All patients with AMI underwent signal averaging within 3.25 ± 2.45 days (mean ± SD, range 1 to 10 days) of the acute event. None of the patients were on antiarrhythmic agents during the signal-averaging procedure and none had arrhythmias, including ventricular ectopic activity, during the signal-averaging procedure. In those patients who had been receiving antiarrhythmic therapy, including prophylactic lidocaine, these agents were withheld for at least five half-lives before the signal-averaging procedure. Signal averaging was done with the subjects lying comfortably supine in an “electrically quiet” but not specifically shielded environment after the experimental nature of the procedure had been explained to them and they had given informed consent. After each subject’s skin was prepared with a mildly abrasive pad and washed with alcohol, seven self-adhesive silver/silver chloride electrodes were attached: the horizontal (X) electrodes in the right and left midaxillary lines at the fourth intercostal space, the vertical (Y) electrodes on the suprasternal notch and V6 position, and the sagittal (Z) electrodes in the V3 position anteriorly and at the corresponding position posteriorly. The seventh, indifferent, electrode was placed on the eighth rib in the right midaxillary line. The signal-processing system, hardware, and methods used in our laboratory have been described in detail previously. After connecting the electrodes to the corresponding preamplifier inputs the three low-gain outputs (gain of 1000) were viewed simultaneously to select the channel with the largest QRS complex for triggering the signal averager. Bipolar X, Y, and Z lead signals were then filtered at filter frequencies of 80 to 300 (24 dB/octave) Hz and then underwent further (gain 250) amplification before being digitized and averaged with a 3001 Norland signal-processing system. Approximately 400 beats were signal averaged. Ten-bit resolution was used for analog-to-digital conversion. The data were stored on magnetic diskettes for subsequent measurement and photography.

The occurrence of ventricular arrhythmias in the coronary care unit was assessed by use of a Hewlett-Packard model 5600A Patient Management System with an arrhythmia monitoring option 78220A/B, which provides a special 9 hr trend display. The system has recall capabilities over 24 hr and editing and monitor strip recording capabilities over 9 hr trend display. Human validation of the rhythm disturbances on the monitoring system was done for every patient by the nursing staff using recall and editing capabilities and the rhythm was confirmed by the senior investigator. In the postcoronary care phase a 24 hr ambulatory Holter recording was obtained within 24 hr of the signal-averaging procedure. All patients were monitored in the coronary care unit for a period of 3 to 5 days and in the intermediate coronary care unit for an additional 10 days.

Definition of terms and calculations. A vector magnitude was calculated for each point of the averaged wave as

\[ V = \sqrt{X^2 + Y^2 + Z^2} \]

The duration of the low-amplitude signals was measured from the end of the signal-averaged QRS vector complex to the first point at which signals reached 40 μV. The end of the QRS vector complex was defined as the point at which the signals were more than twice the peak-to-peak noise amplitude as measured from the TP interval. Root-mean-square (RMS) voltage was measured for the terminal 40 msec of the QRS vector complex. The signal-averaged QRS vector complex was measured from the onset to the end of the QRS vector complex. All signal-averaged parameters were measured with a Norland 3001 computer. Electrocadiographic QRS complex was measured on the surface electrocardiogram obtained on the same day and within 1 hr of the signal-averaging procedure. Nonsustained ventricular tachycardia was defined as 3 or more successive beats of ventricular origin at a rate of 120 beats/min or greater that lasted for 30 sec or less and terminated spontaneously. Sustained ventricular tachycardia was defined as spontaneous tachycardia of ventricular origin that lasted for more than 30 sec or was associated with hemodynamic compromise. Sudden cardiac death was defined as death occurring within 1 hr of the onset of symptoms. Statistical analysis of the data was done by use of the χ2 and Student’s t test for unpaired and paired data. All data are expressed as mean ± SD.

Results
Clinical features. The patients with AMI ranged in age from 45 to 83 years (mean 63 ± 10 years). Twenty-four patients (48%) had an anterior wall AMI, 21 patients (42%) had an inferior wall AMI, and five patients (10%) had a subendocardial AMI. A total of four patients had bundle branch block. In these patients the duration of the electrocardiographic QRS complex and the signal-averaged QRS vector complex was not used to calculate the means and differentiate abnormal from normal values.

Signal-averaged parameters
Normal subjects. Normal ranges of values for signal-averaged parameters were determined by performing signal-averaging studies in 20 male subjects (age 32 ± 6 years) with no evidence of ischemic or other forms of
organic heart disease. The duration of low-amplitude signals ranged from 7 to 37 msec and were less than 42 msec in all subjects (100%). The RMS voltage of the terminal 40 msec ranged from 14.1 to 81 μV and was greater than 20 μV in 19 of 20 subjects (95%). The signal-averaged QRS duration ranged from 76 to 103 msec and was less than 120 msec in all 20 subjects (100%).

**Patients with AMI.** The low-amplitude signals ranged from 11.5 to 98.5 msec (mean 42 ± 10 msec) in duration in the patients. Twenty-two of 50 patients (44%) had abnormally prolonged low-amplitude signals of greater than 42 msec (upper limit of normal for our laboratory +2 SDs). The RMS voltage of the terminal 40 msec of the signal-averaged complex QRS vector ranged from 2.38 to 68.2 μV (mean 24 ± 17 μV). Twenty-one of 50 patients (58%) had abnormal RMS voltage of less than 20 μV (upper limit of normal for our laboratory). The duration of the signal-averaged QRS vector complex ranged from 91 msec to 144.5 msec (mean 114 ± 16 msec) in patients without bundle branch block and was significantly longer (p < .0001) than the electrocardiographic QRS complex duration, which measured 95 ± 10 msec. In 15 of 46 patients (33%) without bundle branch block the mean signal-averaged QRS vector complex was greater than 120 msec.

**Correlation between signal-averaged parameters and other clinical variables.** There was no significant correlation between total CK and low-amplitude signals (r = .218), total CK and RMS voltage (r = .0915), or total CK and signal-averaged QRS vector complex (r = .206). No correlation was found between peak CK-MB fraction and low-amplitude signals (r = .249), peak CK-MB fraction and RMS voltage (r = .136), or peak CK-MB fraction and the signal-averaged vector complexes (r = .165). Similarly, no correlation was noted between the day of study, site of AMI, and the various signal-averaged parameters.

**Ventricular tachyarrhythmias.** On the basis of the occurrence of spontaneous ventricular tachyarrhythmias in the acute and immediate postcoronary care phase of AMI the patients were divided into two groups. Group I consisted of 31 patients (62%) who had no documented ventricular tachycardia and group II of 19 patients (38%) who had one or more runs of spontaneous ventricular tachycardia. Three patients from group I and one patient from group II had bundle branch block. Of the 19 patients in group II, 14 (73.6%) had nonsustained ventricular tachycardia and five (26.3%) suffered sustained ventricular tachycardia/ventricular fibrillation and/or sudden cardiac death. In 11 of the 19 patients in group II, ventricular arrhythmias occurred 2 to 30 days (mean 11 ± 9 days) after the signal-averaging procedure; in six patients the ventricular arrhythmias occurred 1 to 3 days (mean 2.0 ± 1.0 days) before the procedure and in two ventricular arrhythmias were noted on the day of the procedure. In 12 of the 19 patients (63%) the ventricular arrhythmias were noted after the first 3 days of AMI, whereas in the remaining seven patients (37%) arrhythmias were noted within the first 3 days after AMI. Of the five patients who developed sustained ventricular tachycardia, fibrillation, and/or sudden death, ventricular arrhythmias occurred on days 2, 15, 15, and 30 after the signal-averaging procedure in four patients and 3 days before the procedure in the remaining patient. Ventricular arrhythmias occurred within the first 3 days of AMI in only one of these five patients. None of the patients were on-antiarrhythmic therapy when they experienced spontaneous ventricular arrhythmias.

**Clinical differences between the two groups.** Table 1 demonstrates the clinical differences between the two groups. There were no significant differences with respect to age, day of study, peak total CK and CK-MB, or site of AMI, although the mean values for total peak CK and CK-MB and the incidence of anterior wall AMI was slightly higher in group II than in group I patients. The incidence of inferior wall and subendocardial AMI was higher in group I when compared with group II, but these differences did not reach statistical significance.

**Differences in signal-averaged parameters in the two groups.** The data on the signal-averaged parameters for

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**TABLE 1**  
Clinical features of patients in the two study groups

<table>
<thead>
<tr>
<th>Group</th>
<th>Age (yr)</th>
<th>Day of study (days after AMI)</th>
<th>CK (IU/l)</th>
<th>CK-MB</th>
<th>Site of AMI (% of patients)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>AWMI</td>
</tr>
<tr>
<td>I</td>
<td>63 ± 11</td>
<td>3.25 ± 2.42</td>
<td>1498 ± 752</td>
<td>182 ± 129</td>
<td>42</td>
</tr>
<tr>
<td>II</td>
<td>63 ± 8</td>
<td>3.52 ± 2.66</td>
<td>1527 ± 894</td>
<td>201 ± 130</td>
<td>53</td>
</tr>
<tr>
<td>p value</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

AWMI = anterior wall MI; IWI = inferior wall MI; SEMI = subendocardial MI.
the two groups are shown in table 2 and figure 1. The low-amplitude signals were significantly longer (p < .01) in group II compared with group I. Whereas 10 of 31 patients (32%) from group I had low-amplitude signals of greater than 42 msec, 12 of 19 patients (63%) from group II had signals this long. The RMS voltage of the terminal 40 msec of the signal-averaged QRS vector complex was significantly lower (p < .05) in group II than group I. Fifteen of the 31 patients (48%) in group I had an RMS voltage less than 20 μV whereas 16 of the 19 patients (84%) in group II had an RMS voltage less than 20 μV. The signal-averaged QRS vector complex was significantly longer (p < .05) in group II than group I. Whereas five of the 28 patients (18%) from group I without bundle branch block had a QRS vector complex of greater than 120 msec in duration, eight of the 18 patients (44%) in group II without bundle branch block had a QRS vector complex greater than this. Examples of data from patients in groups I and II are shown in figures 2 and 3. All the five patients with sustained ventricular tachycardia/fibrillation and/or sudden death had low-amplitude signals of greater than 42 msec; all also had an RMS voltage of < 20 μV in the terminal 40 msec and four of five patients had a signal-averaged QRS vector complex of greater than 120 msec. An example from one of these patients is shown in figure 3. This patient underwent signal averaging 4 days after AMI.

**TABLE 2**

<table>
<thead>
<tr>
<th>Signal-averaged parameters</th>
<th>Group</th>
<th>LAS (msec)</th>
<th>RMS-voltage (μV)</th>
<th>Signal-averaged QRS complex duration (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>I</td>
<td>30.42 ± 9.8</td>
<td>26.91 ± 15</td>
<td>109 ± 12</td>
</tr>
<tr>
<td></td>
<td>II</td>
<td>50.31 ± 24.1</td>
<td>17.66 ± 15</td>
<td>120 ± 19</td>
</tr>
<tr>
<td>p value</td>
<td>&lt;.01</td>
<td>.05</td>
<td>&lt;.05</td>
<td></td>
</tr>
</tbody>
</table>

LAS = low-amplitude signals.

**FIGURE 1.** Individual signal-averaged QRS complex parameters for patients in groups I and II. A. Distribution of low-amplitude signals in group I (solid circle) and group II (open circle). B. Distribution of RMS voltages in group I (solid circle) and group II (open circle). C. Distribution of durations of signal-averaged QRS vector complexes in group I (solid circle) and group II (open circle). Data from patients with sustained ventricular tachycardia/fibrillation are represented by open triangles (group II).

**FIGURE 2.** Signal-averaged QRS complex obtained 1 day after AMI in a patient in group I. The amplitude of the last 40 msec was 54.32 μV, the duration of the low-amplitude signal was 17 msec (hatched area), and the signal-averaged vector complex was 96 msec.

**FIGURE 3.** Signal-averaged QRS complex obtained 4 days after AMI in a patient in group II who 2 days later developed sustained ventricular tachycardia. The low-amplitude signal (hatched area) was 56 msec in duration, the RMS voltage was 13.05 μV, and the signal-averaged vector complex was 141 msec in duration.
and the results revealed abnormal parameters: low-amplitude signals were 56 msec in duration, the RMS voltage of the terminal 40 msec of the QRS complex was 13.05 μV, and the signal-averaged QRS vector magnitude was 141 msec. Two days after discharge from the coronary care unit the patient complained of palpitations and dizziness associated with profuse diaphoresis. An electrocardiogram was recorded at this time and revealed sustained ventricular tachycardia.

Specificity and sensitivity of signal-averaged parameters. The sensitivity and specificity of signal-averaged parameters in separating patients without ventricular arrhythmias (group I) from those with ventricular tachycardia (group II) and in separating patients with sustained ventricular tachycardia/fibrillation and or sudden death from those without are shown in table 3. The sensitivity and specificity of signal-averaged parameters ranged from 44% to 84% and from 52% to 84%, respectively, in separating patients with from those without ventricular tachyarrhythmias. The sensitivity and specificity of signal-averaged parameters ranged from 80% to 100% and from 42% to 88%, respectively, in separating patients with sustained ventricular tachycardia/fibrillation and/or sudden death from those without.

Discussion

In recent years several investigators have attempted to record late diastolic potentials with high-gain amplification and signal averaging in patients with ventricular tachycardia and with and without left ventricular aneurysm. These studies have demonstrated a high prevalence of late diastolic potentials in patients with recurrent sustained ventricular tachycardia. In addition a good correlation has been noted between the presence of late potentials and left ventricular wall motion abnormalities. The majority of these studies, however, have assessed the presence of late potentials qualitatively. Qualitative assessment of late potentials can be fraught with significant error since it is often difficult to decipher the end of the signal-averaged QRS complex and the onset of the late potentials. To obviate these limitations, Simson proposed quantitative analysis of the high-frequency components of the terminal portion of the QRS complex by computer analysis. Using a 25 Hz filter, Simson observed that 92% of the patients with recurrent ventricular tachycardia had less than 25 μV of high-frequency voltage in the last 40 msec of the QRS complex and in 72% of the patients the QRS duration was longer than 120 msec. Denes et al. have also recently demonstrated that high-frequency analysis of the signal-averaged body surface QRS is a reliable, reproducible method for distinguishing patients with recurrent ventricular tachycardia from those without it. Using a 40 Hz filter and a definition of normal of greater than 20 μV for the amplitude of the last 40 msec, 90% of patients without ventricular tachycardia and 83% of patients with ventricular tachycardia were correctly identified. Furthermore, these investigators found that the use of a 40 Hz filter markedly improved the separation between those with and without ventricular tachycardia and reduced the degree of variability resulting from use of a 25 Hz filter. Other investigators have used 100 Hz filtering. In contrast to the method of Simson and Denes et al., we used 80 Hz filtering to record low-amplitude signals. Although our method is not comparable to that of Simson or Denes et al., we, like the latter investigators, who used 40 Hz filtering, found that use of 20 μV as the normal value for amplitude of the last 40 msec resulted in good separation of normal from abnormal values. Simson, on the other hand, using 25 Hz filtering, found that use of 25 μV provided a separation between patients with and without ventricular tachycardia. Since the optimal bandpass is still unknown, in the future standardization of the use of specific filter frequencies will be needed for comparative purposes.

Myocardial infarction in man offers a unique setting in which to study the high-frequency components of the terminal portion of the signal-averaged QRS complex, to detect abnormal low-amplitude signals, and to determine their significance because the occurrence of these signals and clinically significant ventricular arrhythmias in the acute, subacute, and chronic phase of myocardial infarction can be correlated. In this study abnormally prolonged low-amplitude signals of greater than 42 msec in duration were seen in 44% of patients with an AMI. In addition, the low-amplitude signals in the terminal portion of the QRS vector com-

**TABLE 3**

**Sensitivity and specificity of signal-averaged parameters**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Group I vs group II</th>
<th>Sustained VT/VF/SD vs no sustained VT/VF/SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low-amplitude signals</td>
<td>63</td>
<td>84</td>
</tr>
<tr>
<td>RMS Voltage</td>
<td>68</td>
<td>52</td>
</tr>
<tr>
<td>Signal-averaged QRS vector duration</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td></td>
<td>102</td>
<td>102</td>
</tr>
</tbody>
</table>

**TABLE 3**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low-amplitude signals</td>
<td>63</td>
<td>68</td>
<td>100</td>
<td>62</td>
</tr>
<tr>
<td>RMS Voltage</td>
<td>84</td>
<td>52</td>
<td>100</td>
<td>42</td>
</tr>
<tr>
<td>Signal-averaged QRS vector duration</td>
<td>44</td>
<td>82</td>
<td>80</td>
<td>88</td>
</tr>
</tbody>
</table>

VT = ventricular tachycardia; VF = ventricular fibrillation; SD = sudden death.
plex were less than 20 μV in 58% of patients with AMI. These observations probably reflect regional slow ventricular activation in the border zone of an acute infarct. In 33% of patients the signal-averaged QRS vector complex was greater than 120 msec when the surface electrocardiogram was normal in duration. Again, this observation probably reflects delay in ventricular activation since in this study the delayed potentials were assessed as part of the signal-averaged QRS vector complex rather than as a separate entity. It is not unreasonable to expect abnormal signal-averaged QRS complexes in patients with AMI. However, the detection of these abnormalities may be dependent on one or more of the following factors: (1) The size of the ischemic mass of ventricular myocardium in the border zone of the infarct, rather than the location or the size of the infarct. The latter is suggested by the observation that there was no correlation between any of the signal-averaged parameters and the location of the infarct or the peak total CK and CK-MB values. These findings are in agreement with the preliminary observations of Abdollah et al.22 (2) Heart rate. Whereas late potentials may not be evident at resting heart rates, they may be evident and more pronounced during atrial pacing at rapid heart rates.12 (3) The occurrence of Wenckebach-like conduction block in one or more areas of the ischemic border zone. This block may not be detectable by signal-averaging techniques.6,21

Of considerable importance was our finding that the duration of the low-amplitude signals and the signal-averaged QRS vector complex was significantly longer, whereas the RMS voltage of the terminal 40 msec of the QRS complex was significantly lower, in patients with AMI and ventricular tachyarrhythmias, in contrast to those without tachyarrhythmias. Furthermore, in five patients with sustained ventricular tachycardia/fibrillation and/or sudden death the RMS voltage of the terminal 40 msec was less than 13 μV, the low-amplitude signals were 50 msec or more, and, in four of the five, the signal-averaged QRS vector complex was more than 120 msec. These observations suggest that patients with AMI who have ventricular tachyarrhythmias, particularly sustained ventricular tachycardia/fibrillation and/or sudden death, in the early phase of myocardial infarction demonstrate a greater degree of ventricular myocardial conduction delay, which could provide the substrate for reentrant ventricular tachyarrhythmias. These findings are in disagreement with the preliminary observations of Abdollah et al.,23 who found that low-amplitude signals failed to predict the occurrence of in-hospital ventricular tachycardia in patients with AMI. However, it should be noted that whereas approximately 44% to 84% of our patients with ventricular arrhythmias had one or more abnormal signal-averaged parameter values, 18% to 48% of our patients without ventricular arrhythmias had one or more abnormal values. Thus, the sensitivity (42% to 84%) and specificity (52% to 84%) of signal-averaged parameters were not strong in separating patients with ventricular arrhythmias from those without. However, when patients with sustained ventricular tachycardia/fibrillation and/or sudden death were compared with those without, low-amplitude signals of greater than 42 msec and RMS voltage of less than 20 μV were the most sensitive (100% each) but lacked specificity (62% and 42%, respectively), whereas the duration of the signal-averaged QRS complex was both specific (88%) and sensitive (80%). The low specificity of the duration of low-amplitude signals and the RMS voltage of the terminal 40 msec of the QRS complex may be related to the following: (1) the presence of delayed ventricular activation in ischemic myocardium that was not sufficient to result in reentrant ventricular arrhythmias, (2) the presence of delayed ventricular activation within areas of exit block, and (3) a mechanism of ventricular arrhythmias other than reentry (e.g., triggered automaticity).24,25 In the last case the presence of delayed ventricular activation would be independent of the spontaneous ventricular arrhythmia.

A preliminary report by Haerten et al.26 suggested that the presence of late potentials and inducibility of repetitive ventricular responses help to identify patients at risk of sudden death or ventricular tachycardia after myocardial infarction. More recently Kanovsky et al.27 found that the signal-averaged electrocardiogram provided independent information in identifying patients with ventricular tachycardia after myocardial infarction. However, their study consisted of patients who were referred for electrophysiologic studies because of repeated episodes of ventricular tachycardia. They did not prospectively follow patients with AMI to determine whether signal averaging identified a group of patients at future risk of developing ventricular tachycardia and/or sudden death. Thus, the role of signal-averaging techniques in predicting future arrhythmic events in the postmyocardial infarction patient remains to be defined. Long-term prospective follow-up and sequential signal-averaging studies in a large number of postinfarction patients will be needed before this important and crucial question can be answered since the dynamic nature of myocardial infarction and the healing process could result in changes in late potential parameters.
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