Use of Signals in the Terminal QRS Complex to Identify Patients with Ventricular Tachycardia After Myocardial Infarction

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SUMMARY Small, high-frequency electrocardiographic signals were recorded from the body surface in 39 patients with and 27 patients without ventricular tachycardia (VT). All patients were in normal sinus rhythm, had a previous myocardial infarction, were not taking antiarrhythmic drugs, and did not have bundle branch block. Bipolar X, Y, Z leads were signal averaged and processed by a bidirectional digital filter that allowed low-amplitude signals to be detected in the terminal QRS complex and ST segment. The high-pass filter frequency was 25 Hz.

Patients with VT had a lower amplitude of high-frequency signal in the late QRS complex. In the last 40 msec of the filtered QRS complex, the patients with VT had 14.9 ± 14.4 μV of high-frequency signal; patients without VT had 73.8 ± 47.7 μV (p < 0.0001). Ninety-two percent of the patients with VT had less than 25 μV of high-frequency voltage; only 7% of patients without VT had less than 25 μV (p < 0.0001).

Patients with VT had a longer QRS duration than those without VT, 139 ± 26 vs 95 ± 10 msec (p < 0.0001). The QRS duration was longer than 120 msec in 72% of the patients with VT but in none of the patients without VT (p < 0.0001). In all patients there was no separate and discrete high-frequency signal in the ST segment. Advanced signal processing of the ECG accurately identified the patients in the study with VT after myocardial infarction.

THE SURFACE ECG gives few clues to warn that a patient may develop ventricular tachycardia (VT). Recently, several groups, using special processing techniques, have detected small, high-frequency potentials in the late QRS complex and ST segment in patients and animals prone to VT.1-3 These signals appear to arise from slowly conducting areas of the myocardium.2 Many observers have recorded delayed and disorganized activation directly from infarcted myocardium,4-14 and slow conduction through infarcted tissue is related to reentrant ventricular arrhythmias.6,15

High-frequency, low-amplitude signals from the body surface were studied in two groups of patients after myocardial infarction (MI): a control group without complex ventricular arrhythmias and a group with sustained and inducible VT. Signal averaging15,16 and a bidirectional digital filter were used, which allowed small, high-frequency signals to be detected in the terminal QRS complex. We found a noninvasive measurement that accurately identified patients with VT.

Methods

Patients

Each patient had a documented transmural MI that was more than 2 weeks old. None had left ventricular hypertrophy or right or left bundle branch block on the standard ECG. Each was in normal sinus rhythm and had not received antiarrhythmic drugs for at least 24 hours.

The control group consisted of 27 patients without VT who had no clinical history of complex ventricular arrhythmias. A 24-hour ECG, performed within 2 days of study, showed less than 200 premature ventricular complexes (PVCs) per day and the absence of multiform PVCs, couplets or VT. Ventriculography was performed in 19 patients and showed a ventricular aneurysm in four.

The VT group included 39 patients who had had repeated episodes of symptomatic VT. All VT patients were studied in the clinical electrophysiologic laboratory with previously described techniques17 and had sustained (> 1 minute) VT inducible by one to three VPCs. All patients with VT had ventriculography.

There was no difference in location of infarcts between groups; 14 patients in the control group and 15 patients in the VT group had anterior MI and the remainder had inferior MI. Eighteen of the 39 VT patients and four of the 27 control patients had ventricular aneurysms (p = 0.06). Seventeen control patients and 10 VT patients had infarctions within 3 months (p = 0.003). Thirteen control patients and no VT patients were taking propranolol (p < 0.0001). The incidence of left anterior hemiblock was similar in both groups (two in the control group and five in the VT group) (p = 0.39).

Surface Recording

Bipolar X, Y, Z leads were used. The X lead was between the right and left midaxillary lines at the fourth intercostal space. The Y electrodes were placed at the superior aspect of the manubrium and the proximal left leg. The anterior Z electrode was at the V₃ position and the other was at the identical position on the posterior chest. Positive electrodes were left, in-
sferior and anterior. Recordings were made at the patient's bedside and took approximately 12 minutes. All patients gave informed consent.

**Analogue Signal Processing**

The three-channel amplifier was custom-built and based on an Analogue Devices Model 283J isolation amplifier. The gain was 1000, bandwidth was 0.05–300 Hz and noise was 0.7 µV, referred to input. The common mode rejection ratio was 140 dB.

The signal from each lead was amplified two to five times, passed through a four-pole, 250-Hz, low-pass filter, and then AD-converted to 12-bit accuracy (Analogue Devices AD572) at 1000 samples/sec. Gain was adjusted so that the least significant bit represented 1 or 2.5 µV. The digital information was stored on tape by a Hewlett-Packard 9825 desktop computer. Each lead was sequentially recorded for 133 seconds.

One lead, usually the Z lead, served as a reference for all processing. After bandpassing the signal (two-pole filters, 8-40 Hz), an adjustable comparator set a reference or fiducial bit. Reference jitter was ±0.5 msec, as determined by measuring noise on a portion of the wave form with a steep slope.

**Signal Averaging**

The ECG signals were averaged after passing through a template recognition program to reject ectopic beats and grossly noisy signals. An eight-point template began at the reference time and extended for 128 msec, spanning the distal QRS complex and early ST segment. An initial eight-beat template was accepted if the mean standard deviation, after DC level shifting, was less than 20 µV. All subsequent beats were tested against the template and accepted if the deviation from the template was less than twice the template standard deviation. If so, a 512-msec segment of the beat was averaged, beginning 100 msec before the QRS complex. The template was updated every fourth beat. The template program rejected 0–2% of normal sinus beats and reliably rejected ectopic beats. The noise level of the signal-averaged signal was measured every 8 msec to test for jitter. The signal-averaged leads were recorded on floppy disc memory for further processing. In all, 154 ± 16 beats per lead were signal averaged.

**Digital Filter**

Each averaged lead was then filtered to eliminate low frequencies contained in the QRS complex and ST segments. A bidirectional digital filter was developed to eliminate impulse ringing of the filter. The filter processed forward in time until 40 msec into the QRS complex. The filter was then reset and processed the signal backward in time up to the same point in the QRS complex. By this technique, ringing of the filter after the QRS complex was eliminated.

The digital filter was a four-pole, high-pass Butterworth design obtained by the bilinear mapping technique with frequency prewarping. (Neil Judell, Optimal Systems Lab, North Kingstown, Rhode Island, developed the filter algorithm and it was adapted by the author to work in a bidirectional mode.) The filter corner frequency was 25 Hz (−3 dB) except as noted.

The filtered signals for the three leads were combined into a vector magnitude, \( \sqrt{x^2 + y^2 + z^2} \), which allowed the detection of high-frequency voltage in any lead. The vector magnitude of the filtered signals is referred to as filter output or filtered QRS complex in this study.

Figure 1 illustrates filtering of a test signal. The filter ran unidirectionally and forward in time. The filter rings after the test signal ends, and the peak amplitude of ringing remains above 1 µV for 88 msec (fig. 1A). With the bidirectional filter there is no ringing or filter artifact after the test signal (fig. 1B).

**Analysis**

QRS end points were determined by computer algorithm. The computer measured a noise sample and then searched forward or backward for a 5-msec segment where the average exceeded the mean plus 3 standard deviations of the noise sample. The midpoint of the 5-msec segment was called the end point. The noise sample for the QRS onset was 20 msec wide and began about 50 msec before QRS onset. The noise sample for the end of the QRS was 40 msec wide and began about 60 msec after the QRS. Intraobserver agreement between computer and two observers was ±1.2 msec (standard deviation of the difference). The filter output was plotted with a scale of 15 µV/cm and 25 msec/cm.

Voltages are the root mean square (RMS) in microvolts; the RMS voltage is the square root of the mean square voltage over an interval. Noise level was measured during the ST segment over an 80-msec sample. Voltage (RMS) for the last 40 msec of the QRS complex was separately calculated; 40 msec was chosen as the interval because it is the period of a 25-Hz wave form. Data are presented as the mean ± SD. Statistical analysis was performed with the unpaired t test and the Fisher exact test.

Time histograms of the filter output were compiled with 10-msec cell widths. The RMS voltage was calculated for each cell for each patient. One histogram was aligned with the beginning of the QRS complex, and another was aligned with the end of the QRS complex.

For the unfiltered leads, notching was defined as a wave form greater than 2 µV peak to peak, with at least two areas where the slope equaled 0 less than 30 msec apart.

**Results**

Figure 2 illustrates examples of signal processing from both groups. The patient with VT had a low-amplitude signal in the last 40 msec of the filtered QRS complex (arrow), which was not seen in the filter output from the patient without VT. QRS duration
was 106 msec in both examples. No ringing of the filter occurred after the QRS complex and there was no high-frequency signal other than random noise in the ST segment in both examples. Low-level, high-frequency components were associated with the peak of the T wave (fig. 2A).

Figures 3 and 4 are examples of signal processing in patients with anterior and inferior MIs. There was excellent agreement between the filter output and signal-averaged leads with respect to QRS duration. Thirty patients with VT had notches on the last 40 msec of the unfiltered leads (fig. 4B), but this finding was also present in 12 patients of the control group (p = 0.007).

To study the time course of high-frequency voltage in the QRS complex, histograms of the filtered QRS voltage were compiled (fig. 5). In the control group, the filtered signal peaked at 40–50 msec and declined to noise level at 110 msec. Although lower, the filtered signal from the VT group paralleled that from the control group until 50–60 msec; after that, the filtered signal in patients with VT exceeded that in controls until 160 msec (p < 0.0001).

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Histograms were aligned with the end of the QRS complex (fig. 5B) to observe the time course of high-frequency voltage in the terminal QRS complex, independent of QRS duration. In the control group, the
high-frequency voltage peaks 50–60 msec before the end of the QRS complex and declines abruptly. In the VT group, the high-frequency voltage is lower and declines more slowly to the end of the QRS complex. No signal above noise is present in either group up to 80 msec beyond the end of the QRS complex; ST-segment noise level on the filter output was similar in both groups, 0.8 \( \mu \text{V} \pm 0.4 \mu \text{V} \).

The QRS duration was longer in patients with VT, 139 ± 26 vs 95 ± 10 msec \( (p < 0.0001) \) (fig. 6). Seventy-two percent of the VT patients had a QRS duration longer than 120 msec, but no patient in the control group did \( (p < 0.0001) \).

The patients with VT had low-amplitude signals at the end of the filtered QRS complex (figs. 2–5). The voltage in the last 40 msec of the filtered QRS complex was found to discriminate well between the two groups (fig. 7A). The patients with VT had 14.9 ± 14.4 \( \mu \text{V} \) of high-frequency signal in this segment; in contrast, patients without VT had 73.8 ± 47.7 \( \mu \text{V} \) \( (p < 0.0001) \).

Twenty-five microvolts could be used as a threshold to discriminate the two groups. Only three patients with VT exceeded this level; all but two of the control group exceeded 25 \( \mu \text{V} \) \( (p < 0.0001) \).

The filtered QRS voltage tended to be lower in patients with VT than in the control group, 103 ± 30 vs 127 ± 43 \( \mu \text{V} \) \( (p = 0.10) \). The low amplitude of

**Figure 3.** Signal processing in patients with anterior myocardial infarctions. The QRS duration is longer in the patient with ventricular tachycardia (122 msec) than the control patient (102 msec). A low-level signal (arrow) is seen late in the filtered QRS complex of the patient with ventricular tachycardia.

**Figure 4.** Signal processing in patients with inferior myocardial infarctions. The QRS duration is longer in the patient with ventricular tachycardia (135 msec) than in the patient without ventricular tachycardia (99 msec). A low-amplitude, high-frequency signal (arrow) is again present at the end of the filtered QRS complex in the patient with ventricular tachycardia.
filtered signal in the terminal QRS of VT patients, however, was not a consequence of the lower total filtered QRS voltage (fig. 7B). The difference between the groups remained distinct \( p < 0.0001 \).

A significant difference in voltage in the last 40 msec of the filtered QRS complex remained between control and VT groups when patients were separately analyzed according to anterior MI, inferior MI, QRS duration less than 120 msec, MI older than 3 months, no aneurysm or no propranolol therapy (table 1). Also, in every subgroup the QRS duration was longer in patients with VT (each \( p < 0.002 \)).

To determine if the high-frequency signal could localize an infarct, the filtered X, Y and Z leads were analyzed separately for the anterior and inferior infarctions in the VT group. No significant differences in high-frequency voltage were found between anterior and inferior infarction for each lead \( p > 0.15 \). For the VT patients, the ratio of voltage in the last 40 msec of filtered QRS complex in the X, Y and Z leads was 1:1.5:1.3.

Data from all patients were reprocessed using 50- and 100-Hz filter frequencies. The voltage in the last 40 msec of the filtered QRS remained significantly different between the control and VT group at the two higher frequencies \( p < 0.0005 \). The voltage decreased with higher filter frequencies; for the VT group, the signal in the terminal QRS was 15.2 \( \mu \)V at 25 Hz, 7.5 \( \mu \)V at 50 Hz, and 2.7 \( \mu \)V at 100 Hz. The signal-to-noise voltage ratio declined at higher filter frequency; at 25 Hz the ratio was 25 db and at 100 Hz it was 17 db \( p < 0.001 \).

**Discussion**

This study demonstrates that signal processing of the ECG by computer can separate patients with VT from patients without complex ventricular ectopy.

**Figure 5.** Time histograms of the mean filtered QRS voltages for both groups. (A) The histograms were aligned with the start of the QRS complex. The ventricular tachycardia (VT) group (dashed line) had more high-frequency voltage from 50-160 msec compared with the control group (solid line). (B) The histograms were aligned with the end of the QRS complex. The control group has an abrupt decline of high-frequency voltage during the last 40 msec of the QRS complex. The VT group had less high-frequency voltage at the end of the QRS complex and the signal declined more slowly. No high-frequency signal above noise level is apparent beyond the end of the QRS in both groups.

**Figure 6.** QRS duration for the control and ventricular tachycardia (VT) groups. The dashed lines are the means.
after MI. Two computer-based techniques were used to analyze the signals from the body surface: signal averaging and a digital filter.

**Signal Averaging**

Signal averaging reduces random noise in the ECG and enhances the detection of low-amplitude signals. A mean of 154 beats per lead was averaged and theoretical noise reduction was 12.4 times. An important signal, found in the last 40 msec of the filtered QRS complex in patients with VT, was only 15 µV. Without signal averaging, the estimated noise level would have been 10 µV and would not have allowed reliable detection of the signal. With averaging, the noise level was 0.8 µV and the signal was reliably distinguished.

Signal averaging reduces random noise with respect to the wave form of interest. In this study, electrodes were placed at a distance from the apical impulse to avoid artifact from electrode movement that would be synchronized with the cardiac cycle and, therefore, not minimized by signal averaging.

Inspection of the signal-averaged X, Y, Z leads showed that the QRS duration was longer in patients with VT (fig. 6A). Seventy-two percent of the VT patients had a QRS duration greater than 120 msec in the absence of right or left bundle branch block; none of the control patients did. A 38–50% incidence of nonspecific intraventricular conduction delay in patients with inducible VT has been reported. Uther et al., using signal-averaging techniques, observed that five patients with VT had evidence of ventricular activation up to 170 msec after QRS onset.

Notching of the late QRS complex was seen in 77% of patients with VT but also in 44% of the control group. It was difficult, even in retrospect, to identify with high specificity patients with VT on the basis of the unfiltered X, Y, Z leads.

**Digital Filter**

A high-pass filter is necessary to reject the low frequencies in the ECG associated with the plateau and repolarization phases of the action potential, the ST segment and T wave. The filter enhances relatively the high frequencies corresponding to movement of wave fronts of activation.

A common problem with sharp high-pass filters is ringing of the filter after an abrupt transient. A solution is to use a digital filter that processes backward in time. By this means, low-voltage signals in the terminal QRS complex could be detected without being obscured by ringing of the filter from large-amplitude signals earlier in the QRS complex. Also, by eliminating filter ringing, a lower-frequency filter could be used, improving the signal-to-noise ratio and detection of low-amplitude signals.

Use of this filter revealed that patients with VT had a low-amplitude and slowly declining high-frequency signal at the end of the QRS complex. In contrast, the control group had a different high-frequency energy distribution; the high-frequency voltage at the end of the QRS complex was of larger amplitude, but ended abruptly. Ninety-two percent of the patients with VT had less than 25 µV of high-frequency energy in the last 40 msec of the QRS complex; only 7% of patients

**Table 1. Voltage in Last 40 Msec of Filtered QRS Complex**

<table>
<thead>
<tr>
<th>Subgroup</th>
<th>Control</th>
<th>Ventricular tachycardia</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior MI</td>
<td>88.6 ± 56.5</td>
<td>20.6 ± 20.7</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Inferior MI</td>
<td>55.4 ± 25.2</td>
<td>10.9 ± 5.2</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>QRS duration &lt; 120 msec</td>
<td>73.8 ± 47.7</td>
<td>27.1 ± 21.9</td>
<td>0.002</td>
</tr>
<tr>
<td>MI older than 3 months</td>
<td>75.8 ± 44.5</td>
<td>15.0 ± 15.2</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>No aneurysm</td>
<td>74.0 ± 47.2</td>
<td>14.1 ± 14.9</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>No propranolol</td>
<td>96.4 ± 57.1</td>
<td>14.8 ± 14.4</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

Abbreviation: MI = myocardial infarction.
without VT had less than 25 \mu V in this segment. This measurement appeared to be an accurate marker for VT in this study.

There was no separate, discrete high-frequency signal in the ST segment. All high-frequency wave forms were continuous with the QRS complex. In patients with VT, Rozanski et al. found a high-frequency signal early in the ST segment that disappeared after aneurysmectomy for control of VT. Differences in filter design or electrode configuration may explain the differences between the previous study and this one.

Diastolic Electrical Activity

Electrical activity during diastole has been noted in epicardial and endocardial recordings from animals and man after MI. Diastolic activity may be related to reentrant ventricular arrhythmias. No activity was found in the ST segment in the current study. There are several possible explanations for the absence. All patients were recorded during normal sinus rhythm; in contrast, pacing at rapid rates or with premature complexes is frequently needed to promote diastolic activity in experimental studies. Second, diastolic activity may vary from beat to beat and may not have sufficient time coherence to allow detection with a signal-averaging technique. Third, the continuous diastolic activity is of low amplitude, even when recorded directly from the heart, and may be below the noise level or threshold for detection from the body surface.

Slow conduction in areas of infarcted tissue has been observed in animals and man, both acutely and chronically. Although not fully explained, slow conduction may arise from abnormally low resting potential, decreased rate of depolarization, decoupled cells and prolonged, time-dependent refractoriness of cells. The low-amplitude, high-frequency signals in the distal QRS complex of patients with VT may arise from areas of slow conduction with infarcted tissue. The distribution of high-frequency energy in patients with VT (fig. 5A) suggests that the bulk of ventricular activation occurs with normal velocity, and only late in the QRS complex can the conduction slowing be detected when it is not obscured by activation of normal tissue. In contrast, patients who did not have VT appear to have an abrupt decline in high-frequency energy at the end of the QRS complex, suggesting that there are no areas of markedly slow conduction within the heart.

Patients in the control group had no complex ventricular ectopy and may be in the minority of patients after MI. Further study is needed to determine if the techniques we describe will differentiate those patients prone to sustained VT from all patients after MI.

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THE ONLY EFFECTIVE TREATMENT for ventricular fibrillation is immediate termination by countershock. In an effort to facilitate treatment in a select group of high-risk patients, an AID (Medrad, Inc./Intec Systems, Inc.) implantable defibrillator has been proposed.1 Mirowski et al.2 and Schuder et al.3 demonstrated the feasibility of using such a device in animal preparations. Mirowski et al.2 successfully defibrillated nine of 11 normothermic patients using 5–15 J and a low-energy intraventricular catheter.4

Mirowski et al.3 and Langer et al.6 described an implantable system acceptable in size for implantation in humans. This device delivers up to 30 J, which may be sufficient for direct defibrillation of the human heart.6 We describe two new lead systems for the automatic implantable defibrillator. These electrode systems can be implanted using a subxiphoid approach and eliminate the need for a transpleural or transsternal approach to the mediastinum. These electrodes function as efficiently as the apical cup and catheter system.6

Materials and Methods

Electrode system I consists of two flexible silicone-covered titanium patches (fig. 1). The surface area available for electrical conductivity on each patch is 12 cm². The basilar patch electrode is sutured to the atrial pericardial surface, and the apical patch electrode is sutured to the diaphragmatic pericardial surface. Electrode system II consists of the same apical diaphragmatic patch and a transvenous titanium spring catheter with an electrically active surface area of 12 cm². The ideal position for the apical patch was determined by comparing defibrillation thresholds with a fixed basilar patch electrode over the right atrium and varying the position of the apical patch over the right and left ventricle. In system II the best position for the basilar transvenous spring catheter was determined by comparing defibrillation thresholds with a fixed apical patch electrode over the left ventricle and varying the position of the spring catheter from the junction of the azygos vein with the superior vena cava to a position within the right atrium adjacent to the tricuspid valve.

Most defibrillation thresholds described in the literature4–6 were established using dogs; therefore, dogs were used in establishing the baseline defibrillation thresholds for systems I and II. The midline pericardial–phrenic ligament prevented a subxiphoid approach, so we used a transpleural thoracotomy for...
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