Spectral Analysis of Signal-Averaged Electrocardiograms in Patients With Idiopathic Ventricular Tachycardia of Left Ventricular Origin

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Background. The signal-averaged ECG has been used to detect late potentials, and it is considered a noninvasive marker for areas of slow conduction requisite for reentrant arrhythmia. Late potentials are not usually found in patients with idiopathic ventricular tachycardia (VT); nevertheless, fragmented electrograms are often recorded in those patients during endocardial mapping. The purpose of this study was to investigate the spectral content of the signal-averaged ECGs with use of fast Fourier transform analysis (FFT) in patients with idiopathic VT of left ventricular origin.

Methods and Results. Signal-averaged ECGs were recorded in 12 patients with idiopathic VT originating from the left ventricle (group 1) and 25 age-matched normal volunteers (group 2). Frequency analysis with FFT was performed with a Blackman-Harris window in a segment length of 120 msec from 40 msec before the end of the QRS complex, and the frequency spectrum was displayed in a three-dimensional graph. Area ratio 1 (area of 20–50 Hz/area of 10–50 Hz) and area ratio 2 (area of 40–100 Hz/area of 0–40 Hz) were calculated in all subjects. Late potentials defined by the time domain were negative in all subjects. The area ratios of group 1 were significantly higher than those of group 2. High-frequency components in the three-dimensional graph were confined within the QRS complex.

Conclusions. These results suggest that frequency analysis of signal-averaged ECGs with FFT is an available method for detecting the high-frequency component within the QRS complex in some patients with idiopathic VT of left ventricular origin. (Circulation 1992;85:2054–2059)

KEY WORDS • fast Fourier transform analysis • tachycardia, ventricular, idiopathic • potential, late

The signal-averaging technique has been used to detect low-amplitude ECGs on the body surface at the end of the QRS complex. These signals are called late potentials and are considered a noninvasive marker for areas of slow conduction requisite for reentrant arrhythmia. In patients with ventricular tachycardia (VT) caused by ischemic heart disease, the incidence of late potentials is high, and their presence correlates with development of sustained and nonsustained VT.

Idiopathic VT of left ventricular origin is defined as sustained VT without clinically apparent heart disease and is characterized by its QRS morphology during VT (right bundle branch block). Reports of endomyocardial biopsy findings revealed histological abnormalities in 60–90% of such patients, and fragmented ventricular electrograms were often recorded during endocardial mapping. Reentry has been postulated as the mechanism for the VT on the basis that it can be induced and terminated by extrastimuli. However, the late potentials are not usually found in patients with this idiopathic VT. Mehta et al reported that late potentials were detected in none of six patients with right bundle branch block VT without clinically apparent heart disease.

Fast Fourier transform analysis (FFT) is a powerful analytic method for signal processing in the frequency domain that allows us to avoid the disadvantages caused by the inherent limitations of high-gain amplification and signal filtering required for analysis in the time domain. Lindsay et al demonstrated that differentiation of patients with and without VT by FFT was still possible in the presence of bundle branch block. In addition, spectrottemporal mapping of the ECGs with FFT has been used for better identification of patients with VT. The aim of this study was to analyze the frequency content of signal-averaged ECGs in patients with idiopathic sustained VT of left ventricular origin.

Methods

Subjects

From January 1988 through March 1991, 12 patients (one woman and 11 men) with recurrent sustained VT of left ventricular origin (group 1) but without clinical evidence of heart disease were studied at the National
TABLE 1. Clinical and Histological Findings in Patients With Idiopathic Left Ventricular Tachycardia

<table>
<thead>
<tr>
<th>Case</th>
<th>Age/sex</th>
<th>Rate (bpm)</th>
<th>Clinical VT</th>
<th>Area ratio 1</th>
<th>Area ratio 2</th>
<th>Frag</th>
<th>Endomyocardial biopsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>29/F</td>
<td>186</td>
<td>RBBB LAD</td>
<td>290</td>
<td>46.0</td>
<td>+</td>
<td>Moderate fibrosis and cell infiltration</td>
</tr>
<tr>
<td>2</td>
<td>40/M</td>
<td>180</td>
<td>RBBB RAD</td>
<td>188</td>
<td>29.9</td>
<td>-</td>
<td>Normal</td>
</tr>
<tr>
<td>3</td>
<td>31/M</td>
<td>200</td>
<td>RBBB LAD</td>
<td>219</td>
<td>30.2</td>
<td>-</td>
<td>Mild fibrosis and fat infiltration</td>
</tr>
<tr>
<td>4</td>
<td>30/M</td>
<td>160</td>
<td>RBBB RAD</td>
<td>185</td>
<td>27.6</td>
<td>-</td>
<td>Normal</td>
</tr>
<tr>
<td>5</td>
<td>64/M</td>
<td>210</td>
<td>RBBB LAD</td>
<td>220</td>
<td>30.2</td>
<td>+</td>
<td>Mild fibrosis and fat infiltration</td>
</tr>
<tr>
<td>6</td>
<td>37/M</td>
<td>180</td>
<td>RBBB LAD</td>
<td>176</td>
<td>23.5</td>
<td>-</td>
<td>Normal</td>
</tr>
<tr>
<td>7</td>
<td>35/M</td>
<td>220</td>
<td>RBBB LAD</td>
<td>192</td>
<td>26.2</td>
<td>-</td>
<td>Not done</td>
</tr>
<tr>
<td>8</td>
<td>34/M</td>
<td>200</td>
<td>RBBB LAD</td>
<td>206</td>
<td>29.2</td>
<td>-</td>
<td>Mild fat infiltration</td>
</tr>
<tr>
<td>9</td>
<td>19/M</td>
<td>160</td>
<td>RBBB RAD</td>
<td>222</td>
<td>38.2</td>
<td>-</td>
<td>Mild fibrosis</td>
</tr>
<tr>
<td>10</td>
<td>47/M</td>
<td>170</td>
<td>RBBB LAD</td>
<td>241</td>
<td>43.2</td>
<td>+</td>
<td>Mild fibrosis and cell infiltration</td>
</tr>
<tr>
<td>11</td>
<td>48/M</td>
<td>170</td>
<td>RBBB RAD</td>
<td>245</td>
<td>40.2</td>
<td>+</td>
<td>Moderate fibrosis</td>
</tr>
<tr>
<td>12</td>
<td>20/M</td>
<td>200</td>
<td>RBBB LAD</td>
<td>227</td>
<td>37.2</td>
<td>-</td>
<td>Mild fibrosis and fat infiltration</td>
</tr>
</tbody>
</table>

bpm, Beats per minute; VT, ventricular tachycardia; area ratio 1, area of 20–50/area of 10–50 Hz; area ratio 2, area of 40–100/area of 0–40 Hz; Frag, fragmentations in left ventricle; F, female; M, male; RBBB, right bundle branch block configuration; LAD, leftward frontal plane axis; RAD, rightward frontal plane axis.

Cardiovascular Center, Osaka, Japan. All patients were referred to us from all over Japan. The mean age of the patients at the time of our evaluation was 36.2±12.5 years. The period of time since onset of symptoms was 1–8 years (mean, 3.8 years), and the rate of clinical VT was 160–220 beats per minute. The QRS morphology during VT was a right bundle branch block pattern, defined as having wide terminal R or R' in the V1 lead. The electrical axis during VT was leftward in eight patients and rightward in four patients (Table 1). Exercise-induced VT occurred in three patients. The absence of organic heart disease was diagnosed by 1) normal cardiac examination, 2) normal resting ECG, 3) normal chest x-ray, 4) lack of significant ST depression or ST elevation during or after a submaximal treadmill exercise test, 5) a normal echocardiogram (no structural cardiac abnormalities, no enlargement of the cardiac chambers, normal left ventricular wall thickness, and normal left ventricular wall motion), and 6) a normal radionuclide angiogram. The left origin of the VT was determined by ventricular endocardial mapping during VT. Sustained VT was defined as protracted paroxysmal runs of VT lasting longer than 5 minutes and usually requiring pharmacological or electrical intervention.

Group 2 was composed of 25 age-matched healthy volunteers (five women and 20 men) without signs of heart disease.

Signal-Averaged ECG

Signal averaging was performed using the LVP-101EPX (Arrhythmia Research Technology). Antiarrhythmic medications were discontinued at least 3 days before the study. Signal-averaged ECGs were recorded during sinus rhythm (standard bipolar orthogonal leads X, Y, and Z), and 350 beats were averaged to obtain a noise level of <0.3 μV. The signals were amplified, digitized, averaged, and filtered with a bidirectional filter at frequencies of 25–250 Hz. The duration of the filtered QRS complex and the root mean square of the amplitude of signals in the last 40 msec of the filtered QRS complex were determined by a computer algorithm. Late potentials were considered to be present if any two or all three of the following criteria were met: 1) the duration of the filtered QRS complex was >120 msec, 2) the duration of the filtered QRS complex after the voltage decreased to <40 μV was >40 msec, and 3) root-mean-square voltage during the last 40 msec of the filtered QRS complex was <20 μV.

Fast Fourier Transform Analysis

FFT was performed on the terminal 40 msec of the QRS complex and ST segment of each signal-averaged X, Y, and Z lead. For each region of interest, a 240-point (120-msec) interval was calculated with a four-term Blackman-Harris window to reduce spectral leakage. After FFT, the data were plotted on a high-resolution plotter and expressed as a ratio (area ratio 1) of the area under the spectral plot between 20 and 50 Hz divided by the area under the spectral plot between 10 and 50 Hz. In addition, area ratio 2 was calculated as the area under the spectral curve between 40 and 100 Hz divided by the area between 0 and 40 Hz. For patient-to-patient comparison, the individual X, Y, and Z values for the area ratios were averaged arithmetically and multiplied by 1×10^5 to facilitate graphic display.

Three-dimensional Frequency Analysis

Three-dimensional frequency analysis was performed according to Haberl et al. The ST segment was divided into 25 segments: the first segment started 30 msec after the end of QRS, subsequent segments started progressively earlier in the ST segment in steps of 2 msec, and the 25th segment started 20 msec inside the QRS complex. The frequency components of each segment were calculated by Fourier transform with a Blackman-Harris window (120-msec interval). In the power spectrum, the frequency in hertz (range, 0–200 Hz) was plotted versus transform magnitude in a logarithmic scale (in decibels). The 25 frequency spectra were combined into a three-dimensional plot.

Electrophysiological Studies

Electrophysiological studies were performed within 1–30 days (mean time period, 7 days) after the signal-averaging study. After giving informed written consent,
patients underwent electrophysiological studies while they were in the postabsorptive state. Antiarrhythmic medications were discontinued at least 3 days before the studies. Left and right ventricular endocardial electrograms were recorded by a 6F bipolar catheter with an interelectrode distance of 1 cm. The presence of fragmented electrograms during normal sinus rhythm was examined by recording an electrogram from five sites for the right ventricle and 12 sites for the left ventricle. A fragmented electrogram was defined as multiple components, a duration >60 msec, and a peak-to-peak amplitude <1 mV. Atrial pacing, programmed ventricular extrastimulation, and ventricular pacing were performed for induction of VT as previously reported. Atrial pacing was performed from the right atrium. The duration of stimulation was 30 seconds. For stimulation, an initial rate just above the spontaneous sinus rate was selected. The rate was increased by 10 beats per minute until atrioventricular block occurred. During programmed ventricular extrastimulation, the ventricle was paced at two different cycle lengths (600 and 400 msec). If a single ventricular extrastimulus failed to elicit VT, timed double ventricular extrastimuli were delivered until VT was provoked or until all extrastimuli failed to evoke ventricular responses. If VT was not induced with the extrastimuli, incremental ventricular pacing at cycle lengths of 400–240 msec for periods of 5–30 seconds was performed. Stimulation was first performed at the right ventricular apex, but when VT was not induced, the same stimulation protocols were repeated at the right ventricular outflow tract. When VT was induced neither by stimulation at the right ventricular apex nor at the right ventricular outflow tract, protocols were repeated at the left ventricular apex. The tachycardia was considered inducible only when the induced VT replicated the spontaneous tachycardia in both morphological characteristics and rates.

The site of origin of VT was determined by recording the site of earliest activation during tachycardia of eight sites in the right ventricle and 12 in the left ventricle by the technique of Josephson et al. Cardiac Catheterization and Endomyocardial Biopsy

Cardiac catheterization, including right and left ventricular angiography and coronary arteriography, and endomyocardial biopsies were performed in 11 patients. Cine left and right ventriculograms and coronary arteriograms were qualitatively evaluated by three experienced angiographers for segmental ventricular wall motion abnormalities, valvular lesions, and the presence of significant coronary artery disease defined as a >75% narrowing of the luminal diameter.

Endomyocardial biopsy was performed immediately after the study. Two or three biopsy specimens were obtained from right and left ventricles. Each specimen was evaluated by light microscopy by three of us for endocardial surface alterations, myocardial cell changes, vascular abnormalities, and inflammatory cell infiltrates, and the degrees of fatty infiltration and fibrosis were classified as mild, moderate, and severe.

Statistical Analysis

Data presented are mean±SD. Statistical comparisons between two groups were performed with unpaired Student’s t test. Significance refers to a value of p<0.05.

Results

Electrophysiological Characteristics

Atrial pacing induced VT in two of 12 patients. A single ventricular extrastimulus induced VT in 10 of 12 patients. Rapid ventricular pacing was required to induce VT in two patients. Nonclinical VT that was not electrocardiographically identical to that occurring spontaneously was not observed in any patient. Fragmented electrograms were found in the small area of the left ventricular apex in four patients during normal sinus rhythm. Their durations, however, were shorter (<100 msec) than those seen in patients with VT after myocardial infarction. The site of earliest ventricular activation of the VT that could be determined by endocardial mapping was the left ventricular apex in all patients. Rapid pacing from the right ventricular apex terminated tachycardia in all 12 patients. The coupling interval of premature extrastimuli was inversely correlated to the first cycle of the induced tachycardia, and the entrainment criteria of constant fusion, which progressed at shorter cycle lengths, were seen in these patients.

![Figure 1](image-url) Scatterplots showing filtered QRS duration (left panel), <40 μV duration (middle panel), and root-mean-square (RMS) voltage during the last 40 msec (right panel) for the group 1 patients and group 2 controls. Bars indicate mean±SD. Horizontal dotted lines indicate normal limit.
Root-mean-square voltage in the last 40 msec was slightly lower in group 1 (group 1, 31.2±14.4; group 2, 55.7±35.4; p<0.05), and two patients of group 1 showed abnormal values (<20 μV). No patient, however, showed abnormal values in two or three of these three criteria by the time domain analysis (Figure 1).

**Fast Fourier Transform Analysis**

The mean value of area ratio 1 (20–50/10–50 Hz) in group 1 was significantly higher than those in group 2 (218±31 versus 196±15, p<0.01). Seven (58%) of 12 patients had a value (>215) greater than the maximal value of area ratio 1 in control subjects. The mean value of area ratio 2 (40–100/0–40 Hz) was also significantly higher in group 1 (33.5±7.2 versus 20.9±2.1, p<0.001). Nine (75%) of 12 patients had a value (>28) greater than the maximal value of area ratio 2 in control subjects (Figure 2 and Table 1). Four of 12 patients in group 1 had fragmented electrograms in the left ventricle (Table 1). Their area ratios were relatively higher than those of patients without fragmented electrograms in the left ventricle. These patients also had abnormal histological findings, including infiltrations of inflammatory cells and fibrosis.

**Three-dimensional Frequency Analysis**

The three-dimensional frequency analysis of the terminal QRS complex and ST segment showed no high-frequency components >40 Hz in the segment outside the QRS complex. When the segments started slightly inside the QRS complex, however, spectral peaks in the range of 40–100 Hz appeared and progressively increased in amplitude (Figure 3). These spectral peaks were seen in eight (67%) of 12 patients of group 1.

**Discussion**

The present analysis using time domain techniques failed to detect late potentials in all patients with idiopathic VT of left ventricular origin. Few data have been published on signal-averaged ECGs in patients with idiopathic VT, including right and left ventricular origin.13-15 Buxton et al13 reported that no patient displayed an abnormally wide filtered QRS complex; only one patient had a low-amplitude, high-frequency signal in the

*Cardiac Catheterization and Endomyocardial Biopsy*

Cardiac catheterization, including left ventricular angiography and coronary arteriography, was normal in all patients. Mildly increased interstitial fibrosis or fat infiltration was found in eight (73%) of 11 patients' biopsy specimens, and lymphocyte infiltration was found in two of these eight patients, but normal histological features were found in only three patients (Table 1).

*Signal-Averaged ECGs by the Time Domain*

Filtered QRS durations were <120 msec in all subjects, although the mean value was slightly longer in patients with idiopathic left VT (group 1, 103±7 msec; group 2, 96±10 msec; p<0.05). Values of <40-μV duration were similar in the two groups (group 1, 29.6±8.5 μV; group 2, 26.4±5.8 μV), although only one patient of group 1 showed a slightly abnormal value.

**Figure 2.** Scatterplots showing comparison of values for area ratios in patients with idiopathic ventricular tachycardia (VT) of left ventricular origin (group 1) and control groups (group 2). Left panel indicates area ratio 1 (20–50/10–50 Hz), and right panel indicates area ratio 2 (40–100/0–40 Hz). Horizontal dotted lines indicate maximal value of area ratio in control subjects. Bars indicate mean±SD.

**Figure 3.** Spectral mapping in a normal control (left panel) and a patient with idiopathic ventricular tachycardia (VT) of left ventricular origin (right panel). In segment outside the QRS complex (0 to +30 msec), there are almost no high-frequency components; analysis of segments that start slightly inside the QRS (0 to −20 msec), however, reveals higher spectral peaks (arrow) in the range 40–100 Hz in patient with idiopathic VT. Normal control does not reveal high-frequency components.
terminal QRS; and discrete high-frequency signals in the ST segment were not observed in 30 patients with idiopathic VT originating in the right ventricle. Mehta et al.\(^1\) reported that late QRS potentials were found in seven (18%) of the 38 patients with right and left VT without clinically apparent heart disease. However, none of six patients with right bundle branch block VT had positive late potentials. These findings indicate a poor detectability of late potentials by time domain techniques in patients with idiopathic VT.

Fragmented electrograms are often recorded during endocardial mapping in patients with VT and organic heart disease. These abnormal electrograms are thought to represent an area of slow conduction and are presumed to be a substrate for reentrant arrhythmia. Subsequently, the presence of late potentials, which are low-amplitude, high-frequency potentials recorded on the body surface corresponding to intracardiac delayed potentials, generally suggests the reentrant mechanism.\(^2\) In this study, four of 12 patients (33%) recorded fragmented electrograms by endocardial mapping, although their fragmented electrograms were short and located in the small area of the left ventricular apex. Thus, failure to record a late potential in the time domain may arise from limited and brief fragmented electrograms. However, abnormal high-frequency components of the terminal portion of the QRS complex were detected by FFT in some patients with idiopathic VT of left ventricular origin. These findings indicate that analysis of the signal-averaged ECG in the frequency domain is more sensitive in identifying patients with idiopathic VT than that in the time domain.

The incidence of abnormal ventricular histological findings on biopsy in patients with idiopathic VT was 60% to 90% in previous reports.\(^4\) Mehta et al.\(^1\) demonstrated that late potentials of the patients with idiopathic VT were associated with an increase in fibrosis. The histological findings of an increase of fibrosis or fatty infiltration in the interstitium and subendocardium are similar to dilated cardiomyopathy or arrhythmogenic right ventricular dysplasia,\(^2\) although the findings are very mild. The biopsy can identify disease at a relatively early stage before overt ventricular abnormalities develop.\(^6\) In the present study, the patients with abnormal histological findings had high values of area ratio. Therefore, we suggest that frequency analysis may help identify high-risk patients among those with idiopathic VT.

**Study Limitations**

Frequency analysis has not yet refined criteria for abnormal area ratio or definition of abnormal spectral peak. In this study, area ratio was considered to be abnormal if it was greater than the maximal value of area ratio in control subjects, and high-frequency components in the range 40–100 Hz were considered abnormal spectral peak.

We did not subtract the mean value before FFT.\(^2\) This might decrease the advantage of spectral analysis in separating the data of VT patients from controls. In addition, we used only two spectral area ratios (20–50/10–50 and 40–100/0–40 Hz). Another area ratio might have obtained better results. Furthermore, the possibility that the higher area ratio in patients with idiopathic left VT might be related to the smaller low-frequency area rather than the larger high-frequency area cannot be excluded in the present study, because we did not compare them with the absolute area values.

**Conclusions**

Although some patients with idiopathic VT of left ventricular origin had high-frequency components within the QRS, there was much overlap between group 1 and group 2 controls. Therefore, we should conclude that only some patients with idiopathic VT have these findings and that FFT and spectrotomential mapping are very practical methods to detect such patients. It might suggest that this group is electrophysiologically heterogeneous.

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