

# Spectral mapping of the electrocardiogram with Fourier transform for identification of patients with sustained ventricular tachycardia and coronary artery disease

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*In time domain analysis, detection of late potentials is limited by high pass filtering, noise interference and the necessity to exclude patients with bundle branch block. We therefore used frequency analysis with Fourier transform of multiple segments of the surface electrocardiogram (25 segments, size 80 ms, time shift 3 ms) during sinus rhythm after signal averaging. Thirty-two post-myocardial infarction patients with sustained ventricular tachycardia (VT), 19 post-myocardial infarction patients without VT and 17 healthy subjects were studied. A total of 18 patients had bundle branch block. In 24 out of 32 patients with VT, three-dimensional spectral plots were characterized by spectral peaks > 10 dB in the range of 40–200 Hz in segments only at the end of QRS and the early ST wave, but not far outside the QRS. In only 2 out of 19 patients without VT and in 1 out of 17 healthy subjects could such peaks be observed. Noise caused spectral peaks throughout all segments. Sixteen out of 18 patients with bundle branch block were correctly classified with spectral mapping. With the Simson method, patients with bundle branch block had to be excluded, abnormal results were found in 10 out of 19 patients with VT, but also in 5 out of 15 patients without VT and in 3 out of 16 healthy subjects.*

*Thus, spectral mapping of the electrocardiogram offers promise for better identification of patients prone to sustained VT in the presence of coronary artery disease.*

## Introduction

Delayed ventricular activation in the early ST segment of the electrocardiogram during sinus rhythm has been considered a non-invasive marker for sustained ventricular tachycardia (VT) in patients who have had myocardial infarction. Reliable detection of these very small signals (1–20  $\mu\text{V}$ ) from the body surface is at the limits of technical performance. The current methods in the time domain after signal averaging and high amplification have severe limitations: high pass filters are inevitable and may disturb the signals; discrimination between noise and late potentials is difficult; the definitions of abnormal findings are inconsistent; and patients with bundle branch block in general have to be excluded.

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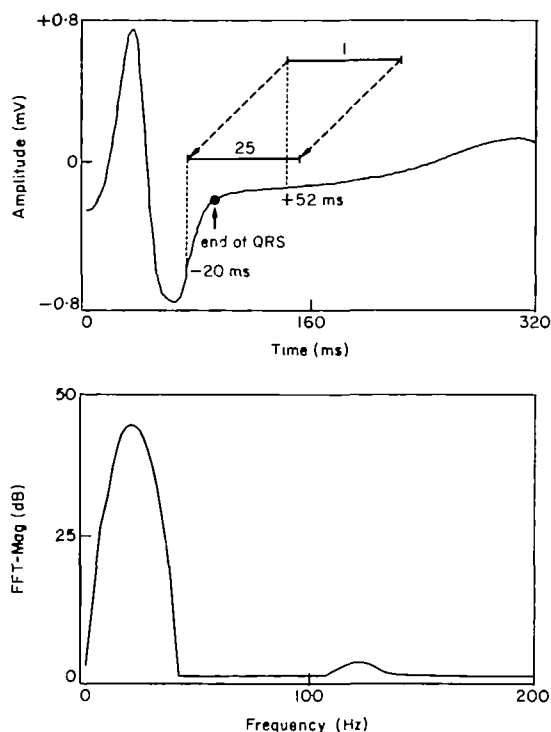
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To overcome these limitations we developed a new method which identifies late potentials by a higher frequency content in the otherwise low frequent ST segment.

## Methods

Three bipolar, orthogonal electrocardiograms were recorded simultaneously with a special low-noise, high-gain amplifier (filter: 0–300 Hz). The signals were digitized with 12-bit accuracy at a sampling rate of 1000 Hz. The data were analysed and stored on a Hewlett Packard system (computer model 9836A and multiprogrammer 6944A). On-line signal averaging was performed until the noise level was < 1  $\mu\text{V}$ . The trigger point was determined by cross-correlation of each beat with a template. Grossly noisy signals and extrasystoles were eliminated by a computer algorithm before signal averaging.

The end of the QRS complex was defined as the point where the spatial vector velocity of the electrocardiogram decreased below 5  $\text{mV s}^{-1}$ . This algorithm allowed clear definition of the end of the



**Figure 1** Spectral mapping with Fourier transform: method. Upper panel: In the time domain 25 segments are defined within the ST wave (segment size 80 ms). The first segment starts 52 ms after the end of QRS (segment 1), the subsequent segments approach the QRS complex in steps of 3 ms. Lower panel: of each segment, a Fourier transform is calculated by the computer after multiplication with a Hanning window. The power spectrum gives the FFT magnitude in a logarithmic scale (dB) as a function of frequency (Hz). The spectrum of segment 1 is shown here as an example: spectral peaks occur at 20 Hz and 120 Hz. The spectra of 25 segments are then combined to give a three-dimensional plot (as in Fig. 2).

QRS even in patients with bundle branch block, leaving delayed ventricular activation *outside* the QRS complex. As a reference, the method of Simson<sup>[1]</sup> was applied in all patients without bundle branch block: orthogonal electrocardiograms were combined into a vector magnitude after bidirectional high pass filtering at 25 Hz. A filtered QRS duration > 120 ms or a RMS voltage < 25  $\mu$ V was considered abnormal.

For frequency analysis, the ST segment was divided into 25 segments: the first segment started 52 ms after the end of QRS (segment size 80 ms), the subsequent segments started progressively earlier in the ST segment in steps of 3 ms, thus, the 25th

segment started 20 ms inside the QRS complex (Fig. 1). The frequency components of each segment were calculated with Fourier transform (FFT): the segments were multiplied point by point with a Hanning window to avoid edge discontinuities. The data points were set at the beginning of an array of 512 elements, the remaining points were set to zero. Direct current offset was eliminated before Fourier transform. In the power spectrum (Fig. 1), the frequency in Hertz (range 0–200 Hz) is plotted vs FFT magnitude in a logarithmic scale (dB). The 25 frequency spectra were combined into a three-dimensional plot. The frequency spectrum of segment 1 (which started far outside the QRS) was defined as a reference spectrum; the spectra of segments 2–25 were compared with this reference spectrum by cross-correlation in the frequency range 40–150 Hz<sup>[2]</sup>. The similarity of spectra was indicated by the correlation coefficient: zero if two spectra showed no similarity; one if two spectra were identical.

A 'factor of normality' (NF) was calculated. The mean of the correlation coefficients of segments 20–25 was divided by the mean of the correlation coefficients of spectra 1–5, multiplied by 100. High frequency content at the end of QRS (spectra 20–25) which is absent far outside QRS (spectra 1–5) causes NF to be low. NF ranges between 0 and 100% (0 = strong evidence of late potentials, 100% = no evidence of late potentials). NF below 30% is considered abnormal (see discussion).

Three groups of patients were analysed:

#### GROUP 1

This consisted of 32 consecutive patients after myocardial infarction (for a median of 24 months) with documented, sustained VT. Cardiac catheterization and an electrophysiologic study were performed in all patients. Single and double extrastimuli were applied at the right ventricular apex and pulmonary outflow tract (basic drive 100, 120, 150 and 180 beats  $\text{min}^{-1}$ , rectangular pulses, twice diastolic threshold, duration 2 ms). In each case antiarrhythmic drugs were withdrawn for at least five half-lives. In 28 out of 32 patients a sustained VT could be induced (duration > 30 s or termination necessary because of haemodynamic deterioration). In four patients a non-sustained VT was induced.

#### GROUP 2

This consisted of 19 consecutive patients referred a median of 20 months after a myocardial infarc-

Table 1 Clinical data from patients in groups 1 and 2

	Group 1 MI with VT	Group 2 MI without VT
n =	32	19
Age (years)	57 ± 9	55 ± 11
Sex (m/f)	24/8	14/5
Anterior infarction	19	14
Posterior infarction	14	5
Aneurysm	14	6
Ejection fraction (%)	38 ± 19	42 ± 13
Bundle branch block	13	4

MI = myocardial infarction; VT = ventricular tachycardia.

tion for routine coronary arteriography. None had arrhythmias, syncope or palpitation. A 24 h ambulatory electrocardiogram revealed < 10 ventricular ectopic beats  $h^{-1}$  and no ventricular pairs. The clinical features of group 1 and 2 patients are given in Table 1. The incidence of ventricular aneurysm and myocardial performance (expressed as ejection fraction) was similar in both groups.

### GROUP 3

This comprised 17 healthy volunteers without heart disease (age  $34 \pm 9$  years; 11 men, 6 women). Patients with bundle branch block (QRS duration > 0.12 s) were not excluded. Statistical evaluation was done with the Wilcoxon rank sign test for unpaired data.

## Results

### RESULTS IN REPRESENTATIVE PATIENTS

Frequency analysis of the ST segment in a post-myocardial infarction patient without VT is shown in Fig. 2. In the three-dimensional plot, the lowest spectrum represents Fourier analysis of a segment which started 52 ms after the end of QRS; the spectrum at the top shows the analysis of a segment which started 20 ms inside the QRS complex. All frequency plots revealed a spectral peak at a low frequency (15 Hz), which was caused by the slow fundamental component of the ST segment. Above 50 Hz were no high frequency components. Since all spectra were almost identical, the correlation coefficient between each spectrum and reference spectrum 1 never fell below 0.9. The factor of normality was 100%.

Fig. 3 demonstrates a patient in the chronic phase after a myocardial infarction with recurrent,

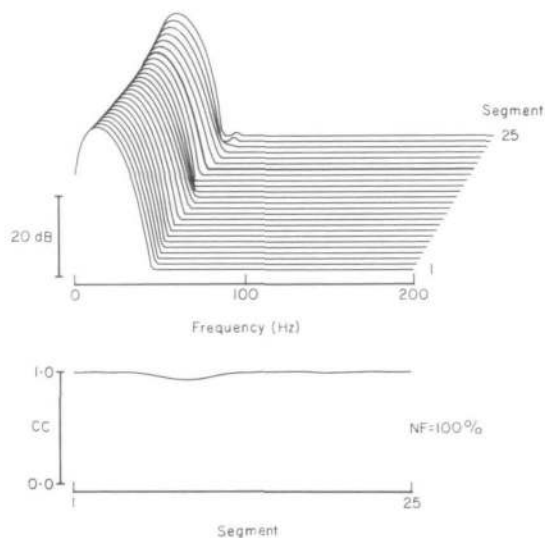
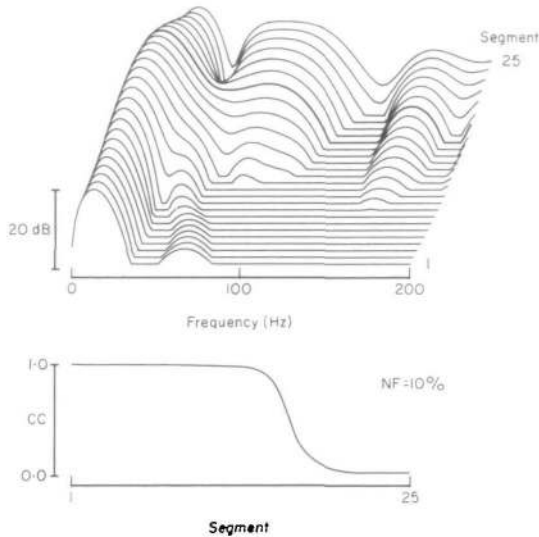


Figure 2 Spectral mapping of the ST segment in a post-myocardial infarction patient without VT. Segment 1 starts 20 ms inside the QRS, segment 25 starts 52 ms after the end of QRS. The three-dimensional plot of 25 spectra does not reveal high frequency components above 50 Hz. The correlation coefficient between each spectrum and the reference spectrum 1 does not fall below 0.9 (CC in lower panel). The factor of normality (NF) is 100%.

sustained VT. He showed late potentials (amplitude  $7 \mu V$ ) at the end of QRS in the time domain after signal averaging and high gain amplification. The filtered QRS complex according to Simson was 125 ms wide, the RMS voltage  $6 \mu V$ . The three-dimensional frequency analysis of the ST segment did not show high frequency components above 50 Hz in segments far outside the QRS complex (segments 1–12). However, when the segments started close to the end or slightly inside the QRS complex (segment 13–25), spectral peaks in the range 50–200 Hz appeared and progressively increased in amplitude. This means that high frequency components existed at the end of QRS which were absent far outside the QRS complex; a characteristic finding due to delayed ventricular activation. As a consequence, the correlation coefficient gradually fell to zero in segments 13–25. The factor of normality in this patient was 10%, because the correlation coefficients of segments at the end of QRS were markedly lower than of those far outside the QRS.

Fig. 4 gives another example of a post-myocardial infarction patient without VT. In a high-gain recording of the ST segment, late potentials could



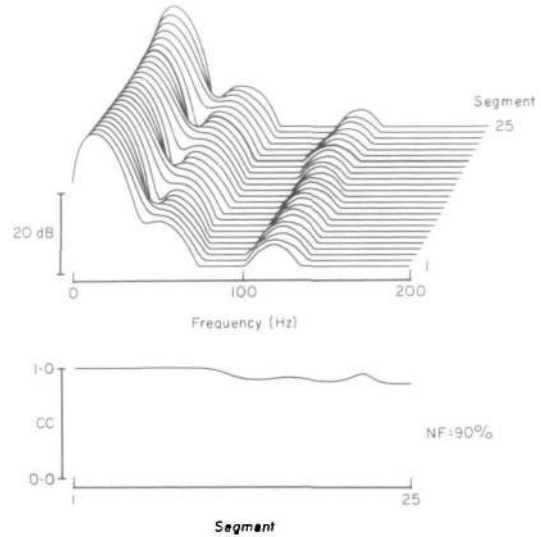
**Figure 3** Spectral mapping in a post-myocardial infarction patient with sustained VT. In segments far outside the QRS complex (segments 1–12) there are almost no high frequency components; however, analysis of segments which start at the end of the QRS or slightly inside the QRS reveals progressively higher spectral peaks in the range 50–200 Hz (segments 13–25). Therefore, the correlation coefficient (CC) drops to zero in these segments; the factor of normality (NF) is 10%. This is the characteristic spectral representation of delayed ventricular activation.

not clearly be identified in the time domain because of low-amplitude noise interference. QRS duration according to Simson was 123 ms, RMS voltage 24  $\mu$ V. In the three-dimensional frequency plot there was a peak again at the fundamental frequency (15 Hz), but also high-frequency components at 50 and 120 Hz. However these high frequency components were present equally in all spectra, both far outside the QRS (segments 1–12) and at the end of the QRS complex (segments 13–25). Therefore, these components did not represent localized delayed ventricular activation, but noise interference (i.e. line disturbance or muscle noise). The correlation coefficients between the spectra (lower panel) did not fall below 0.8 in this patient, the factor of normality was 90%. Thus, there was no indication of delayed ventricular activation in this patient.

#### RESULTS IN PATIENT GROUPS

Fig. 5 gives the results in our three patient groups. In each patient the factor of normality (NF) is calculated.

In post-myocardial infarction patients with VT 24 out of 32 had a factor of normality below 30%.



**Figure 4** Spectral mapping in a patient after myocardial infarction without ventricular tachycardia. High frequency components at 50 and 120 Hz uniformly spread through all spectra. They are caused by noise interference. The correlation coefficient (CC) does not fall below 0.8. The factor of normality (NF) is 90%. There is no indication of delayed ventricular activation.

In 2 out of 19 post-myocardial infarction patients without ventricular arrhythmias, and only one healthy subject, NF was below 30%. The difference between patient group 1 and groups 2 and 3 was highly significant ( $P < 0.001$ ). Thus, spectral mapping of the ST segment allowed identification of patients with myocardial infarction prone to sustained VT in a large majority of cases.

#### COMPARISON WITH SIMSON METHOD

The comparison between spectral mapping and the Simson method is given in Fig. 5 and Table 2. Patients with bundle branch block had to be excluded when using the Simson method—thereby reducing sensitivity—but they could be evaluated with Fourier transform. Eleven out of 13 patients with bundle branch block in group 1 and all patients with bundle branch block in groups 2 and 3 were correctly classified by spectral mapping of the ST segment. Even in the remaining patients the concordance between Fourier transform and the Simson method was not complete: In a total of six patients (three in group 1, two in group 2, one in group 3) the Simson method yielded abnormal results, although Fourier transform showed a typical pattern of

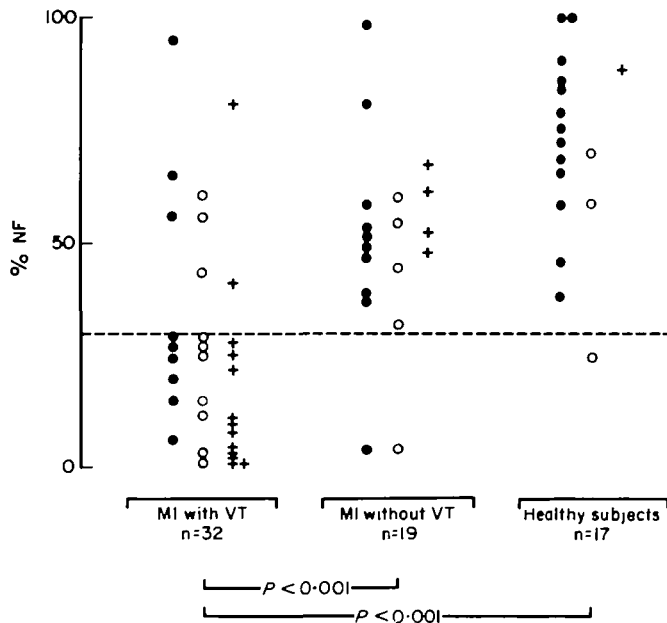


Figure 5 Comparison of Fourier analysis of the ST segment in patient groups 1–3. Results of the Simson method are included: ○ abnormal, ● normal, + excluded because of bundle branch block. In post-myocardial infarction patients with ventricular tachycardia, NF is lower than 30% in 24 out of 32 patients. Only 2 out of 19 post-myocardial infarction patients without arrhythmias and 1 out of 17 healthy subjects showed NF values < 30%.

Table 2 Comparison of Fourier analysis of the ST-segment and the Simson method

	MI with VT n = 32		MI without VT n = 19		normal subjects n = 17	
	FFT (%)	Simson (%)	FFT (%)	Simson (%)	FFT (%)	Simson (%)
Abnormal finding	75	31	10	26	6	18
Normal finding	25	28	90	53	94	76
Not to be evaluated	0	41	0	21	0	6

VT = ventricular tachycardia; MI = myocardial infarction; FFT = fast Fourier transform. For other definitions see method.

‘noisy’ signals (as in Fig. 4). Results were normal according to the Simson method in five group 1 patients, in whom spectral mapping strongly suggested the presence of delayed ventricular activation (Table 2).

## Discussion

Frequency analysis of the surface electrocardiogram has been proposed as an alternative to time

domain analysis for detection of late potentials in patients after myocardial infarction<sup>[3,4]</sup>. This method is based on the idea that delayed ventricular activation should be characterized by a higher frequency content than the otherwise low-frequency ST segment. Fourier transform is a mathematical algorithm which allows the calculation of the power spectrum of a periodic time series<sup>[5]</sup>. Non-periodic signals like the electrocardiogram may also be analysed with Fourier transform, however, some

methodological prerequisites have to be considered (adequate segments, window function etc.)<sup>[6]</sup>.

Frequency analysis of multiple segments of the ST wave ('spectral mapping') considerably enhances the detection of late potentials in patients after myocardial infarction. The method has distinct advantages:

First, spectral mapping consistently allows discrimination between late potentials and noise, which both have a typical spectral representation. Noise can easily be identified by spectral peaks that are present in all segments (Fig. 4). Late potentials give rise to spectral peaks only in segments at the end of the QRS, but not far outside the QRS complex. In the presence of late potentials, spectral peaks with progressively higher amplitudes appear in segments 13–25 as soon as the low amplitude signals are included in the segments and better pass the window function. Thus even single beat analysis is possible. In contrast, the definitions of the Simson method depend directly upon the noise level, a possible source of error (example in Fig. 4).

Second, our results suggest that patients with bundle branch block need not be excluded. This was also shown by Lindsay *et al.* who used a similar approach with Fourier transform of the surface electrocardiogram<sup>[7]</sup>. As seen in our patient groups 1 and 2, bundle branch block is a common finding in patients with organic heart disease and impaired left ventricular function. Since time domain analysis of signal-averaged electrocardiograms is limited in patients with intraventricular conduction abnormalities, frequency analysis considerably increases clinical applicability.

Third, spectral mapping of the ST segment does not require exact definition of the onset and end of the QRS, which often is difficult to determine.

Fourth, distinction between normal and abnormal is simpler and does not require more or less arbitrary criteria (as given in [8–10]). Instead, the factor of normality is calculated as a single parameter.

Last, complex high-pass filtering is not necessary and discussion about filter artifacts, signal distortion and the choice of adequate cut-off frequencies can be avoided<sup>[11,12]</sup>.

#### COMPARISON WITH PREVIOUS WORK WITH FOURIER TRANSFORM

Some prerequisites have to be carefully kept in mind when frequency analysis with Fourier transform is applied on the electrocardiogram:

DC-components have to be eliminated before Fourier transform, the spectra should not be normalized and the segments should not include more than 20 ms of the QRS complex<sup>[6]</sup>. Most of these requirements have not been fulfilled in previous studies<sup>[3,7,13–15]</sup> resulting in misinterpretation and conflicting results<sup>[14,16]</sup>. The choice of an appropriate segment is crucial: the segments should be short to focus on the late potentials, and multiplied by a Hanning window for better frequency resolution.

The three-dimensional spectral plots significantly improve the interpretation of the frequency spectra: a single spectrum may also reveal high frequency spectral peaks in the absence of late potentials due to noise pollution. With multi-segment analysis, noise can clearly be differentiated from delayed ventricular activation (Fig. 4). Analysis with cross-correlation of the spectra and calculation of a 'factor of normality' overcomes limitations of 'spectral areas' or 'area coefficients': the area under the frequency plot is affected by noise and artifacts and the 'valleys' between the spectral peaks. Information about delayed ventricular activation, however, is given only by the peaks, but not at all by the valleys between the peaks.

#### Conclusions

Multi-segment spectral mapping of the electrocardiogram with Fourier transform introduces a powerful analytic method: it significantly improves recent approaches with single spectrum evaluation of the electrocardiogram and it might overcome some limitations of conventional time domain analysis. It allows better detection of late potentials and discrimination towards noise. Thus, even single beat analysis is possible. In this retrospective study criteria for the identification of late ventricular activation have been worked out using frequency analysis of the electrocardiogram. This method should now be tested prospectively in larger groups of patients. It might enhance identification of patients prone to sustained VT after myocardial infarction.

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