

Can Heart Rate Variability Analysis Predict the Acute Onset of Ventricular Tachyarrhythmias?

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Abstract

The changes that occur in the electrocardiogram just prior to the onset of fatal ventricular tachyarrhythmias (VTAs) have been the focus of much research in the last decade. Changes in heart rate variability (HRV) have been detected immediately prior to the onset of VTAs, however due to conflicting findings in studies with only small numbers of patients, the exact nature of these changes remains unclear. We used the technique of Wavelet Transform HRV analysis as well as more conventional techniques, to see if VTAs could be predicted in coronary care unit patients.

1. Introduction

Given the current dismal survival rates of cardiac arrest occurring in the community, efforts directed towards the early detection of ventricular tachyarrhythmias (VTAs) are crucial [1]. The area of heart rate variability (HRV) behaviour prior to the onset of life-threatening VTAs, offers exciting possibilities. Newer improved analytical techniques such as wavelet transform analysis (WTA) and improved processing power, have led to easier signal acquisition and analysis. Previous studies looking at HRV changes prior to the acute onset of VTAs have shown conflicting results.

The aim of this study is to see whether the technique of Wavelet Transform HRV analysis as well as more conventional techniques, can be used to help predict the acute onset of VTAs.

2. Theory

The wavelet transform of a continuous time signal, $x(t)$, is defined as:

$$T(a,b) = \frac{1}{\sqrt{a}} \int_{-\infty}^{+\infty} x(t) \psi^* \left(\frac{t-b}{a} \right) dt \quad (1)$$

where $\psi^*(t)$ is the complex conjugate of the wavelet function $\psi(t)$, a is the dilation parameter of the wavelet and b is the location parameter of the wavelet. In order to be classified as a wavelet, the function must satisfy certain mathematical criteria. These are:

1 - A wavelet must have finite energy:

$$\text{i.e. } E = \int_{-\infty}^{\infty} |\psi(t)|^2 dt < \infty \quad (2)$$

2 - If $\hat{\psi}(f)$ is the Fourier transform of $\psi(t)$,

$$\text{i.e. } \hat{\psi}(\omega) = \int_{-\infty}^{\infty} \psi(t) e^{-i(\omega)t} dt \quad (3)$$

then the following condition must hold:

$$C_g = \int_0^{\infty} \frac{|\hat{\psi}(\omega)|^2}{\omega} d\omega < \infty \quad (4)$$

This implies that the wavelet has no zero frequency component, i.e. $\hat{\psi}(0) = 0$, or to put it another way, it must have a zero mean. Equation 4 is known as the *admissibility condition* and C_g is called the *admissibility constant*. The value of C_g depends on the chosen wavelet.

3 - For complex (or analytic) wavelets, the Fourier transform must both be real and vanish for negative frequencies.

The contribution to the signal energy at the specific a scale and b location is given by the two-dimensional wavelet energy density function known as the scalogram:

$$E(a,b) = |T(a,b)|^2 \quad (5)$$

The total energy in the signal may be found from its wavelet transform as follows:

$$E = \frac{1}{C} \int_{g=-\infty}^{+\infty} \int_{a^2}^1 |T(a,b)|^2 da db \quad \left[= \int_{-\infty}^{+\infty} x(t)^2 dt \right] \quad (6)$$

In practice a fine discretisation of the continuous wavelet transform is computed where usually the b location is discretised at the sampling interval and the a scale is discretised logarithmically. The a scale discretisation is often taken as integer powers of 2, however, we use a finer resolution in our method where the a scale discretisation is in fractional powers of two. The discretised continuous wavelet transform (CWT) is made distinct from the discrete wavelet transform (DWT) in the literature. In its basic form, the DWT employs a dyadic grid (integer power of two scaling in a and b) and orthonormal wavelet basis functions and exhibits zero redundancy. Our method, i.e. using a high resolution in wavelet space as described above, allows individual maxima to be followed accurately across scales, something that is often very difficult with discrete orthogonal or dyadic stationary wavelet transforms incorporating integer power of two scale discretisation. Further background information concerning continuous wavelets can be found in references [2] and [3].

3. Methods

Equipment was installed within the Coronary Care Unit of the Royal Infirmary of Edinburgh to collect ECG signals continuously from all six beds within the unit over an 18-month period [4]. The signals were sampled at 500 Hz using 16 bits per sample. The ECG signals from all patients who had an episode of VTA during their stay were scrutinised. Those that were not suitable for HRV analysis were discarded [Figure 1]. Those signals that were not suitable included patients who were not in sinus rhythm, patients who experienced multiple ectopic beats, and signals that had high levels of noise interference. The 60-minute ECG signal directly before the VTA event in the remaining 8 patients that were suitable for inclusion was then analysed. The R points were found using a wavelet transform-based algorithm [5] and then checked manually.

Ectopic beat frequency was calculated and ectopic R points were corrected to obtain the NN series. Time domain and wavelet domain HRV parameters were then computed. In the time domain we considered the NN 50 and the SDNN. The wavelet transform of the NN series was calculated. The mother wavelet employed was the Mexican hat. In the wavelet domain we considered the energy of the three standard bands, high frequency (HF; 0.15–0.40 Hz), low frequency (LF; 0.04–0.15 Hz) and very low frequency (VLF; 0.003–0.04 Hz), and also the LF/HF ratio. The changes in these parameters were then graphically represented over the 60-minute period prior to

the VTA event and wavelet transform plots were made for all patients.

4. Results

Trends in heart rate, HF, LF and VLF energy, LF/HF ratio, NN50 and SDNN were all noted in some patients towards the progression to a VTA event. These changes were not consistently present in all patients, and occurred for different periods prior to the VTA event.

Table 1 summarises the changes found in HRV characteristics amongst our 8 patients. Two patients had no change in any HRV characteristic. Two patients had a rise in HR prior to the VTA event [Figure 2], and one a fall. Five patients showed no change.

There was a rise in SDNN in two patients prior to the VTA event, a rise and a fall in NN50 in one patient each, and a rise a fall in LF again in one patient each.

LF/HF ratio fell in two patients prior to the VTA, HF rose in two patients, and VLF rose in three patients prior to the VLF. The ectopic beat count showed no change in 7 of the 8 patients prior to the VTA, however there was a significant increase in ectopic beat count in one patient 30 minutes prior to the VTA event [Figure 3].

There were no consistent changes in HRV characteristics in our 8 study patients, however there were some significant changes in individual HRV characteristics in individual patients. Figure 4 shows a typical wavelet transform plot for one of the study patients.

5. Discussion and conclusions

The characteristics of HRV immediately prior to the onset of VTAs are still unclear. Some studies report significant changes in HRV in the period immediately preceding a VTA. An increase in HR prior to an episode of VTA is one change that is commonly found [6]. Some studies have reported an increase in HR but no change in HRV spectra characteristics prior to VTAs [7]. Vybril et al. however [8], found no consistent changes in HRV indices or HR in 24 patients wearing Holter devices who developed VF.

Huikuri et al. [9], found a significant reduction in HR, SDNN, HFP, LFP and VLFP in post-MI patients who developed VT or cardiac arrest, compared to normal controls and post-MI patients who did not suffer an arrhythmia. These changes occurred in the one-hour period prior to the onset of the VTA and were more pronounced in patients developing sustained VT than those with non-sustained VT. These findings were also found by Shusterman et al [10], who noted a rise in HR and a fall in LFP, and LFP: HFP ratio before the onset of VT. Pruvot et al. [11], found an increase in HR and a significant reduction in HRV prior to the onset of a VTA

in post-MI patients. Other studies have shown a rise in VLF power and a decline in HF power [12], a decline in HF power but no VLF power changes [10], and a rise in LF: HF ratio [6,13].

Our results and those of previous studies strongly suggest an alteration in the interaction between the sympathetic and parasympathetic nervous system prior to the onset of VTAs. The effect on HR and HRV variables is likely to be heterogeneous and affected by individual patient characteristics, which may explain the conflicting evidence in the literature. This is compounded by the small numbers of patients studied, their differing drug treatment, underlying cardiac pathology, pre-existing medical problems and methods of recording and analysing the ECG.

We conclude that the changes in HRV in the hour period prior to a VTA event are not simple. The changes in heart rate and HRV variables are likely to be heterogeneous and affected by individual patient characteristics, compounded by their differing drug treatment, underlying cardiac pathology and pre-existing medical problems. Awareness of the heterogeneity of HRV behaviour emphasises the importance of building up much larger ECG databases from patients at risk of developing life-threatening VTAs and the increasing use of available sources i.e. coronary care units [4]. This will enable the acquisition of the vast quantity of signals that will be required to enable useful algorithm development, leading to an improved understanding of the role of HRV in VTAs in the future.

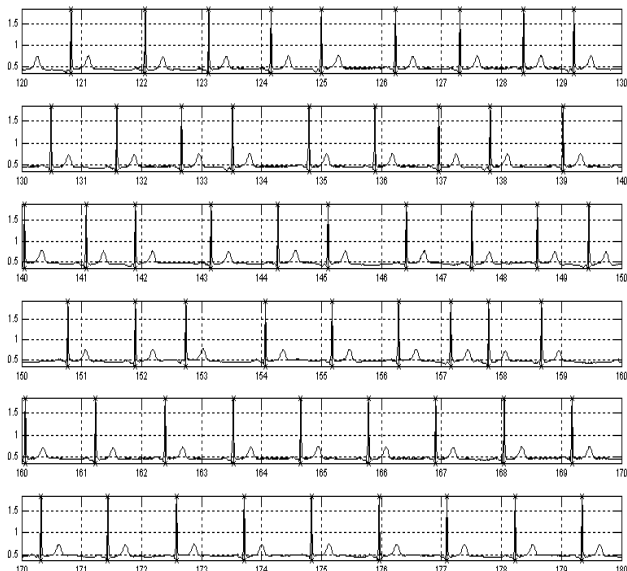


Figure 1. A section of an ECG signal obtained from a Coronary Care Unit, showing an irregular rhythm that precludes useful HRV analysis. The locations of the R-waves as detected by our R-wave detection algorithm prior to editing are represented by the red vertical lines.

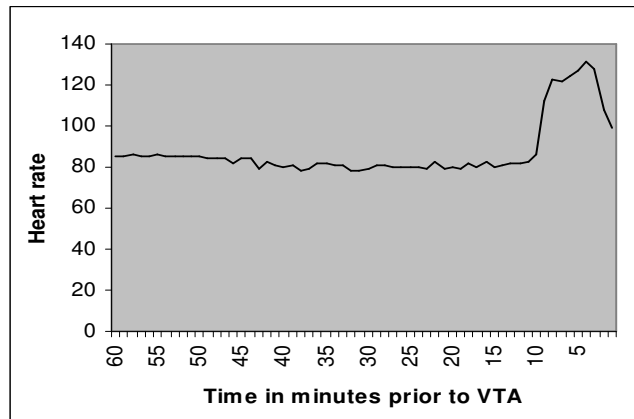


Figure 2. Change in HR prior to the onset of a VTA in one study patient.

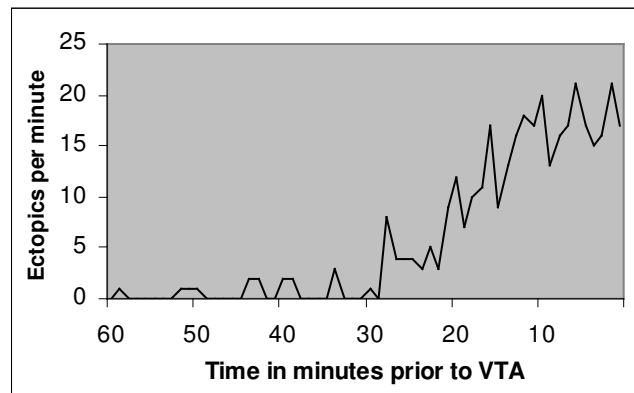


Figure 3. Change in ectopic beat frequency prior to the onset of a VTA in one study patient.

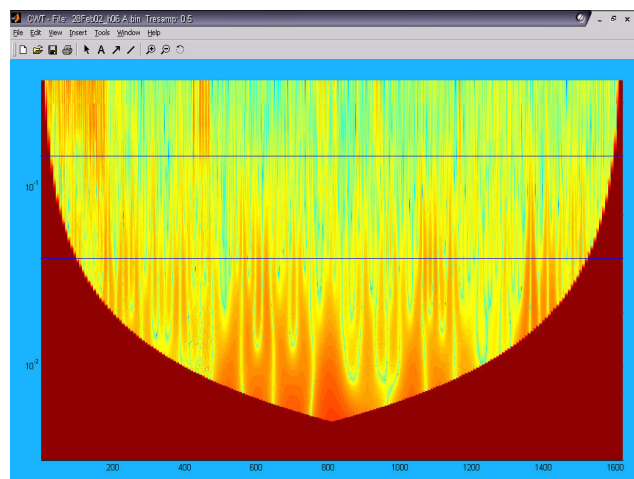


Figure 4. Wavelet transform plot for the one hour period prior to the onset of a VTA in one study patient.

Patient	HR	SDNN	NN50	LFP:HFP	HFP	LFP	VLFP	Ectopics
60502	↔	↔	↔	↔	↔	↔	↔	↔
111102	↑ 9 mins	↔	↔	↓ 8 mins	↔	↓ 8 mins	↔	↔
120303	↔	↔	↓ 28 mins	↔	↔	↔	↔	↔
150302	↔	↑ 5 mins	↔	↔	↑ 3 mins	↑ 3 mins	↑ 3 mins	↔
181202	↔	↔	↔	↔	↔	↔	↑ 8 mins	↔
220402	↑ 10 mins	↔	↔	↔	↔	↔	↔	↔
280202	↔	↔	↔	↔	↔	↔	↔	↔
300902	↓ 30 mins	↑ 40 mins	↑ 28 mins	↓ 30 mins	↑ 29 mins	↔	↑ 10 mins	↑ 30 mins

Table 1. Summary of HRV characteristics prior to the onset of the VTA episode in all 8 study patients.

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