Detection of Autonomic Modulation in Atrial Cycle Length During Atrial Fibrillation

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Abstract

A new method for estimation of changes in the atrial cycle length during atrial fibrillation is presented. The objective is to investigate whether or not respiration, through the autonomic nervous system, modulates the atrial cycle length in patients with permanent atrial fibrillation. A group of eight patients with permanent atrial fibrillation, AV block III and permanent pacemaker was studied during rest, rhythm controlled respiration at a rate of 0.125 Hz, and during rhythm controlled respiration after full vagal blockade using atropine. Using the new method, it was possible to detect a prominent spectral peak at 0.125 Hz during rhythm controlled respiration in two of the eight patients; the spectral peaks disappeared after full vagal blockade.

1. Introduction

It is well-known that the heart rate during sinus rhythm is modulated by respiratory activity through the autonomic nervous system. Such activity has commonly been quantified by power spectral analysis. Traditionally, the power spectrum describing heart rate variations is divided into low frequency (0.04-0.15 Hz) and high frequency components (0.15-0.4 Hz). The high frequency components reflect modulation of vagal tone, primarily through respiration, and thus reflect parasympathetic activation. The low frequency components reflect modulation of both sympathetic and parasympathetic tone by baroreflex activity. It has been shown that the parasympathetic activity is attenuated during vagal block [1].

ECG based analysis of atrial fibrillation has so far mainly been directed towards amplitude and frequency (cycle length) measures describing the over-all structure of the f-waves. The average fibrillation frequency was introduced in order to represent the average repetition rate of the f-waves for a given interval, [2], [3]. Recently, more detailed methods based on time-frequency analysis were developed for the purpose of presenting trends of the fibrillation frequency with a second-to-second resolution [4], [5]. Using such method, changes in the fibrillation frequency during interventions were possible to track. It was also possible to identify intervals with regular activation patterns.

The purpose of the present method is to study whether or not respiration has a modulatory influence on the atrial cycle length (or inversely the atrial cycle length) of the f-waves during atrial fibrillation. In order to quantify such an influence on the fibrillation frequency, we have developed a new method for detection and estimation of possible modulatory properties in the atrial fibrillation frequency. The method performs prefiltering, suppression of pacemaker spikes, separation of atrial and ventricular activities by spatiotemporal QRST cancellation [6], time-frequency analysis of the resulting residual ECG; estimation of the fibrillation frequency is done in the time-frequency domain using a second-to-second resolution. Special care has been given to the problem of missing or unreliable values in the trend of the fibrillation frequency. The trend is then subjected to power spectral analysis, and the resulting power spectrum is analyzed in order to capture a possible autonomic modulation frequency.

2. Material and acquisition

A group of 8 patients (2 men; mean age 65 years) with permanent AF, AV block III and permanent pacemaker programmed to VVI were used. The patients were studied during baseline rest (7 min), during rhythm controlled respiration (7 min) and during rhythm controlled respiration after full vagal blockade using 2 mg atropine intravenous (5 min rest + 7 min), see Table 2. The full vagal blockade interval was introduced in order to make sure that possible detections during the controlled respiration interval affected the atria via the autonomic nervous system. Each of the eight 27 minute recordings were sampled at 1 kHz using acquisition equipment from Siemens-Elema, Sweden. Only lead V_1 was analyzed although leads V_2 and V_3 were used in the cancellation process.

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Interval	*Length (min)*	*Analyzed data (min)*
Baseline (B)	7	01.00-06.00
Controlled respiration (CR)	7	08.00-13.00
Rest	6	
Controlled respiration post atropine (PA)	7	22.00-27.00

Table 1. Recording protocol

3. Methods

We propose a new method for estimation of frequency modulation in atrial fibrillation. The preprocessing stage is described in Sec. 3.1. Our new method for decomposition of time-frequency distributions of atrial signals which generates frequency trends is summarized in Sec. 3.2. In Sec. 3.3, these frequency trends are further analyzed using spectral analysis in order to estimate the dominating atrial modulation frequency. The spectra are then evaluated as described in Sec. 3.4.

3.1. Preprocessing

Each signal was preprocessed in order to eliminate baseline wander. Then, all pacemaker spikes were deleted using linear interpolation which is possible since a pacemaker spike interval is short compared to the atrial wavelength. Beats were detected and classified into classes based on cross-correlation techniques. Our spatiotemporal QRST cancellation scheme, which uses beat averages from different leads in order to cancel the ventricular activity in each lead, was then applied to the signals [6]. The advantage of using this method is that it combines the beat averages from the different leads in order to compensate for morphologic changes in the QRST compex. Each beat class was assigned its own beat average. The beat classes were constructed such that different types of ectopic beats were classified into different classes while all other beats belonged to one class.

3.2. Frequency trend estimation

Recently, we have developed a new method which decomposes a time-frequency distribution of an atrial signal into a spectral profile, describing the atrial signal waveform, and a set of trends describing e.g. the frequency and amplitude variations of atrial arrhythmias [5]. The main idea is to decompose the time-frequency distribution of the atrial signal such that each spectrum can be represented by a frequency-shifted and amplitude-scaled version of the spectral profile; frequency shifting is possible due to the combination of an harmonic signal and a logarithmic frequency scale. The method is described in a recursive fashion in order to allow adaptation of the spectral profile. A set of parameters are introduced in order to describe the shape and signal-to-noise ratio of the spectral profile.

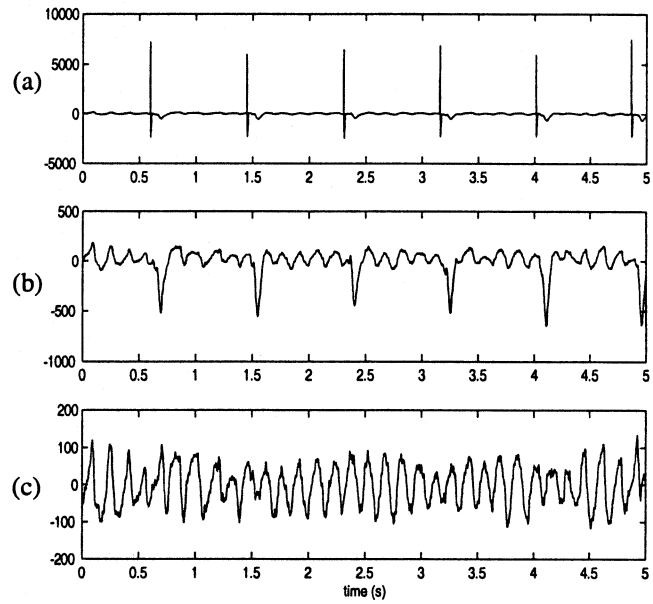

Figure 1. Preprocessing of a 5 seconds interval of atrial fibrillation: (a) Before preprocessing, (b) After pacemaker spike removal and (c) after QRST cancellation. Note that the amplitude scales differ in the different plots.

Such a description is exemplified in Fig. 2 for a one minute segment containing atrial fibrillation. The time-frequency distribution is plotted in Fig. 2a where a fundamental frequency around 6 Hz representing the repetition rate can be seen. In this case, two harmonics representing the shape of the waveform are also observed. The spectral profile, shown as the solid line in Fig. 2b, is recursively updated for each time slice in the time-frequency distribution. The profile is then shifted in order to find the frequency trend which is shown in Fig. 2c. Other parameters such as the exponential decay in the spectral profile (see dash-dotted lines in Fig. 2b) and the amplitude are also presented in the bottom right corner of the figure.

3.3. Spectral analysis of frequency trends

Reliability problems detected in earlier stages, e.g. ectopic beats or a non-valid spectral profile in the frequency estimation procedure, are summarized in a trend $g(n)$, which contains binary values indicating if a sample in the frequency trend, $y(n)$, is reliable or not. When some samples are missing, the autocorrelation function can be estimated by [7],

$$r(k) = \frac{\sum_{n=1}^{N-k} y(n)g(n)y(n+k)g(n+k)}{\sum_{n=1}^{N-k} g(n)g(n+k)} \qquad (1)$$

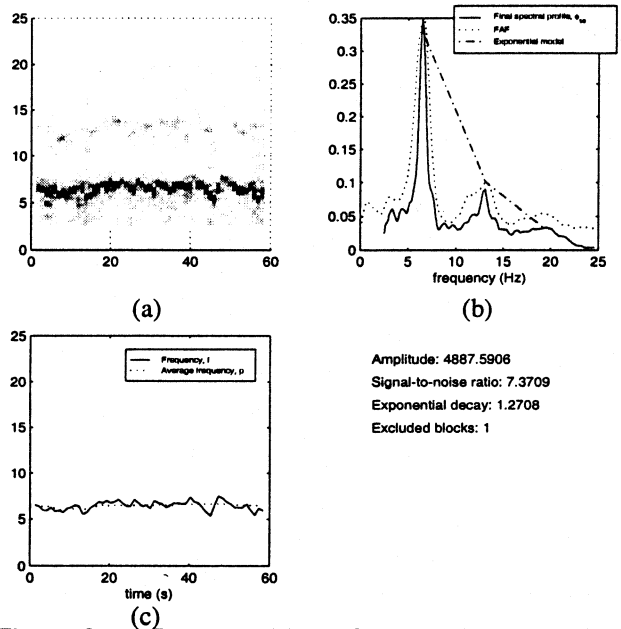

Figure 2. Decomposition of a one 1 minute time-frequency representation: (a) time-frequency distribution of atrial fibrillation signal, (b) spectral profile (solid), power spectrum of the entire minute (dotted) and exponential decay model (dash-dotted), (c) frequency trend (solid) and sliding average frequency (dotted).

where it is required that the trend has zero mean which easily can be arranged ($y(n) = y(n) - m_y$). The power spectrum is calculated using the FFT and possible modulation of the fibrillation frequency is described by this spectrum.

3.4. Evaluation

Two different approaches are considered for evaluation: first, the dominant atrial modulation frequency is assumed to be unknown and the dominant modulation frequency is estimated using peak detection for a given frequncy interval, $[f_0 ... f_1]$, in the modulation spectrum $\mathbf{S}$

$$f_{mod} = \arg \max_{f_0 \leq f \leq f_1} \mathbf{S} \quad (2)$$

In the second approach, it is assumed that a possible peak appears at 0.125 Hz. It is then desirable to know if a peak is present or not. We have here chosen to use the ratio, μ, between the spectral energy at 0.125 Hz, $S_{0.125}$, and the average energy in the HF band, S_{HF}. The reason for this is that the modulation is expected to be related to spontaneous respiration and thus present in the HF band when it is not under rhythm control.

$$\mu = \frac{S_{0.125}}{S_{HF}} \quad (3)$$

4. Results

The results are divided into three parts: first the dominant modulation frequency is presented during controlled respiration. Then, the 0.125 Hz/HF power ratio is presented for baseline rest, controlled respiration and controlled respiration postatropine. Finally, the average fibrillation frequency for the different subjects and intervals are given.

4.1. Dominant modulation frequency

Peak detection in the power spectrum of each frequency trend during CR was performed and the peak frequencies are presented in Table 2.

Subject	$\hat{f}_{mod}$ *CR*
1	0.21
2	0.08
3	0.13
4	0.13
5	0.13
6	0.08
7	0.08
8	0.18

Table 2. Maximum modulation frequency for the eight patients $f_{mod} = \arg\max \mathbf{S}$

In three subjects (3,4 and 5), the maximum frequency was 0.13 Hz (resolution=0.0067 Hz) and corresponds to the respiration frequency. In the other subjects, other frequencies have maximum power. The minimum frequency, f_0, was set to 0.08 Hz.

In two of the subjects (3 and 5) for which 0.125 Hz (0.13 Hz) was found as the maximum frequency, these peaks are prominant in the spectrum (see Figs. 3b and 4b). As can be observed in Figs. 3a and c and 4a and c, these spectral peaks were not present during the baseline rest interval and disappeared after full vagal blockade.

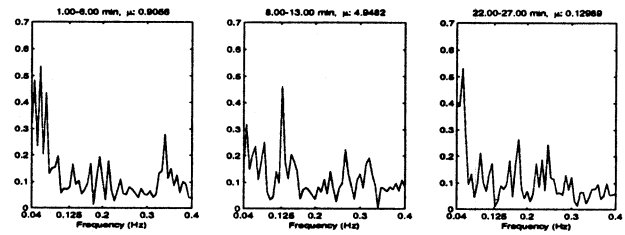

Figure 3. Modulation spectrum for subject 3 during (a) Baseline rest, (b) Controlled respiration and (c) Controlled respiration postatropine

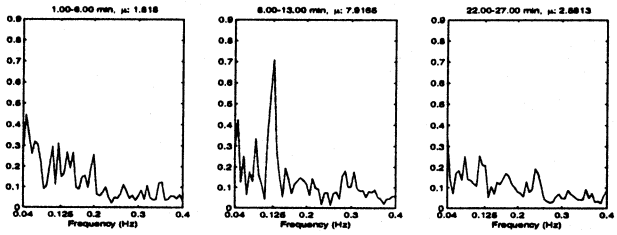

Figure 4. Modulation spectrum for subject 5 during (a) Baseline rest, (b) Controlled respiration and (c) Controlled respiration postatropine

4.2. Ratio between power at 0.125 Hz and HF power

The 0.125 Hz/HF power ratios of the eight subjects are for the three intervals shown in Fig 5. It is observed that subjects 3 and 5 are easily identified by having a much larger ratio during CR than during B and PA. Subjects 4 and 6 also have slightly higher values during CR. It is noted that patient 8 have a slightly lower ratio for CR than during B and a much higher value during PA while subjects 1,2 and 7 have a larger ratio during CR than during B but even larger ratios during PA.

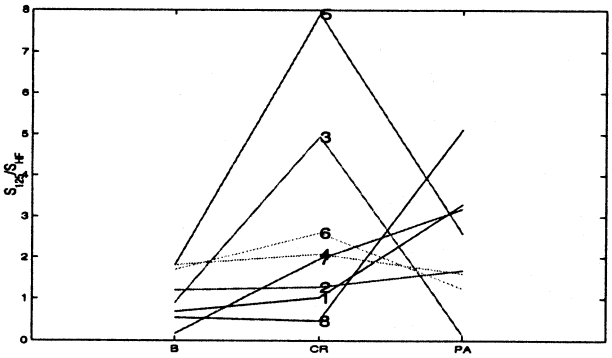

Figure 5. Ratio between power at 0.125 Hz and HF power for the different subjects and intervals.

4.3. Average fibrillation frequency

The average fibrillation frequency of each interval is for each subject plotted in Fig 6. Our result show that there is a decrease in 5 of the 8 patients but an increase in the others. It is noted that the fibrillation frequency is decreased post atropine for patient 3 while it is increased for patient 5. An interesting observation is that the two patients (3 and 5) that were influenced by controlled respiration are among these with the lowest fibrillation frequency.

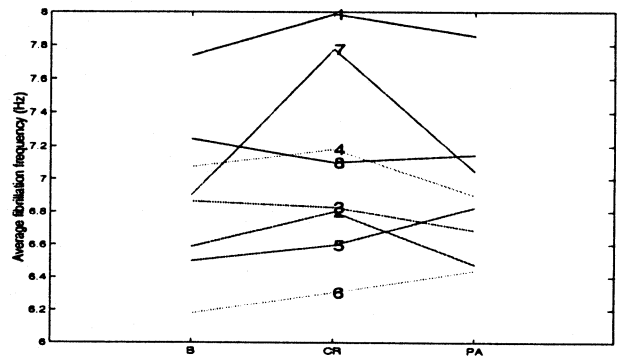

Figure 6. Average fibrillation frequency for the different subjects and intervals..

5. Conclusion

The new method is capable of detecting modulatory frequencies in atrial fibrillation signals. In two of the eight patients the fibrillation frequency was found to be influenced by controlled respiration and the effect could be attenuated by vagal blockade. In a future study, it would be desirable to include a larger group of patients with more organized atrial fibrillation.

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