Derivation of Respiration from Electrocardiogram during Heart Rate Variability Studies

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Abstract

A method was developed to derive the respiration signal from the ECG signal based on the observation that the body-surface ECG is influenced by electrode motion relative to the heart and that fluctuations in the mean cardiac electrical axis accompany respiration. ECG signals were taken from 9 healthy subjects during rest, paced breathing and exercise. The respiration was derived from the recorded ECG signals. The ECG-derived respiration was compared with the original respiration recorded through an impedance pneumography device. The derived respiration shows an excellent correspondence with the original respiration. Statistical analysis indicates that the ECG-derived respiration has a high correlation with the original respiration in the frequency domain.

1. Introduction

As a tool, the spectral analysis of heart rate variability has permitted the biomedical investigator to explore the autonomic nervous systems in a non invasive manner. Some recent studies indicate that spectral analysis of heart rate variability holds a significant potential for the diagnosis of neurocardiac disorders. However, research protocols thus far have focused on establishing the association between the signal power in some fixed frequency bands and their speculated physiological origins. In this method, the heart rate spectrum is divided into three frequency bands. A low-frequency band below 0.05 Hz is correlated with vasomotor control and/or temperature control. A mid-frequency band ranging from 0.06 to 0.15 Hz is associated with baroreceptor-mediated blood pressure control. A high-frequency band ranging from 0.15 to 0.4 Hz has been linked with respiration [3, 4].

It has been known for more than one century that there is an influence of respiratory variation on heart rate, so-called respiratory sinus arrhythmia (RSA). RSA is a rhythmical fluctuation in heart periods at the respiratory frequency that is characterized by a shortening and lengthening of heart periods in a phase relationship with inspiration and expiration, respectively [5]. RSA is being used increasingly as a measure of vagal control of the heart in psychophysiological studies. In spectral analysis of heart rate variability, the best-known and best-defined peak reflects changes in interbeat interval that cycles up and down at the same frequency as respiration. This respiration peak corresponds approximately to the RSA, and it is purely parasympathetic in origin [6]. However, the frequencies of spontaneous respiration are not limited to within the narrow band (0.15-0.4 Hz); they can spread over a wider range. The normal respiration rate can be as low as only a few breaths per minute at rest and as high as up to 40 breaths per minute during intense exercise [7]. Experimental results obtained at the Kessler Institute for Rehabilitation indicate that some subjects have a respiration rate lower than 0.1 Hz at rest and a respiration rate above 0.5 Hz in a test of exercise at 4 times resting metabolic rate.

As the study of power spectral analysis of heart rate variability proceeds, more and more ECG data taken from ambulatory subjects are processed in order to obtain the neurocardiac control information in health and in disease during normal life throughout the day. Since the respiration information is absent, the vagal tone is not obtainable because the respiration frequency of the vagal tone peak can not be determined. Therefore, the neurocardiac control mechanism can not be assessed confidently.

Due to the above mentioned drawbacks, we studied a method to derive the respiration signal from the ECG signal based on the observation that fluctuations in the mean cardiac electrical axis accompany respiration [1, 2].

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2. Methods

The influence of respiration on lead I and lead III ECG signals is shown in Figure 1. In time period t_1, the subject was asked to do a deep inhalation and hold. In time period t_2, the subject was asked to do a deep exhalation and hold. During inhalation and hold, the amplitude of lead I is decreased and that of lead III increased significantly. During exhalation and hold, the amplitude of lead I is increased and that of lead III decreased greatly. This observation can be explained by noting that during inhalation the apex of the heart is stretched towards the abdomen due to the filling of the lungs and diaphragm moving inferiorly and during exhalation the apex of the heart is compressed towards the chest due to the emptying of the lungs and diaphragm moving superiorly. Due to the anatomical changes of the heart in the chest during respiration, the angles of the mean QRS vector vary within a range as shown in Figure 1, which causes the amplitude changes in leads I and III.

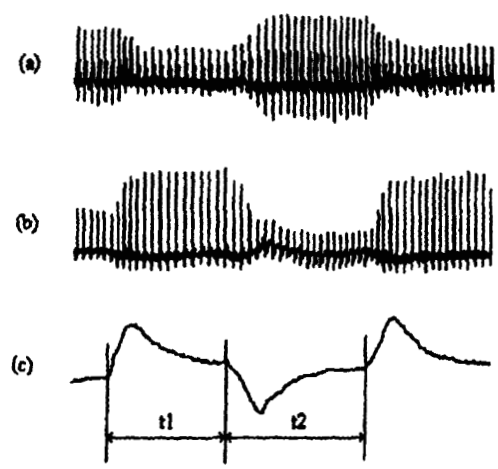

Fig. 1 ECG signals influenced by respiration (a) lead I, (b) lead III, (c) respiration wave

In our study, the area of each normal QRS complex in each of two leads was measured over a fixed time window. Since the window width was fixed, the area was proportional to the amplitude of the ECG signal, hence to the projection of the mean cardiac electrical vector on the lead axis (Figure 2). Assuming that the leads are orthogonal, the arc tangent of the ratio of the areas measured in the two leads yields the angle of the mean axis of the QRS vector with respect to one of the lead axes. The angle values were interpolated to produce a continuous ECG-derived respiratory signal.

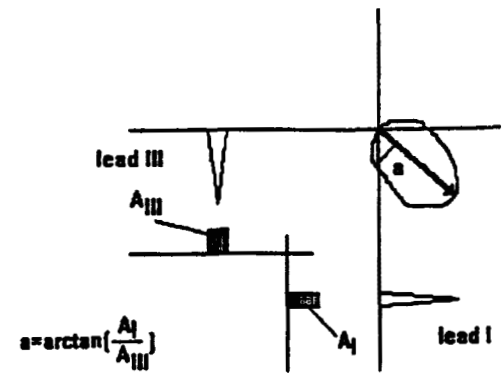

Fig. 2 Calculating the direction of the mean QRS vector axis. A_I: area for QRS complex in lead I; A_{III}: area for QRS complex in lead III; *a:* the angle of the mean QRS vector axis respect to lead III axis

Two lead ECG signals (leads I and III) were collected on 9 healthy subjects during resting, paced breathing (at 8, 12, and 18 breaths per minute), exercise (at 2, 3, and 4 times resting metabolic rate), and recovery using a Quinton Q4000 Stress Test Monitor/Controller (Quinton Instrument, Co., Seattle, WA). In order to compare the ECG-derived respiratory signals with the real respiration, a respiration wave was recorded simultaneously by an impedance pneumography device (RESP1, UFI, Morrow Bay, CA).

The ECG and respiration data analysis was performed on an IBM-compatible 486/50 MHz computer. The data analysis software package used was S-Plus for windows V3.1 (Statistical Sciences, Seattle WA). Since we were interested in the spectrum of the derived respiration signal, the FFT was applied to both the ECG-derived respiration and the original respiration signal recorded from the impedance pneumograph.

3. Results

Figure 3 shows results from one subject. There are four graphs from each test. In Figure 3 (a), the upper left graph is a two minute sample of the original respiration signal recorded from the impedance pneumography device; the upper right graph is the spectrum of the original respiration; the lower left graph is the ECG-derived respiration signal, lower right graph is the spectrum of the ECG-derived respiration. The respiration signals both from recording and derivation are arranged in the same column on the left and their spectra in the same column on the right so as they can be compared easily. In Figure 3 (a), there are 11 respiration

waves both in the original respiration signal and ECG-derived respiration signal. The ECG-derived respiration is 180 degrees out of phase with the original respiration because the signals are interpreted in the opposite way in these two conditions. In the impedance pneumograph, an increase in signal amplitude represents inhalation and a decrease in signal amplitude represents exhalation. In the ECG-derived respiration, we calculated the changes in the angle of the electrical axis of the mean QRS vector using lead III axis as reference. The angle value decreases during inspiration and increases during expiration. Although there is a difference in phase between the original respiration and the ECG-derived respiration, it does not influence the spectra. In the right column of Figure 3 (a), the peak of the spectrum of the ECG-derived respiration is at the same frequency as the peak in the spectrum of the original respiration. Both spectra are very similar in shape. If we examine the remainder of Figure 3, we find that even the derived spectra of the exercise tests (Figure 3 (b)) have a remarkable similarity in shape with the original spectra even though the body is moving during exercise.

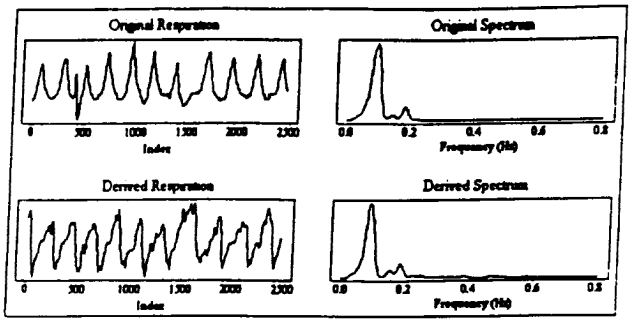

Fig. 3 a

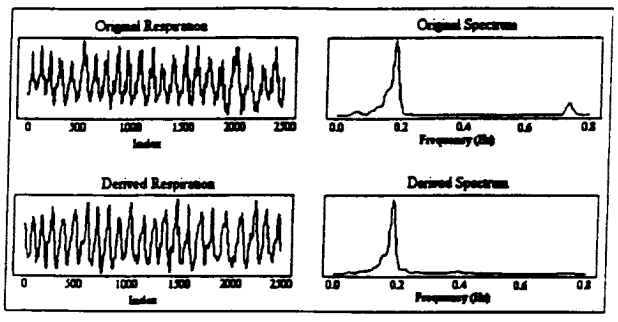

Fig. 3 b

Fig. 3 Comparison of actual respiration with ECG derived respiration and their spectra (a) rest. (b) exercise

To characterize statistically the similarity of the derived spectrum to the original spectrum , we performed a correlation test and a paired t-test. We first calculated the central frequency for both spectra. Figure 4 shows how the central frequency was calculated. The amplitude of the spectral peak was detected first. Then the frequency values at 30% of the spectral peak were located on both sides; i.e. the low frequency f_l and the high frequency f_h in Figure 4. The central frequency f_c was computed so that the area under the spectral curve between f_l and f_c was equal to the area between f_c and f_h.

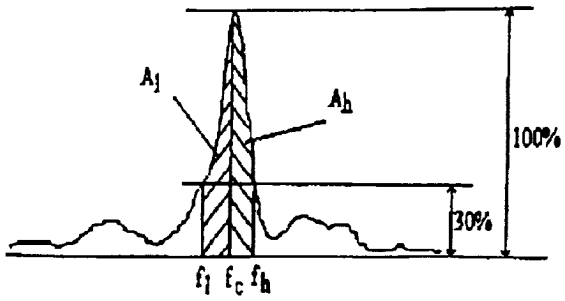

Fig. 4 Central frequency definition

The two samples used in the paired t-test and correlation test were the central frequency of the original spectrum f_{co} and that of the derived spectrum f_{cd}. The two tests were performed on the central frequencies for all tests from all subjects and on each individual test for all subjects. In the paired t-test, our null hypothesis was that the difference between the mean of the derived central frequency μ_{fcd} and the mean of the original central frequency μ_{fco} is zero. The test was set to give a 95% confidence interval for $\mu_{fcd} - \mu_{fco}$. The p values from the paired t-test are all greater than 0.05, indicating that there is no difference between μ_{fcd} and μ_{fco}. The overall correlation coefficient is 0.9977 and the correlation coefficient for each individual test varies from 0.8241 (pacing at 8 breaths per minute) to 0.9983 (exercise at 3 times resting metabolic rate). The high correlation coefficients indicate that there is a strong association between the derived central f_{cd} and the original central frequency f_{co}. This high correlation is also shown in the scatter plot of Figure 5.

Since our goal was to use the derived respiration in cases where the true respiration was not available, we next considered ECG from Holter recordings. In our laboratory, signals from a DMS scientific model 423 Holter monitor were recovered by playing the Holter cassette on a JVC TD-W10 consumer cassette player and

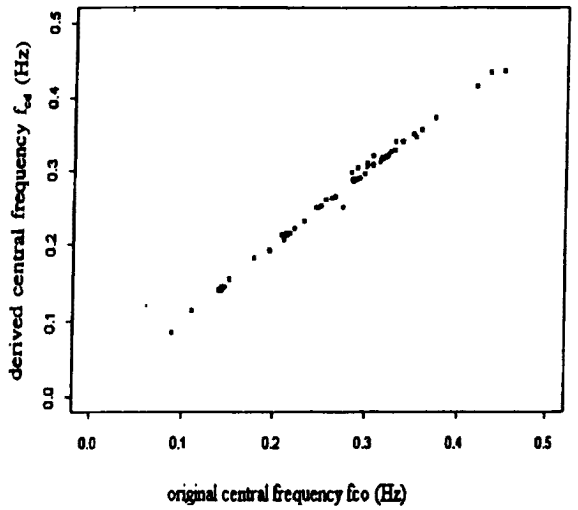

Fig. 5 Scatter plot of the derived central frequency f_{cd} versus the original central frequency f_{co}

sampling at 9.67 KHz to achieve a sampling rate for the original signal of 200 Hz. The experimental procedure was the same as above except that ECG leads I and III were now recorded on the Holter recorder. Impedance pneumography was used to produce a respiration signal for comparison with the derived respiration. Figure 6 again shows the high correlation between derived and original central frequencies for Holter data from 3 normal subjects. Correlation coefficients ranged from 0.9011 to 0.9999 for all tests except for pacing at 8 breaths per minute where a value of 0.6846 was obtained. The overall correlation coefficient was 0.9965.

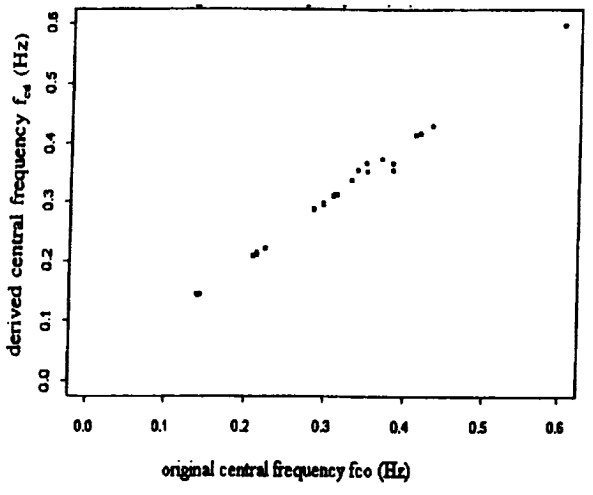

Fig.6 Scatter plot of the derived central frequency f_{cd} versus the original central frequency f_{co} for Holter data

4. Conclusions

The ECG-derived respiration provides a method to obtain the respiration from the ECG signal when respiration information is not directly available. It is therefore possible to do spectral analysis of heart rate variability and determine the frequency of the spectral peak occurring at the respiration frequency. In the future, this method will be applied to the derivation of respiration information from a Holter 24 hour ambulatory recording of ECG, where respiration information is difficult or impossible to obtain. In this way, it will be possible to compare the heart rate variability of normal subjects with that of stroke survivors during "normal" daily activity and during rehabilitation therapy.

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