Methods of Quantifying Respiratory Modulation in Human PR Electrocardiographic Intervals

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Abstract – Respiratory Sinus Arrhythmia (RSA) is a clinically useful measure of heart rate variability which can be estimated by analyzing RR intervals. Respiratory related variation in atrioventricular nodal conduction (which we term RCV) may also be physiologically and clinically significant. RCV can be calculated through its influence on PR intervals. Two methods of estimating RCV in spontaneous and paced breathing are presented; these are cross spectral coherence and cosinor fitting measures. It was found that both techniques show statistically significant respiratory modulation of RR and PR interval duration, although the magnitude of RSA was found to be greater than RCV as expected. Changing posture from supine to standing caused a statistically significant drop in RSA for spontaneous breathing, but not paced breathing. The postural change also reduced RCV for both spontaneous and paced breathing, but this change was not statistically significant.

Keywords – ECG, RR, PR intervals, RSA, respiratory modulation, cross spectral density, cosinor, coherence

I. INTRODUCTION

The respiratory oscillations seen in heart rate are predominantly due to fluctuations in vagal discharge; this modulation effect is usually referred to as RSA. The primary physiological basis for RSA is the degree of vagal efferent activity at the intrinsic pacemaker, the sinoatrial node; RSA is a well recognized and studied phenomenon manifest in the electrocardiogram RR characteristic intervals. The assessment of RSA has found clinical use as a measure of cardiac function in a variety of settings.

A secondary site in the heart under direct neural control is the atrioventricular (AV) node, through which all normally conducted beats pass. The AV conduction delay is directly affected by autonomic neural activity and by the refractory properties of the AV tissue. Given that the AV node is vagally innervated, it seems reasonable that variability in AV conduction delay should also have components influenced by respiration. However, assessment of respiratory atrioventricular conduction variability has not yet been extensively considered; we present two possible methodologies for quantifying RCV using the PR interval, a generally accepted indicator of AV conduction delay.

II. METHODOLOGY

Results are presented from two different RSA and RCV quantification methods applied to data collected from nine normal healthy subjects in sinus rhythm with a mean age of 24 (SD 3 years, range 22-31 years). The data consists of single lead ECG signals of 70 minute duration recorded using the Lewis lead electrode configuration (modified Lead II) to highlight atrial activity. This simplifies the task of automated PR interval extraction. Signals were digitized at a sampling rate of 1kHz using the Biopac™ (Biopac Systems Inc., CA). Respiration was monitored simultaneously by measuring chest wall volume using a calibrated respiratory inductance plethysmograph (Respitrace, Ambulatory Monitoring, Ardsley, NY). Signals were recorded in both supine and standing positions. Two standard tests of cardiac autonomic responsiveness were also carried out – sections of two minute duration deep breathing.

ECG processing is aimed at reliable onset detection of the QRS and P wave; the RR interval is defined here as the time between QRS complex onsets and the PR interval as the time from the P wave onset to the next QRS complex onset. The extraction method used is a wavelet based approach [1], modified to process the Lewis lead data.

A recognized method [2] of measuring the effect of respiratory variation is to calculate the discrete Fourier transform of the RR and PR interval values. The lung volume information is not required for processing. The method is applicable for paced deep breathing but is not suited to spontaneous breathing since the respiratory frequency is not known.

However, if the respiratory signal is known, respiratory effects on RR and PR intervals can be assessed through cross spectral density (CSD). We have defined coherence measures based on CSD estimates applied to the RR and PR interval series and the lung volumes recorded at the corresponding times. These measures are defined as the ratio of the area under the CSD peaks in a specific frequency band to the total area. For deep breathing at 6 min⁻¹ or 0.1Hz, a frequency band of 0.09–0.11Hz is used. For normal breathing, a frequency band of 0.02Hz centered about the location of the peak CSD estimate in the range 0.1–0.4Hz is used.

An alternative measure is a signal averaging approach using both ECG and respiratory signals termed the cosinor method [3]. The beginning and end of each respiratory cycle are defined by the transitions from expiration to inspiration; each respiratory cycle is mapped to the range 0 to 2π radians. The associated PR and RR intervals are determined and then the percentage differences from their respective mean values across the dataset section under analysis. It is then possible to achieve a cosine fit with multiple regression.

III. RESULTS AND DISCUSSION

Fig.1 shows the coherence measure and the cosinor amplitude for data measured under all postural and breathing conditions. For control purposes, results for shuffled data are also presented on the same plots (i.e. where both lung volume measures and intervals are randomly reordered, removing all temporal correlation).
The coherence measure shows statistically significant respiratory modulation on both PR and RR intervals for all subjects and under all postural and breathing conditions, (P<0.05) using ANOVA (ANalysis Of VAriance). The coherence measure shows that the postural change causes a statistically significant decrease in RSA for spontaneous breathing (P=0.0001), but not for paced breathing. This change is in agreement with [2] and [4], indicating a decrease in vagal influence while standing. The coherence measure shows that RCV decreases as a result of the postural change, but this was not statistically significant. There were reductions in the coherence measure in spontaneous breathing compared with paced breathing for both RR and PR interval, which were not statistically significant using ANOVA.

The cosinor amplitude measure shows statistically significant respiratory modulation on both PR and RR intervals in paced breathing except for one subject. For spontaneous breathing, however, the measure was only statistically significant in slightly over half the data sets. Again, the measure shows that the postural change causes a statistically significant decrease in RSA for spontaneous breathing (P<0.0001), but not for paced breathing. There were marked and significant reductions in cosinor amplitude in normal breathing compared with deep breathing for both RR (P=0.0002) and PR interval (P=0.003).

The magnitude of RSA was found to be higher than RCV for each case, using both coherence and cosinor methods, in agreement with the spectral approach applied by [2] who used P peak and R peak detection.

IV. CONCLUSION

The cross correlation spectral density between ECG intervals and respiration, in addition to the cosinor method, provides an insight into cardio-respiratory interaction and particularly atrioventricular conduction variability. This may have diagnostic and prognostic applications in cardiology.

REFERENCES


Fig. 1. Individual and group median measures of RR and PR interval variability, for deep breathing and normal breathing. The measures are shown for analyses performed on the original data (raw) and shuffled versions of these data (shf). Each subject is represented by the same symbol in all plots; the summary measure is the median, with the error bar denoting the 25th and 75th percentile.