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J. D. Schipke, M. Pelzer, G. Arnold

Heart rate variability (HRV) is the consequence of various influences of the autonomic nervous system on heart rate. The influence of the respiration rate on HRV is well accepted (respiratory sinus arrhythmia: RSA). The extent of different respiration rates on HRV however, is not greatly appreciated. In our study, we stored a modified chest wall electrocardiogram on magnetic tape (Tracker, Reynolds) for later analysis (Pathfinder IV, RR-tools, Reynolds). 15 volunteers performed controlled respiration at six different 6 min-intervals: below the low-frequency range (LF) of the power spectrum (0.03 Hz), within LF (0.08, 0.10 and 0.13 Hz), within the high-frequency range (HF; 0.25 Hz), and above HF (0.50 Hz). HRV was expressed in the time domain in terms of standard deviation (SDNN), root mean square successive difference (RMSSD) and the percentage of differences between adjacent normal RR intervals that are > 50 ms (pNN50). After fast Fourier transformation, HRV was expressed in the frequency domain in terms of LF power (0.05–0.15 Hz), HF power (0.15–0.45 Hz), and the ratio (R) of LF to HF.

Heart rate (72 ± 11 min⁻¹) remained unchanged throughout the protocol, indicating a steady haemodynamic state. HRV differed up to 33 % in SDNN, 37 % in RMSSD and 75 % in pNN50 between the different respiration rates. LF power differed up to 72 % (p < 0.10), HF power up to 36 % and R up to 48 % (p < 0.10).

Reproducibility seems satisfactory for the short-term analysis. Respiration rate-induced changes in the time domain measurements were not significant. Because of the large scatter of RMSSD and pNN50, we suggest SDNN as a reliable, easily accessible, and illustrative measurement for assessment of HRV. Respiration clearly affected the frequency domain measurements of HRV via shifts in the respiratory sinus arrhythmia. If major changes in respiration rate are to be expected, these should be interpreted with caution. On the other hand, the characteristic RSA-induced shifts in the frequency range could possibly be useful to determine respiration rate in freely moving individuals. J Clin Basic Cardiol 1999; 2: 92–5.

Key words: heart rate variability, respiration, volunteer, short-term, respiratory sinus arrhythmia

Heart rate variability (HRV) is the result of various influences of the autonomic nervous system on heart rate. While it is known that respiration rate affects HRV (respiratory sinus arrhythmia [1]), the extent of its influence on HRV is not well recognized. We investigated the effect of different respiration rates using linear models that are frequently used in the clinical setting [2] and performed experiments on 15 volunteers with controlled respiration at six different rates. To investigate short-term reproducibility, three respiration rates were chosen from the low frequency range.

Methods

15 healthy, non-smoking volunteers (3 females) were included in the study. They are members of the German Underwater Club of Düsseldorf, all experienced SCUBA divers, ie, they were familiar with different breathing patterns and with apnea. The mean age was 33 ± 10 ys, and mean body weight was 73 ± 10 kg. The heart rate was monitored using a two-channel ECG tape recorder (Tracker, Reynolds).

Experiments were performed with the volunteers in supine position. Six different respiration rates were investigated: one rate was located below the LF range (2 min⁻¹ = 0.03 Hz). Three rates were located within the LF range (5, 6 and 8 min⁻¹ = 0.08, 0.10 and 0.13 Hz). The physiologic rate was located in the HF range (15 min⁻¹ = 0.25 Hz) and another beyond the HF range (30 min⁻¹ = 0.50 Hz). The volunteers monitored their respiration rate using a stop watch. Each respiration cycle lasted 8 min followed by a 2 min pause. Heart rate variability was analyzed during a steady state 6 min-interval within this cycle.

Data were analyzed with an ECG analyzer (Pathfinder 4, RR Tools, Reynolds). Three measures of heart rate variability were obtained in the time domain: standard deviation of the normal-to-normal intervals (SDNN), root mean square successive differences of RR intervals (RMSSD), and percentage of the differences of successive RR intervals greater 50 ms normalized to all differences within the interval (pNN50). After fast Fourier transformation, three measures in the frequency domain were investigated: low frequency (LF) power, high frequency (HF) power and the ratio (R) of LF to HF as an estimation of sympathetic/parasympathetic balance [3]. In accordance with the literature [4–6], low frequency was designated the range from 0.04 to 0.15 Hz and high frequency the range from greater 0.15 to 0.45 Hz.

To assess possible effects of the relatively high respiration rate of 30 min⁻¹ on blood gases, capillary blood samples were taken from the hyperaemised ear lobe of 12 of the 15 volunteers at control, and 5 and 10 min after the onset of tachypnea and analyzed for pO₂, pCO₂ and pH (ABL 505, Radiometer Copenhagen).

Data were processed using a personal computer and the statistics program SYSTAT [7]. A one-way analysis of variance was applied to single protocol steps. To isolate the effect of an intervention, a multiple comparison procedure according to Bonferroni was performed. A p-value less than 0.10 was considered to represent statistical significance. Results are presented as means ± SEM.

Results

Heart rate (72 ± 11 min⁻¹ at control) remained unchanged throughout the protocol.

Depending on the respiration rate, HRV differed up to 33 % in SDNN, 37 % in RMSSD and 75 % in pNN50 between the different respiration rates. LF differed up to 73 % (p < 0.10), HF up to 36 % and R up to 48 % (p < 0.10) (Tab. 1). Scat-
Respiration rate affects heart rate variability

Heart rate variability (HRV) was investigated in 15 volunteers with controlled respiration at six different 6 min-intervals. In the present study, respiration had a considerable effect on the measures of HRV investigated. Thus, not only physical and psychological stress, but also respiration need to be controlled or at least taken into account during short-term analyses of HRV.

In the last five years, about 1,500 papers on heart rate variability were quoted in the Index Medicus. Respiration was referenced in approx. 15 % of those papers, of which only 20 % recognized the interaction between HRV and respiration (eg, [8–12]). The effects of respiration rate on HRV measurements from the frequency domain has already been demonstrated elegantly in more recent studies [13, 14]. In contrast to these studies, the range of respiration rates was further extended in the present study and the effects were also analyzed in the time domain. Extension of respiration rates in this study is not necessarily exaggerated, if one considers that in other studies on the effects of respiration on heart rate in humans even the range between 0.017 and 1.000 Hz [8] or up to 2 or 3 Hz (a yoga breathing technique [15]) was reported. To evaluate possible effects of hyperventilation on interactions between respiration and the autonomic nervous system, blood gases and pH were measured. In addition, three respiration rates were chosen within the low frequency range to prove the reproducibility of the method.

**Time domain HRV measurements**

Standard deviation of the normal-to-normal intervals (SDNN) reflects trends and inconstancies in the average heart rate. SDNN does not permit detailed analysis of autonomic tone since it incorporates all portions of the HRV to a similar degree. At the physiologic respiration rate of 15 min\(^{-1}\) mean SDNN was 57 ms, within the range considered normal [16]; SDNN values less than 20 ms are considered as risk factor for postinfarction patients [17]. Apparently, the variation in SDNN values is relatively large in our quietly lying volunteers but does not seem to be respiration-dependent.

The other two measurements in the time domain, root mean square of successive differences of RR intervals (RMSSD) and percentage of differences of successive RR intervals greater 50 ms (pNN50) mainly reflect HF frequency oscillations and hence parasympathetic activation [2]. Both measurements tended to increase with increasing respiration rate, indicating increased vagal activity. In this study, however, these measurements show prominent scatter.

None of the three time domain measurements of HRV showed a statistically significant dependence on respiration rate. SDNN exhibited the smallest dependency and together with its relatively low scattering between the respiration rates seems to present a reliable, easily accessible, and illustrative measurement for the assessment of short-term HRV.

**Discussion**

Heart rate variability (HRV) was investigated in 15 volunteers with controlled respiration at six different 6 min-intervals. In the present study, respiration had a considerable effect on the measures of HRV investigated. Thus, not only physical and psychological stress, but also respiration need to be controlled or at least taken into account during short-term analyses of HRV.

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on particle deposition and gas uptake in the lungs [16], and that the respiration rate is a potential determinant of pollutant doses to target sites in the lungs [19].

Critique of methods

The present results could have been influenced by the length of short-term intervals. In a previous study on influence of respiration on the RR interval power spectra, only 128 s intervals were analyzed to interpret measurements in the frequency domain [13]. In other studies [20–22] and in ours [23], it was shown that the interval length of short-term measurements from frequency domain can validly be shortened to 5 min. We showed that measurements from time domain can even be shortened to 3 min without significant loss of information [23]. Thus, we feel confident that the interval length of 6 min did not introduce a major error.

The location of the low- and high-frequency ranges within the power spectrum, on the other hand, very likely affects the results of frequency domain measurements: if the low-frequency (LF) range had been extended to lower frequencies, the peak attributable to the respiration rate of 2 min$^{-1}$ (0.03 Hz) would have remained within the LF range. Conversely, if the high-frequency (HF) range had not been truncated at 0.45 Hz, the respiration rate of 30 min$^{-1}$ (0.50 Hz) would have remained in the HF range. However, the location of our LF- and HF ranges is in good accordance with the literature [4–6], and our particular choice of locating them has no effect on the present findings.

It must also be discussed that control of respiration might have modified the results. Control of respiration increases parasympathetic activity [24], and a strict control induces a considerable proportion of variability in the LF range even at higher respiration rates [18, 20]. On the other hand, differences depending on spontaneous or controlled respiration do not seem to exist in normal subjects [14] or in subjects used for the measurement of HRV from the frequency domain. This feature could, in chronic severe mitral regurgitation. Circulation 1993; 88: 127–35.


Pomeranz B, Macaulay RJ, Caudill MA, Kurtz I, Adam D, Gordon D, Kilborn KM, Barger AC, Shannon DC, Cohen RJ, Benson H. Assessment of auto-

Summary and conclusion

Reproducibility of short-term measurements seems satisfactory. Respiration rate via the RSA clearly affects measurements of HRV from the frequency domain. This feature could, in turn, be useful to determine respiration rate from these measurements. Very high respiration rates (eg, 0.5 Hz) are not necessarily associated with hyperventilation. Since RMMSSD and pNN50 have relatively large scattering and are not widely used in the short-term analysis, standard deviation (SDNN) seems to present a simple, reliable and relatively respiration rate-in-sensitive measurement of HRV in the clinical setting. Changes in respiration rate need to be respected, and measurements of HRV from the frequency domain must be interpreted with caution.

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