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Effect of respiration rate on short-term heart rate variability

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 of 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 pO_2, pCO_2 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). Scatter-

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ing within respiration rates from within the LF range was lower on average: 11 % in SDNN, 28 % in RMSSD, and 33 % in pNN50 in the time domain and 14 % in LF, 14 % in HF, and 12 % in R in the frequency domain (Tab. 1).

Analysis of pO_2, pCO_2 and pH at a respiration rate of 30 min^{-1} showed no major changes (Tab. 2). The minimum standard deviation for pCO_2 and pO_2 was 10 and 8 %, and thus comparatively large in relation to the maximum respiration-induced effect of only 1 to 3 %. pH showed only minor scatter: both the standard deviation and the effect of tachypnea were not larger than 2 %.

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.

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 2 to 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.

Table 1. Effect of six different respiration rates (RR) on three measurements of heart rate variability from the time domain (standard deviation: SDNN; root mean square of successive differences of RR intervals: RMSSD; percentage of the differences of successive RR intervals > 50 ms normalized to all differences within the interval: pNN50) and three measurements of heart rate variability in the frequency domain (LF: area under the spectrum of a fast Fourier transformation in the range from 0.05 to 0.15 Hz; HF: area under the spectrum in the range from > 0.15 to 0.45 Hz; R: ratio between LF and RF)

RR (Hz)	0.03	0.08	0.10	0.13	0.25	0.50
SDNN (ms)	71±32	66±40	73±41	67±43	57±37	55±24
RMSSD (ms)	37±22	39±30	48±37	50±44	50±54	47±40
pNN50 (%)	12±13	12±16	16±17	16±18	19±27	21±25
LF (a.u.)	570±290	609±337	692±354	615±336	412±184	399±221
HF(a.u.)	468±343	437±386	496±364	482±371	596±529	437±432
R	1.5±0.7	1.7±0.6	1.7±0.7	1.5±0.4	0.9±0.3	1.2±0.4

Table 2. Effect of tachypnea (30 min^{-1}) on pO_2, pCO_2 and pH in capillary blood samples taken from 12 volunteers at control, and 5 and 10 min after the onset of tachypnea from the hyperaemised ear lobe

	pO_2 (mmHg)	pCO_2 (mmHg)	pH
ctr	92±10	39±3	7.4±0.1
5 min	93±9	38±4	7.4±0.1
10 min	92±9	38±4	7.0±0.0

Frequency domain HRV measurements

Vagal activity is primarily manifested in respiratory sinus arrhythmia (RSA). RSA, in turn, is essentially affected through vagal efferents [18]. Thus, for physiologic, spontaneous ventilation, a large proportion of the activity in the HF range is RSA-induced. In fact, HF power at a respiration rate of 15 min^{-1} was increased compared with the other rates. LF power, in turn, was relatively small at that physiologic rate, so that R, the measurement of sympathetic/parasympathetic balance, was close to unity.

Reductions of respiration rate shifted the RSA into LF range or even below LF range. In accordance with this shift, LF power was increased whereas HF power was reduced, resulting in an increased ratio of HF to LF power. The respiration rate of 30 min^{-1} was also associated with reduced HF power, because, at this rate, the RSA fell beyond the HF range. This led, together with the almost unchanged LF power, to a non-significant increase in R, erroneously suggesting a changed sympatho-vagal balance.

On the other hand, our results are in good accordance with results from other studies that showed that the amount of the RSA-related power in the frequency domain varies with the respiration rate: it is high at low rates and starts to decrease at a rate of about 7 min^{-1} [8, 13]; in our experiments, LF power tended to decrease from below a rate of 6 min^{-1}. Once the RSA is equal to power in a frequency band around respiratory rate, it can shift throughout the spectrum depending on the respiration and thus, obscure the effects under investigation.

This drawback can, on the other hand, possibly become an advantage. We could readily derive respiration rate from our power spectra from the corresponding frequency band. Thus, if short-term steady state ventilation can be achieved, respiration rate from freely moving individuals (eg, athletes or heavy labourers) could be obtained via HRV analysis. In the context of occupational health, this property might become useful if one remembers that ventilation has an important influence

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 to controlled respiration [8]. Because our experienced SCUBA divers are used to moderate respiration control (bubble-induced noise during exspiration while diving), and control was not rigid in this study, this control probably had no significant effect on the present results.

At relatively high respiration rate of 30 min^{-1}, hyperventilation might have introduced an error, in which changes of blood gases (pO_2 and pCO_2) and/or pH might have stimulated central respiratory control. Direct measurement excludes such an effect, however, since the changes in blood gases or pH were minor compared with their spontaneous variation.

In the more recent literature, many physiological parameters are reported to be the subject of chaotic processes [25]. Assuming that relation between respiration rate and heart rate should be non-linear, employment of linear systems to describe changes in heart rate variability would not be appropriate. In fact, although strictly controlled respiration should result in one single peak, two peaks have been reported in the frequency domain [9, 26] supporting a non-linear relation between respiration and heart rate.

The reproducibility of measurements of HRV is still controversial. Reproducibility is described to be either poor [27] or adequate [24] for short-term analyses. In contrast, 24 h analyses seem to provide a satisfactory reproducibility both in the time domain and in the frequency domain, as long as they are assessed via autoregression [28, 29]. Because scattering for the three respiration rates from within the LF range was relatively small compared with scattering for the entire range of respiration rates investigated in this study, our results support the concept that reproducibility in short-term analysis is satisfactory both in time domain and in frequency domain.

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 RMSSD 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-insensitive 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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