

Effect of Respiration and Posture on Heart Rate Variability

I. ŠIPINKOVÁ, G. HAHN¹, M. MEYER¹, M. TADLÁNEK, J. HÁJEK

*Institute of Physiology, Medical Faculty, Comenius University, Bratislava, Slovak Republic and
¹Department of Physiology, Max Planck Institute for Experimental Medicine, Göttingen, Germany*

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Summary

The physiological control system of the heart produces a highly complex pattern of cardiac rhythmicity which is reflected in the variability of heart rate. The aim of this study was to analyse the effects of posture and breathing frequency on the cardiac control system by various noninvasive techniques. Seven healthy subjects (24 ± 5 years, mean age \pm S.D.) were studied in the supine and sitting positions while breathing spontaneously or at a fixed rate (3, 6, 12, 24, 48, 60 breaths.min⁻¹). Time series of instantaneous beat-to-beat heart rates were evaluated by spectral analysis and by the dimensionless approximate entropy parameter. The total spectral power as well as the low (<0.05 Hz) and mid frequency (0.05–0.12 Hz) spectral components were higher in the sitting position. Mean approximate entropy (\pm S.D.) (0.85 ± 0.15 in sitting and 0.87 ± 0.16 in lying subjects) was unaffected by postural changes or breathing frequencies higher than 6 breaths.min⁻¹. Analysis in the frequency domain revealed that the activity of the autonomic components controlling heart rate was modified by ventilation and postural changes, whereas approximate entropy, a unique measure of the complexity and integrity of the cardiac control system, was almost unaffected by respiration and posture.

Key words

Heart rate variability – Breathing rate – Posture – FFT – Entropy

Introduction

Regulation of the heart rate rhythm is a highly complex phenomenon governed by many interrelated feedback systems. The dynamics of the physiological autonomic control system of the heart results in nonlinear rhythmicity of heart action. Several techniques for studying the cardiac control system have been reported. The noninvasive analyses are typically based on the study of either spontaneous or evoked fluctuations of heart rate.

The effect of basic physiological influences, namely of posture and respiration, on the cardiac rhythm was followed in our study. Both of these effects alter the autonomic input to the heart. Changes in respiration modify vagal nerve traffic to the heart and orthostasis leads to increased efferent sympathetic traffic to the heart.

The heart rate variations, resulting from postural and respiratory changes, were studied by different noninvasive methods in the time and frequency domains and by means of a newly introduced

parameter, approximate entropy. By implementation of various noninvasive approaches we tested the ability of the chosen indices to follow the dynamics of the generated heart rate rhythm and to distinguish several information levels in the analyzed heart rate signal.

Methods

Experimental protocol

Experiments, conforming with the principles outlined in the Declaration of Helsinki, were performed on 7 healthy subjects (6 males, 1 female; mean age \pm S.D., 24 ± 5 years) with no cardiovascular history. All experimental subjects were non-smokers and were not being treated with any drugs. The experiments were started at least 2 hours after the last food intake, preferentially at the same time of the day. Body temperature was 36.5 ± 0.3 °C (mean \pm S.D.). Experiments were carried out under indifferent thermal conditions (mean temperature 22.4 ± 1.1 °C).

All measurements were performed with the subject either sitting or lying. In both positions, the

subjects were breathing at different metronome-aided fixed breathing frequencies of 3, 6, 12, 24, 48, 60 breaths.min⁻¹ as well as quietly at an individual spontaneous breathing rate. The subjects were instructed to breathe through the nose, the respiratory depth was not regulated. Prior to the measurements with a newly chosen respiratory rate the subjects were allowed to practice the new breathing pattern. At each respiratory rate 5–6 individual measurements each lasting 3 min were carried out. The measurements were separated by short intervals of 2–3 min.

In each setting, the electrocardiogram (ECG, lead II) and chest wall movements (plethysmographic belt) were continuously registered on a strip chart recorder (Mingograf, Siemens-Elema, Sweden) and read on-line into a computer.

Data evaluation

Time series of instantaneous beat-to-beat heart rate, i.e. tachograms, were determined from the R-R intervals of the ECG identified by computerized R-wave detection. Similarly, the respiratory signals were grouped into sequences of instantaneous breathing rates based on the identification of inspiration onset. The mean heart rates of individual subjects at separate breathing rates for both body positions as well as the mean quiet spontaneous breathing rates were then calculated.

For analysis in the frequency domain heart rate tachograms were interpolated with a frequency of 4 Hz utilizing the algorithm of Berger *et al.* (1986). Equidistant sampling of the registered heart rate was thus ensured by this procedure. Spectral analysis of the heart rate variability was performed using an interpolating FFT algorithm (1024 points, Hanning window). The heart rate power spectra were determined in the range of 0.01 Hz to 1.00 Hz. The power in the low (0.01–0.05 Hz), mid (0.05–0.12 Hz) and high (0.12–0.25 Hz) frequency range, as well as the total power (0.01–1.00 Hz), were computed.

To quantify the cardiac system complexity, a recently introduced parameter, the approximate entropy (Pincus 1991, Pincus *et al.* 1991), was used. The principle of its determination is schematically summarized in Figure 1.

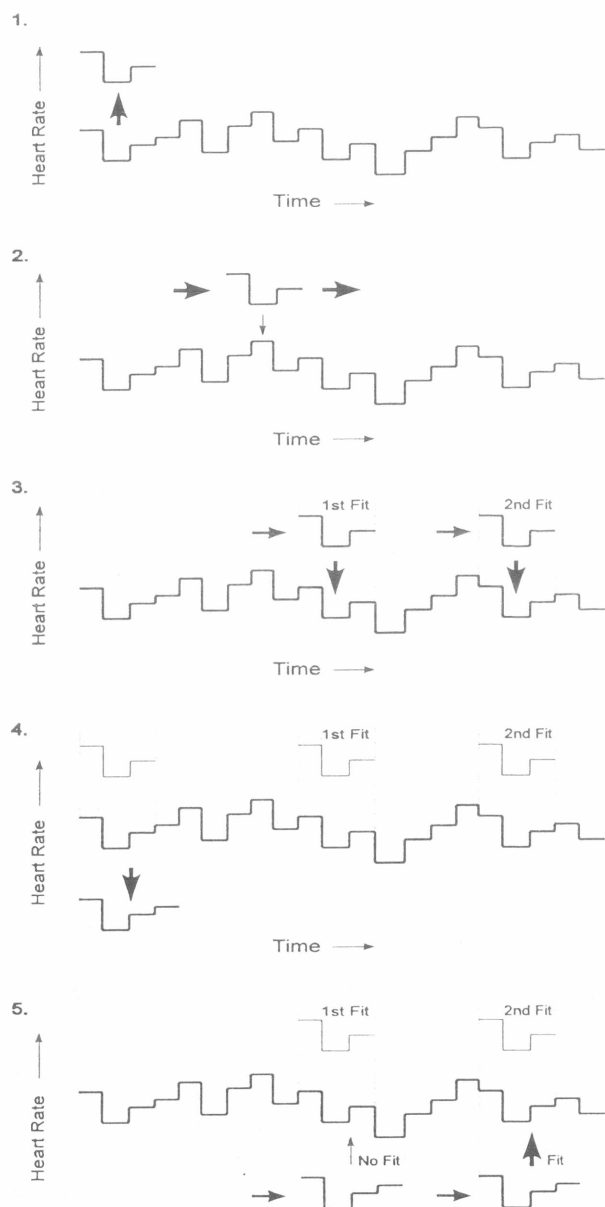


Fig. 1. Computation of approximate entropy from a series of beat-by-beat heart rates (i.e. tachogram). A reference pattern composed of consecutive heart rates is chosen from the registered tachogram (1). Heart rate sequences similar to the chosen reference pattern are thereafter sought in the tachogram (2) and their occurrence is counted (3). After the reference pattern had been compared with all heart rates comprised in the studied tachogram the reference pattern is incremented by one heart beat (4). The heart rate sequences found in the first run are also incremented by one heart beat and compared with the new reference pattern (5). The number of heart rate sequences that remain close to the new reference pattern on its incrementation is then counted. The procedure is repeated for all possible reference patterns that can be chosen from the tachogram, i.e. the first reference pattern equals the first, the second and the third heart rate in the tachogram incremented by the fourth heart rate, the second reference frame is the second, the third and the fourth heart rate incremented by the fifth heart rate, etc.

Briefly, the occurrence of heart rate sequences similar to a reference pattern of a chosen number of beats is counted and the probability that the found heart rate sequences follow closely the increased number of heart beats in the reference pattern is calculated according to the equation

C1

$$\ln \frac{\text{C1}}{N-m+1}$$

C2

$$\ln \frac{\text{C2}}{N-m+1}$$

ApEn(m,r)=
$$\frac{\ln \frac{\text{C1}}{N-m+1}}{N-m+1} - \frac{\ln \frac{\text{C2}}{N-m+1}}{N-m+1}$$

where *ApEn* is approximate entropy, *m* number of beats in the reference pattern, *r* tolerance level for comparison with the reference pattern, *C1* sum of heart rate sequences with *m* heart beats similar to the reference pattern, *C2* sum of heart rate sequences with *m*+1 heart beats similar to the incremented reference pattern and *N* number of beats in the currently analyzed tachogram. In our study *m* was set to 2 and *r* to 0.02, i.e. *ApEn*(2,0.02) was computed.

The significance of differences between the computed data was determined by the Wilcoxon signed-rank test. A P value <0.05 was considered as significant.

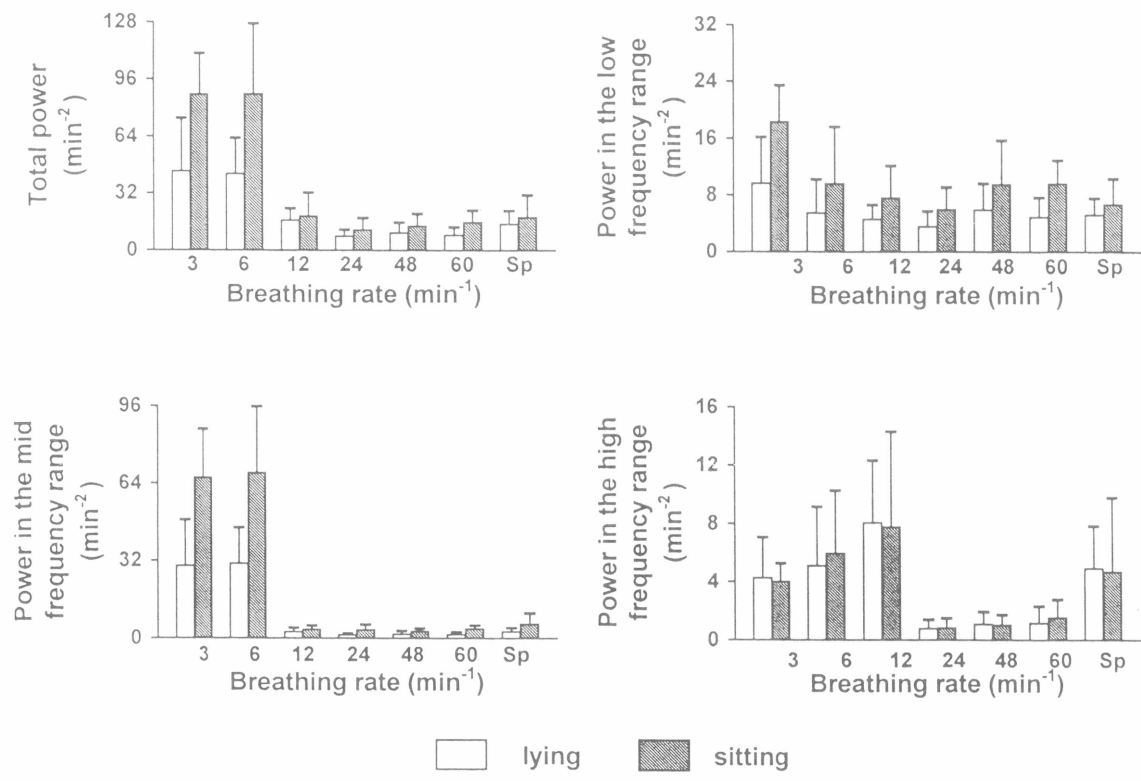


Fig. 2. Spectral analysis of heart rate: mean total power (upper left panel) and power in the low (0.01–0.05 Hz) (lower left panel), mid (0.05–0.12 Hz) (upper right panel) and high (0.12–0.25 Hz) frequency range (lower right panel) in lying and sitting subjects at different fixed breathing rates and during spontaneous breathing (Sp). Data are means ± S.D.

Results

Data analysis in the time domain

The mean heart rates at all breathing rates in both lying and sitting positions are summarized in Table 1. The heart rates in lying subjects are significantly lower than in sitting ones. Mean heart rate in the horizontal position ± S.D. was 67.5±1.2 beats.min⁻¹, and 75.2±1.6 beats.min⁻¹ in the sitting position.

Arterial blood pressure was measured before and repeatedly during each experimental session. Mean systolic blood pressure in sitting subjects was 120 mm Hg, in lying subjects 115 mm Hg, mean diastolic pressure was 80 and 70 mm Hg, respectively. The difference between the respective blood pressures in the two body positions did not exceed 15 mm Hg in any of the subjects.

The spontaneous breathing rate in lying subjects was 13.8±3.6 breaths.min⁻¹, and 15.2±4.2 breaths.min⁻¹ in sitting subjects. The differences between the breathing rates in the two positions were not significant.

Table 1
Mean heart rates \pm S.D. at different set breathing rates and spontaneous breathing (Sp) in lying and sitting subjects.

Breathing rate (min ⁻¹)	Lying (min ⁻¹)	Sitting (min ⁻¹)
3	67.8 \pm 10.3	77.2 \pm 11.3
6	68.2 \pm 11.5	74.6 \pm 11.1
12	69.1 \pm 13.6	76.7 \pm 9.7
24	65.8 \pm 12.2	74.2 \pm 9.1
48	68.2 \pm 9.5	73.6 \pm 11.8
60	66.0 \pm 14.2	75.1 \pm 10.9
Sp	67.2 \pm 10.5	75.7 \pm 11.8
mean \pm S.D.	67.5 \pm 1.2	75.2 \pm 1.6

Data analysis in the frequency domain

The determined total power and the power in the low, mid and high frequency range for all the performed breathing rates in the two body positions are shown in Figure 2. The total power and the power

in the low and mid frequency range were significantly greater ($p<0.02$) in the sitting position compared with the supine position. The power in the high frequency range in upright subjects was not statistically different from the values obtained in supine subjects.

The spectral analysis of heart rate also confirmed the stability of the metronome set breathing rhythm. Narrow respiratory peaks, reflecting the steadiness of the breathing rate, were found in all the analyzed spectra.

Approximate entropy

The mean values of $ApEn(2,0.02)$ at different breathing rates in the two body positions are shown in Figure 3. $ApEn$ at higher breathing rates (>6 breaths.min⁻¹) was unaffected by postural changes or breathing frequencies. However, $ApEn$ during fixed breathing at 3 and 6 breaths.min⁻¹ was significantly lower ($p<0.02$) than at higher breathing rates in both body positions. No significant differences appeared between the corresponding $ApEn$ values in the sitting and lying position at the same breathing rate. Mean $ApEn(2,0.02)\pm$ S.D. was 0.85 ± 0.15 and 0.87 ± 0.16 in the sitting and lying subjects, respectively.

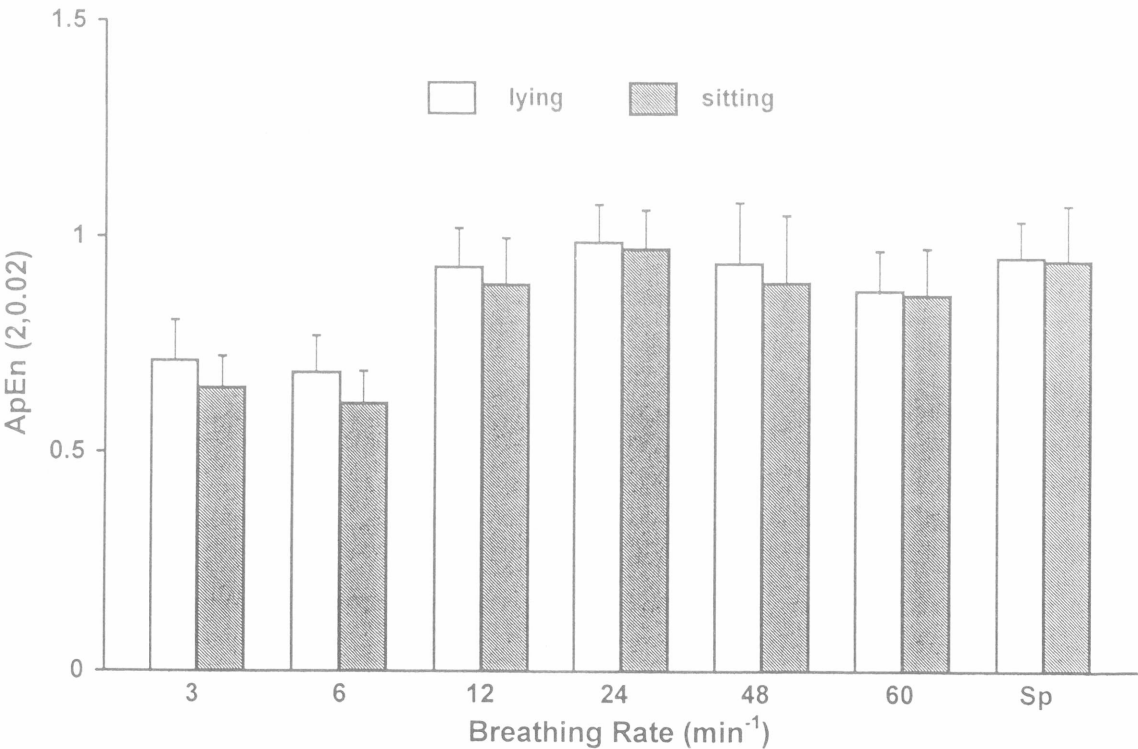


Fig. 3. Mean approximate entropy ($ApEn$) of heart rate \pm S.D. in lying and sitting subjects at different fixed breathing rates and during spontaneous breathing (Sp).

Discussion

The aim of our study was to determine the effect of basic physiological parameters, respiration and posture, on the heart rate rhythm. In contrast with the previous studies that followed the effect of controlled breathing or orthostasis on heart rate variability (Pagani *et al.* 1986, Kamath *et al.* 1987), a large number of combinations of the two mentioned modulatory effects with breathing rates ranging from 3 to 60 breaths.min⁻¹ was used. The results were evaluated not only by the usual methods of heart rate variability analysis, which describe linear or strongly periodical signals, but also by a system parameter, characterizing the overall functional state of the physiologic cardiac control system. In the following text, the different analytical tools applied to the same heart rate data will be discussed with respect to their applicability and to the type and amount of information that can be retrieved.

Analysis of the heart rhythm in the time domain revealed 1) an independence of heart rate on the breathing frequency, which was the result of a relatively constant work of breathing due to spontaneous adjustment of the respiratory tidal volume at different fixed breathing rates, and 2) higher mean heart rates at all breathing frequencies in seated subjects when compared with the supine ones, a phenomenon expected to occur on the background of a different sympathetic-parasympathetic balance due to the change in posture. However, simple analysis in the time domain cannot differentiate if this is due to increased sympathetic or decreased parasympathetic activity, or to opposite changes in tone of both parts of the autonomic nervous system.

Spectral analysis addresses the periodically occurring variations in heart rate. In general, three typical components are identified in the heart rate variability spectrum: a peak at around 0.05 Hz resulting probably from thermoregulatory activity (Sayers 1973) or slow humoral regulation (Akselrod *et al.* 1985), a component at around 0.1 Hz arising from the arterial baroreceptor reflex response (Pagani *et al.* 1986, Peñáz *et al.* 1978) and a component which is located at around 0.25 Hz during spontaneous quiet breathing, reflecting respiratory sinus arrhythmia. These three components of the heart rate spectrum are usually termed as low, mid and high frequency peaks.

The three spectral peaks were identified and quantified in the studied heart rate variability spectra. The power in the low and mid frequency range, as well as the total power reflect the combined effect of sympathetic and parasympathetic activity (Akselrod *et al.* 1985, Berger *et al.* 1989, Saul *et al.* 1990). The higher values found in the seated subjects correspond thus to the shift in the sympathetic-parasympathetic balance. The high frequency peak, however, is affected only by

the parasympathetic output (Chess *et al.* 1975, Randall *et al.* 1991). The power in the high frequency range was not influenced by postural changes in the range of studied breathing rates. The observed significant increase of power in the low and mid frequency range during orthostasis can therefore be attributed solely to enhanced sympathetic tone. A similar conclusion was drawn by Pomeranz *et al.* (1985) in a study on standing and supine subjects breathing at a single fixed rate of 15 breaths.min⁻¹ who were followed after administration of a muscarinic and a β -sympathetic blocker.

As far as the applicability of spectral analysis is concerned in this connection, the following aspects are critical. The method is very sensitive to noise or outliers in the studied signal (e.g. extrasystoles). Large data sets are usually needed for reliable analysis. Attention has to be paid to the selection of a suitable windowing procedure and the interpretation of the data mainly in the low frequency range has to be very cautious. Depending on the frequency of the input perturbations of the cardiac control system, an overlapping of oscillations can occur (e.g. in the current study the power in the low and mid frequency range is influenced by respiration at very low breathing rates).

The advantage of approximate entropy, a parameter derived from the chaos theory, in comparison with other analytical methods is the fact that it addresses the complexity of the generated heart rhythm and the intactness of the cardiac control system. The more complex dynamics are presented as greater entropy. The beneficial feature of ApEn is its principal independence of the mean and variance of the studied heart rate signal and its robustness with respect to signal outliers (Pincus 1991, Pincus *et al.*, 1991).

ApEn is a relative statistical parameter sensitive to the chosen input values – the number of beats in the heart rate pattern and the tolerance level. Pincus *et al.* (1991) recommend that the number of data points (i.e. heart rates) in the analyzed time series should be at least 10^m, where *m* equals the number of heart beats in the reference pattern. Hence, a reference pattern of two heart beats was chosen in the current study, where individual measurements lasted 3 min. A tolerance level corresponding to 10–25 % of the mean signal standard deviation is recommended. Therefore, the tolerance level of 0.02 was used for ApEn computation. To check the effect of different tolerance levels, test computations were also performed at higher levels (0.025, 0.03 and 0.035). At rising tolerance levels, a fall in ApEn was observed as a consequence of declining sensitivity and loss of detailed information.

The currently computed ApEn values of healthy young subjects are comparable with the results of Kaplan *et al.* (1991). Although no exact information on the mean spontaneous breathing rate is given in this

paper, the reported value of 0.90 in supine spontaneously breathing subjects is close to 0.95 determined in our study under similar ventilatory and postural conditions.

An interesting phenomenon is the finding of low ApEn at low breathing rates (3 and 6 breaths.min⁻¹). A comparable observation was made by Saul *et al.* (1989) who analyzed the cardiac system transfer characteristics in man during several respiratory rates (the range of performed respiratory rates was more narrow than in our experiments and was applied in the form of a broad band input signal). It was found that in upright subjects one part of the power spectrum located at around 0.07 Hz cannot be accounted for merely by respiration. They suspected that an independent resonance or other unknown non-respiratory physiological mechanisms could play a role in this frequency range. The generally low ApEn values at low breathing rates seem to speak in favour of a similar conclusion, as more regular signals (i.e. resonance) produce lower ApEn values.

The source of this greater regularity may arise from a reflex reaction elicited by carotid and aortic baroreceptors. The baroreflex reaction is expected to be pronounced during deep inspirations. Venous return to the heart is temporarily reduced and leads to decreased cardiac output and arterial blood pressure. Activation of baroreceptors results in a reflex increase in heart rate. In the present study, the subjects breathing periodically at low breathing rates inspired large tidal volumes. This led to regularly occurring high negative intrathoracic pressure with an accentuated baroreflex reaction detected by ApEn due to the marked regularity of heart rate. In nonlinear dynamics such stereotype behaviour is referred to as the slaving mechanism (Goldberger *et al.* 1984).

The generation of the heart rate rhythm is also under the influence of chemoreceptors. Breathing controlled by a dictated frequency and accompanied by

spontaneously assumed tidal volumes may result in hyperventilation (Vizek *et al.* 1991) and the lowered PaCO₂ may, in turn, affect the heart rate. The particular pattern of breathing may differ with respect to the duration of inspiration and expiration especially during very low breathing frequencies. The different patterns of breathing will elicit different patterns of chemoreceptor and mechanoreceptor stimulation, which may also be related to the results obtained with breathing rates of 3 and 6 breaths.min⁻¹.

ApEn at spontaneous and at higher fixed breathing rates was shown to be independent of the posture and breathing rate indicating that the integrity of the cardiac control system was not changed. This finding suggests that the observed significant differences in ApEn in healthy and elderly human subjects (Kaplan *et al.* 1991) cannot be attributed to differences in the sympathetic and parasympathetic balance due to posture or to various breathing rates. In the future, it would be of interest to study patients with known disturbances of the cardiac control system and to determine if significant changes in ApEn are associated with the disease and if ApEn can be applied as a new diagnostic tool.

Conclusion

The modulatory effect of breathing rate and posture on the cardiac control system was studied by several noninvasive techniques. The cardiac control system was characterized for a wide range of breathing rates in two body positions. The shifts in the sympathetic and parasympathetic balance were discernible by analysis in the time and frequency domain, whereby more detailed information was derived from the spectral analysis. The information on the overall functional state of the control system of the heart was obtained by implementation of the system parameter – approximate entropy.

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MUDr. I. Šipinková, CSc., Department of Anaesthesiological Research, Centre of Anaesthesiology, Emergency and Intensive Care, Medical Faculty University of Göttingen, Robert-Koch-Str. 40, TL 195, D-37075 Göttingen, Germany.