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Observations on Respiratory and Cardiovascular Rhythmicities During Yogic High-Frequency Respiration

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Summary

Yogic high-frequency respiration – kapalabhati (KB) – was studied in 24 subjects from a point of rhythmicity. Respiratory movements, blood pressure and R–R intervals of ECG were recorded in parallel and evaluated by spectral analysis of time series. Respiratory signals during KB were modulated by 0.1 Hz rhythm in 82 % of experiments. This component was also present in R–R intervals and blood pressure during KB. Frequency (0.2–0.3 Hz) was observed in 67 % of respiratory records. The presence of the component 0.2–0.3 Hz in respiration was dependent on resting respiratory frequency. This frequency component was reduced in R–R intervals but increased in blood pressure during kapalabhati as compared to that at rest. The occurrence of both frequency components in respiration during KB supports the hypothesis about the integrative role of cardiovascular and respiratory rhythms in physiological states characterized by altered respiratory frequency.

Key words:
Respiration – Blood pressure – Rhythms – Spectral analysis – Yoga

Introduction

Spontaneous rhythmical variations in cardiovascular and respiratory parameters were already described in the last century (Hering 1869, Mayer 1876, Knoll 1885). In animals, the usual way to investigate the rhythms in blood pressure, heart rate and in phrenic nerve activity is to study these parameters in asphyxia or during artificial ventilation. In humans, several studies demonstrated the presence of rhythms at a frequency of spontaneous respiration, i.e. 0.2–0.3 Hz, and with frequency 0.1 Hz in blood pressure, heart rate and striated muscle tone during voluntary apnoeas (Matthes 1951, Wagner 1954, Schmidt van der Heyden and Koepchen 1970, Stančák Jr. *et al.* 1987). However, a study using voluntary respiration with a frequency high above the usual frequency of spontaneous respiration is lacking. Yogic breathing exercises comprise high-frequency respiratory patterns and offer thus a suitable object for study of cardiovascular and respiratory rhythms in humans. One of these exercises, kapalabhati (KB), consists of rapid respiration through the nose with the usual frequency 2–3 Hz. This breathing exercise involves active expirations performed by voluntary contraction of the anterior abdominal wall. The inspirations are realized by releasing the contraction of the anterior abdominal muscles (Kuvalayananda 1966, Sivananda 1987, Vishnudevananda 1988). Several rhythms were observed in respiration and heart rate in most of our electrophysiological investigations of KB. Their frequencies were in the ranges 0.2–0.3 Hz and 0.07–0.13 Hz. The preliminary results based on spectral analysis of respiratory and R–R interval signals during KB were reported previously by Fabián *et al.* (1982). The present paper intends to formalize the observations on rhythmical amplitude modulation of respiration, blood pressure and R–R intervals of ECG which occurs during KB. The observations refer to the following points: 1. Which frequency components modulate respiratory and cardiovascular signals during KB? 2. How does the occurrence of various frequency components in respiration during KB depend on the resting respiratory frequency?

Methods

Twenty-eight experiments were performed on 24 subjects in two independent experimental series. All the yoga practitioners mastered KB already for periods from 1 year up to 20 years. The first series included 11 experiments on 7 subjects (4 males, 3 females, mean age 43.9 years, SD 16.0). The subjects performed KB for 10 min after 5 min of initial rest in this series. The subjects in the second series were recruited from the Sivananda Yoga Organization. This sample was composed of 17 subjects (11 females, mean age 36.3 years, SD 11.4 and 6 males, mean age 32.7 years, SD 4.1). Three 5 min periods of KB (KB1–KB3) were performed after the initial 5 min resting period (R1). The successive periods of KB were separated by three spontaneous respiratory cycles. Two 5 min resting periods (R2, R3) were recorded after the last KB period. All subjects were sitting in a cross-legged position with eyes closed throughout the whole experiment.

ECG was recorded by the Frank system. Blood pressure was monitored nonivasively by Ohmeda Finapress 2300 BP Monitor. Abdominal respiratory movements were recorded by a pressure transducer Tesla LPD 165 in the first series. Thermistor electrode placed in a small nasal mask was used in the second experimental series. The frequency response of the thermistor electrode enabled faithful reproduction of frequencies up to 0.067 Hz. Lead X of ECG, analog pulse pressure and respiratory signals were fed into DEC PDP 11/34 computer and digitized by 5 ms sampling interval in the first series. In the second series, the IBM PC system described elsewhere (Stančák Jr. *et al.* 1991) was used. The outputs were ASCII files of beat-to-beat values of R–R intervals, systolic blood pressure (SBP), diastolic blood pressure (DBP) and respiration in both recording systems. The respiratory signal was resampled by 10 Hz.

Time series analysis was performed systematically on data from second experimental series. R–R intervals, SBP and DBP were interpolated by the lowpass-filter method. The sampling frequency was set on 2 Hz. The respiratory signals were smoothed by 9-point 4th order polynomial filter to eliminate the high-frequency component associated with individual abdominal strokes and to prevent aliasing. The signals were then reduced by 5 so that 2 Hz sampling frequency was obtained. Linear and quadratic trends were removed from all series if present. Maximum entropy spectral analysis was used. The order of the autoregressive AR process was set either automatically according to Akaike AIC criterion or by the operator. There were outliers in cardiovascular signals which precluded a proper estimation of power spectral densities in about 12 % of signals. The method of robust spectral analysis developed by Fabián (1988) was used in these signals. The method of robust spectral analysis is a combination of maximum entropy spectrál analysis and non-linear robust transformation of the original series. The transformation is performed by assumptions of certain theoretical distribution function and degree of robustness. Robust spectral analysis was performed *via* facilities of ROBAN program system (Fabián *et al.* 1989). Sixty frequency components were calculated in the range 0.0 – 0.5 Hz in all power spectral densities.

Joint-interval histograms were occasionally plotted. This method depicts the time series in two-dimensional way. Every point Si of the series is defined by Si and Si-1 values of the series. The scatters provided by the method enable to interpret features of the signal others than the rhythmicity. Kolmogorov-Smirnov test of differences between two empirical distributions and test of contingency were used (Reisenauer 1974).

Results

Original records

The original record of KB abdominal respiratory movements is shown in Fig. 1. The fast respiratory movements of 2 Hz frequency were modulated by slower rhythms with frequencies about 0.3 Hz and 0.13 Hz in the present case. The faster component was observed on polygraphic records in 9 out of 11 experiments in the first experimental series and in 10 out of 17 experiments in the second experimental series, i.e. in 67 % of all experiments. The component 0.08 – 0.13 Hz frequency was observed in 7 cases out of 11 in the first series and in 16 cases out of 17 in the second series, i.e. in 82 % of the experiments. The difference in occurrence of both rhythmical components in two experimental series was nonsignificant according to the test of contingency ($X^2(1) = 1.23, p > 0.05$).

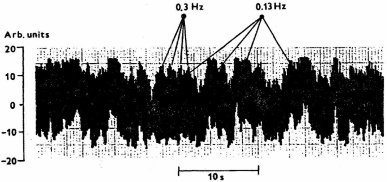

Fig. 1
Record of abdominal respiratory movements during kapalabhati. The amplitudes of fast abdominal excursions are modulated by slower rhythms with frequencies about 0.3 Hz and 0.13 Hz.

Important rhythmicities

Local maxima were identified in respiration, blood pressure and R – R interval spectra in all resting and KB records in 17 subjects of the second experimental series. The local maximum was classified as a primary peak if it had been the largest peak in the given spectrum. All other local maxima besides the primary ones were classified as secondary peaks. The numbers of spectral peaks which were found in both conditions at every frequency were summed across subjects and plotted in the form of a histogram (Fig. 2). The primary peaks are represented by the filled area and the secondary peaks by the empty area of the histograms.

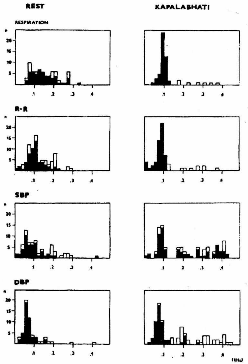

Fig. 2
Histograms of spectral peaks of respiration, R–R intervals, systolic (SBP) and diastolic (DBP) pressure at rest (left) and during kapalabhati (right). Filled area represents the primary and empty area represents the secondary spectral peaks.

The shapes of histograms (Fig. 2) were quite different at rest (left panel) and in KB (right panel) in all four types of signals. There was higher occurrence of peaks around 0.1 Hz in respiration in KB compared to rest. In fewer cases the faster component with the frequency between 0.2 – 0.4 Hz was observed in KB. Similarly to respiration, there were more peaks around 0.1 Hz in R–R intervals in KB than at rest and less peaks around 0.2 Hz in KB compared to rest. The largest difference in systolic blood pressure (SBP) between KB and rest was in the range of 0.2 – 0.35 Hz. There were more peaks in KB spectra compared to the resting ones in this frequency region. The same difference consisting in an increased occurrence of peaks in the range 0.2 – 0.35 Hz was found in diastolic blood pressure (DBP). Another component with a frequency 0.4 Hz was found in SBP and DBP spectra of several subjects during KB. We consider this component as a high-frequency noise due to occasional distortions of pulse waves by the fast respiratory strokes. This artifactual effect was stronger in SBP than in DBP. The differences between the empirical distributions were tested by the Kolmogorov-Smirnov test separately for every type of signals. The differences surpassed 99 % probability in all four types of signals.

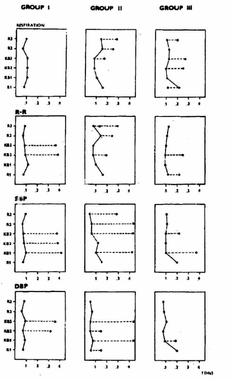

Fig. 3
Typical patterns of spectral peak frequencies under resting conditions (R1 – R3) and during kapalabhati (KB1 – KB3) in respiration, R – R intervals and systolic and diastolic blood pressure. Three groups of subjects were created according to the occurrence of primary spectral peaks (full circles) and secondary spectral peaks (empty circles) in respiratory spectra in R1 period.

Individual patterns

The primary and the secondary peaks in successive phases of the experiment were plotted separately for all 17 subjects of the second experimental series. Three groups of spectral patterns were found according to the primary and the secondary spectral peaks in respiration . Their representative cases are shown in Fig. 3. Filled circles represent the primary peaks and empty circles represent the secondary peaks.

Group I (n=6) was characterized by the low respiratory frequency during the initial resting period falling in the region 0.075 – 0.150 Hz. These subjects had only one spectral peak around 0.1 Hz in subsequent KB and resting conditions. The pattern of spectral peaks in R – R intervals, SBP and DBP followed closely the pattern of respiratory spectral peaks. A representative case of Group I patterns is shown in Fig. 3 (left).

Group II (n=6) had one respiratory peak in the range 0.16 – 0.3 Hz during the initial rest. The primary peak was always found by 0.1 Hz during KB. However, another peak appeared in the range 0.18 – 0.30 Hz during 2nd or 3rd KB periods. This component persisted in at least one of the post-exercise periods. The component 0.1 Hz and occasionally another component with frequency 0.2 – 0.3 Hz were observed in SBP and DBP during KB. Unlike the R – R interval spectra, the patterns of blood pressure spectral peaks were not so close to the respiratory ones. An illustrative case of Group II subjects is shown in Fig. 3 (center).

Group III (n=4) was characterized by two spectral peaks with frequencies 0.1 Hz and 0.2 Hz during the initial resting period. Secondary peaks with frequency in the range 0.2 – 0.3 Hz were observed in respiration both during KB and during post-exercise periods. Although both components were found in R – R interval and blood pressure spectra, the correspondence between peak frequencies in respiration and cardiovascular signals was loose in this group of subjects (Fig. 3 right). One subject could not be categorized according to the above described groups.

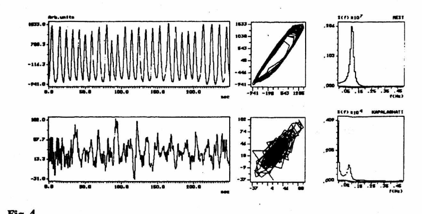

Fig. 4

Raw signals, joint-interval histograms and power spectra of respiration at rest (upper panel) and during kapalabhati (lower panel) in one subject.

Other differences

Although the present study deals primarily with respiratory and cardiovascular rhythmicities, another important difference was observed between KB and rest in respiration. This difference is documented in Fig. 4 which represents the raw signals, joint-interval histograms and power spectral densities at rest (upper panel) and during KB (lower panel) in one Group I subject. Note the regular rhythm of 0.1 Hz frequency in the resting respiratory signal. About the same frequency was found during KB. Yet the respiratory signal in KB was more scattered. This difference is illustrated by joint-interval histograms. The histograms show periodical character of both signals according to their looping characters. The loops at rest unlike to those from KB are uniform pointing to a difference in regularity of the main periodicity. This observation was supported by differences in the estimated order of AR process in these two types of signals. The estimated order of the AR process was usually 3–5 in most of the resting respiratory signals but it was above 7 in the KB signals.

Discussion

Respiration, blood pressure and R–R intervals are modulated primarily by 0.1 Hz rhythm during KB. This finding is supported by visual analysis of original records and by empirical distributions of spectral peaks. The component 0.1 Hz occurs independently of the pre-exercise respiratory frequency.

The rhythm 0.1 Hz is traditionally considered as a cardiovascular rhythm (Matthes 1951, Wagner 1954, Peňáz 1958, Golenhofen and Hildebrandt 1958). Most of the recent authors hypothesize its origin in the nonlinearity of the baroreceptor feedback loop (Delius *et al.* 1972, De Boer *et al.* 1985, Madwed *et al.* 1989). The hypothesis about baroreflex involvement in generation of 0.1 Hz rhythm provides a reasonable basis for explanation of 0.1 Hz oscillations in respiratory movements during KB. Two alternative routes should be considered. One route could be through modulation of activity of striated muscles of the anterior abdominal wall by a baroreflex-mediated rhythm. This alternative is supported by previous findings of Schmidt Van der Heyden *et al.* (1970) and Schmidt Van der Heyden and Koepchen (1970) about 0.1 Hz oscillations in amplitude of the patellar tendon reflex synchronized with vasomotor waves. Baroreflex stimulation was found to alter the tone of various striated muscles (Gellhorn 1970) including the abdominal ones (Bishop 1974). The other route is represented by baroreflex modulation of medullary neurones which are the part of the central respiratory generator. Not only the sensitivity of the baroreflex depends on the phase of respiration (Seller *et al.* 1968, Eckberg and Orshan 1980) but also the stimulation of carotid baroreceptors provokes changes in timing of inspiration and expiration *via* a central mechanism (Eldridge 1980).

Another rhythmical component in the frequency region 0.2–0.3 Hz was found in respiration and cardiovascular parameters. It always has a lower amplitude and it occurs in fewer cases in respiration than the 0.1 Hz component. Its presence is determined by the respiratory frequency in the pre-exercise period, i.e. it occurs only in subjects whose respiratory frequency before exercise was equal or higher than 0.16 Hz. The finding about the dependence of 0.2–0.3 Hz component on the pre-exercise respiratory frequency suggests a central interaction between the 0.1 Hz component and the component 0.2 – 0.3 Hz. Synchronization of the respiratory and 0.1 Hz rhythm is well documented. During the state of synchronization of both rhythms only one high-amplitude 0.1 Hz oscillation was observed in blood pressure and heart rate (Peňáz 1958, Golenhofen and Hildebrandt 1958, Hyndman *et al.* 1971). Our findings show that synchronization once achieved spontaneously at rest (Group I pattern) tends to be maintained also by the absence of eupnoeic respiratory movements. If both oscillators are not entrained during spontaneous breathing, i.e. the respiratory frequency is outside the region of 0.1 Hz rhythm (Group II and Group III patterns), both rhythmical components appear during high-frequency respiration.

The observations about the increased occurrence of spectral peaks in SBP and DBP and decreased occurrence of R – R interval peaks in the range 0.2 – 0.3 Hz in KB compared to rest are congruent with our previous results which were discussed elsewhere (Stančák Jr. *et al.* 1991, in press). Increase of spectral power in the band 0.15 – 0.33 Hz in SBP and DBP and decrease of R – R interval variability in the same band was observed in KB in that paper. The authors suggested that the decrease of respiratory sinus arrhythmia, which normally modulates the respiratory fluctuations of blood pressure (De Boer *et al.* 1985), was the factor which has lead to the increase of blood pressure variability in the band 0.2 – 0.3 Hz.

Still one more important difference between KB and rest was found in respiration. This difference consists in the unstable nature of the periodicities of respiration during KB. The difference was documented by joint-interval histograms and original respiratory signals in Fig. 4. Approximately the same frequency was found in both signals in this Group I subject. Yet the variability of the dominant periodicity was much greater in KB than at rest. This observation can be explained by the absence of regular afferent stimulation from the chest and lungs which normally helps to maintain the regular eupnoeic rhythmicity of the central respiratory generator (Cohen 1979). Two physiological properties of KB exercise contribute to lower regular afferent stimulation from the chest and lungs. First, the chest is maintained fixed in an intermediate chest position throughout the exercise, second, the tidal volume is low in KB – only 130 ml on the average (Karambelkar *et al.* 1982).

Another physiological mechanism possibly accounting for the occurrence of spontaneous respiratory and cardiovascular rhythms is represented by chemical stimulation of brain-stem oscillators. Recently, Haxhiu *et al.* (1989) were able to evoke sustained blood pressure waves after stimulation of the ventral medullary surface by chemical activating agents in cats. Kawahara *et al.* (1989) and Kawahara and Yamauchi (1990) observed a rhythm in phrenic nerve activity at a frequency of spontaneous respiration in vagotomized, artificially ventilated cats during hyperoxic hypocapnia. KB involves increased minute ventilation reaching 21 l/min (Karambelkar *et al.* 1982, Bhole 1982, Gore and Gharote 1987), slightly increased alveolar ventilation 6.5 l/min (Bhole 1982) and decrease in end-tidal CO_2 concentration (Karambelkar *et al.* 1982) although the latter effect is 3.5 times less than after voluntary hyperventilation of equal duration. These ventilatory characteristics of KB support the hypothesis that the occurrence of rhythms in respiratory movements and cardiovascular parameters during KB might be facilitated by chemical regulatory mechanisms. However, the proportion of the chemical reflexes and other factors, like fatigue of respiratory muscles or stimulation of nasal passages, in inducing of the spontaneous respiratory and cardiovascular rhythms during high-frequency respiration remains to be clarified by further investigations.

Our study shows that the rhythmical modulation of respiration and cardiovascular parameters can be observed, besides spontaneous breathing, also during yogic high-frequency respiration. Furthermore, both frequency components, i.e. 0.1 Hz and 0.2 – 0.3 Hz, were found during apnoeas in blood pressure (Matthes 1951), R – R intervals (Stančák Jr. *et al.* 1987), the patellar tendon reflex (Schmidt Van der Heyden and Koepchen 1970) and in the reaction-time task (Engel and Hildebrandt 1964). Thus convergent evidence exists that both rhythms occur during different breathing patterns and apnoea. We suggest their integrative role in the organism in the sense that central structures responsible for these rhythms overtake the integration of various physiological systems when the eupnoeic respiratory movements are out of play due to various behavioural demands. The integrative role of respiratory and cardiovascular rhythms is in line with neurophysiological concepts of a "common brain stem system" (Langhorst *et al.* 1980) and "cardiorespiratory neurones" (Koepchen *et al.* 1983). Both these concepts suppose flexible integration of brain-stem neurones into regulation of all effector organs according to the actual pattern of afferent stimulation received from peripheral and visceral sensory organs.

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