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ARTICLE

Acute Effect of Kapalbhati Yoga on Cardiac Autonomic Control Using Heart Rate Variability Analysis in Healthy Male Individuals

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ABSTRACT

Kapalbhati is well known for improving cardiovascular health. But there are some reports of heart attacks while practising kapalbhati. We hypothesize that ill-effect of kapalbhati could be because of autonomic dysfunction to heart. In the present study, we aim to understand the acute effect of kapalbhati yoga on heart rate dynamics using heart rate variability (HRV) analysis. Resting heart rate (HR) varies widely in different individuals and during various physiological stresses, particularly, exercise it can go up to three-fold. These changes in heart rate are known as heart rate variability (HRV). Variability in heart rate reflects the control of autonomic system on the heart and which can be determined during brief periods of electrocardiographic (ECG) monitoring. HRV measures the effect of any physical exercise on the heart rate using time- and frequency-domain methods. Frequency-domain method involves power spectral analyses of the beat-to-beat intervals (R-R intervals) variability data. When total power vs. frequency, fast fourier transform analysis of R-R intervals data is done, it shows three well-defined peaks/rhythms in every individual, which contain different physiological information. Thus, the total spectral power of R-R intervals data can be divided into three components or bands viz., the very low frequency (VLF) band, the low-frequency (LF) band and the high frequency (HF) band. VLF represent very long time-period physiological phenomenon like thermoregulation, circadian rhythms etc. and thus are not seen in short-term recordings like in this work. LF band power represents long period physiological rhythms in the frequency range of 0.05-0.15 Hz and LF band power increases as a consequence of sympathetic activation. HF band represent physiological rhythms in the frequency range of 0.15-0.5 Hz and they are synchronous with the respiration rate, and arise due to the intrathoracic pressure changes and mechanical vibrations caused by the breathing activity. In this work, twenty healthy male volunteers were trained in kapalbhati yoga and their ECG waveforms (2 min.) were obtained while doing kapalbhati (breathing at 1 Hz frequency for 2 min.) and were compared with the baseline (just 2 min. before the start) and post-*kapalbhati* (immediately 2 min. after completing the practice) HRV data. Our results showed a significant decrease in the time-domain measures i.e., NN50, pNN50 and the mean heart rate interval during-*kapalbhati* when compared statistically to the respective before practice or "pre"-kapalbhati ($p < 0.05$, student's paired t-test) values. Frequency-domain indices showed that during-kapalbhati there is a significant increase (~48%) in the LF band power which suggests sympathetic activation and a significant increase (~88%) in the low frequency to the high frequency power ratio (LF/HF ratio) which indicates sympathetic system predominance. A significant decrease (~63%) in the HF component was also noted during-*kapalbhati* as compared to the "pre-*kapalbhati*" values which shows decrease in parasympathetic tone. Thus, these results suggest that during-*kapalbhati* there is drastic increase of sympathetic tone whereas parasympathetic activity is reduced. We propose these changes in autonomic system control on heart are responsible for the myocardial ischemic attacks induced during kapalbhati in some individuals.

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1. Introduction

Yoga is a union of body and the mind. Kapalbhati is a fast (high frequency) yogic breathing technique which involves short, strong and rapid forceful exhalations at the rate of 1 to 2 Hz and inhalation is automatic such that mind is directed to the flow of breath [1]. Regularly practicing kapalbhati improves cardiac and mental health which makes it a very popular technique [2-6]. Despite reported health benefits, there are case reports available, suggesting people had undergone myocardial ischemic attacks while performing kapalbhati [7,8]. In a study, a decrease in cardiac vagal tone during kapālabhāti was reported due to decreased sensitivity of arterial baroreflex and increase in systolic blood pressure and low frequency blood pressure oscillations [7]. In another case, spontaneous pneumo-thorax caused by kapālabhāti was reported [8]. As Kapalbhati is a very popular technique for improvising cardiovascular health and practised widely by people of all age groups (especially by old age), it is very important to determine its deleterious effect on cardiovascular function if any. Intense exercisers say that watching heart rate variability (HRV) during any physical excercise can give an edge and a boost in workout performance. When HRV returns to normal after exercise, this shows that the person has fully recovered from workout, which tells it's safe to exercise again and helps avoid overtraining. Thus, HRV is a good indicator to study any ill-effects of kapalbhati on cardiac function. In this work, we aim to study the acute effect of kapalbhati practice on cardiac autonomic tone using HRV analyses while kapalbhati is done, so that maximum health benefits can be explored by every individual without deleterious side-effects. HRV analysis involves assessment of successive heart beat periods (between successive ECG wave R-R peak-to-peak intervals) using time- and frequency-domain methods. Time-domain measures involve measurement of different variables on the beat-to-beat intervals data. Frequency-domain indices involve decomposing R-R intervals into power-frequency spectrum which divides R-R intervals into three spectral components/bands viz., very low frequency band; the high frequency (HF) (0.15-0.5 Hz) and the low frequency (LF) (0.05-0.15 Hz) band [9-11]. Although there are several controversies in the scientific community regarding using LF power to indicate sympathetic activation but still till date it is used for the same [12]. On the other hand, HF band power and time-domain measures mainly reflect parasympathetic tone. Few reports are available on the effect of kapalbhati on cardiac function using HRV analyses. Raghuraj *et al* studied the effect of kapalbhati on autonomic function using HRV analyses before and after the practice using twelve male volunteers. They found significant increase in the LF power and LF/HF power ratio and significant decrease in the HF power immediately after doing 1 min. kapalbhati (at 2 Hz) when compared to the immediately before or "pre"-kapalbhati values [13]. Telles *et al* studied the effect of kapalbhati on HRV using thirty-eight male volunteers and found a significant decrease in NN50, pNN50 and the mean RR intervals while and after doing three rounds of 5 min. kapalbhati at 2 Hz but they found no change in any of the frequency domain measures during and after kapalbhati [14]. The above reports suffer from several limitations. First, they measure the effect of kapalbhati practice before and after the practice not during kapalbhati. Second, reproducibility of their results because of few reports is highly questionable. Thus, the present study is designed to study the acute effect of kapalbhati yoga on heart rate using HRV analyses during its practice.

2. Materials and Methods

2.1 Subjects

Twenty healthy young untrained male volunteers (age range, 18-35 years) not suffering from any known cardiovascular or neurological disorder and are not taking any medication were selected for the study. They were trained by a yoga instructor for one week in 1 Hz kapalbhati using a stop watch before the actual experiment. We made sure that participants did not have tea, coffee or meals at least two hours before participating in the study.

2.2 Recording

Volunteers were asked to sit in padmasana with back erect, rigid and wrist over the knee. Two ECG chest leads were placed below the collar bone on the left and right side and third one on the left fifth intercostal space at the mid axillary line (V_6). Baseline ECG waves

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(pre-*kapalbhati* data) were recorded with normal breathing for 2 minutes in the steady state immediately before starting kapalbhati and stored at 250 Hz frequency using portable Alice PDX polysomnography instrument (Philips-Respironics, USA). Then, volunteers were asked to do kapalbhati for 2 minutes (60 expirations in 1 min.) i.e. at 1 Hz frequency and ECG waves were continuously recorded (During-*Kapalbhati* data). After that, subjects took normal breaths, keeping the posture erect and ECG was recorded continuously for another 2 minutes (post-*kapalbhati* data). Thus, it took a total of six minutes to complete the entire recording from one individual and recording is done only once per volunteer. All the experiments were performed between 10:00 to 12:00 a.m. every time. ECG amplitude data (in μV) of pre-, during- and post-*kapalbhati* was separately exported into separate ASCII files and analyzed. The recording during-*kapalbhati* was collected only for 2 minutes because our aim in this study is to measure HRV during-*kapalbhati*, which can only be done continuously for two minutes by the recruited individuals.

2.3 Analysis

HRV was analysed in the pre-, during- and post-*kapalbhati* ECG data. ECG waveform was reconstructed from the amplitude data using the Kubios 2.2 HRV software (Biomedical Signal Analysis Group, Department of physics, University of Kuopio, Finland) and analysis was done using the time- and frequency-domain HRV methods. Power spectrum was separated into low frequency (LF, 0.04 – 0.15 Hz) and high frequency (HF, 0.15 – 0.4 Hz) components and a ratio of the LF and HF expressed in normalized units (n.u) (LF n.u./HF n.u.) was calculated. The following time-domain measures were analyzed: (i) mean R-R interval (the mean of the intervals between adjacent QRS complexes), (ii) mean heart rate, (iii) NN50 (the number of interval differences of successive NN intervals greater than 50 milliseconds), (iv) pNN50 (the proportion derived by dividing NN50 by the total number of RR intervals) in the 2 min. recordings. All the changes between the pre-, during-, post-*kapalbhati* were compared using paired student t-test. A p-value < 0.05 was considered statistically significant. All statistical analysis was done using Graph pad prism software.

3. Results

Table 1 shows comparison of different HRV variables pre-, during- and post-*kapalbhati*.

Table 1. Comparison of heart rate variability pre-, during- and post-*kapalbhati* in healthy male individuals

Variables	Pre-Kapalbhati (n=20)	During-Kapalbhati (n=20)	Post-Kapalbhati (n=20)
Heart rate (bpm)	73.59 ± 1.89	92.63 ± 1.62	77.00 ± 1.64
LF (nu)	38.31 ± 4.58	73.50 ± 3.67	55.74 ± 4.31
HF (nu)	53.49 ± 4.59	19.57 ± 2.80	39.15 ± 4.02
LF/HF (nu)	0.69 ± 0.19	5.88 ± 1.61	1.78 ± 0.88
Mean RR (seconds)	0.87 ± 0.02	0.66 ± 0.02	0.85 ± 0.03
NN_{50} (count)	31.5 ± 9.46	7.3 ± 4.11	21.7 ± 6.09
pNN_{50}	33.67 ± 5.23	5.40 ± 2.59	30.36 ± 4.43

Notes: All values are expressed as Mean +/- Standard Error. bpm - beats per minute; LF – Low Frequency; nu – normalized units; HF – High Frequency.

3.1 Time-domain Analysis

Table 1 shows mean ± Standard Error values (n = 20) of mean heart rate; pre, during and post-*kapalbhati*. During-*kapalbhati*, mean heart rate increased by 19 beats per minute from pre-*kapalbhati* values. Pre-*kapalbhati* mean heart rate data was statistically compared with during-*kapalbhati* mean heart rate data using paired student t-test. The two-tailed p-value came out to be 0.0006 which is < 0.05 which suggests that the change in mean heart rate is significant. Mean R-R interval, NN50 and pNN50 were also computed. During-*kapalbhati*, a significant decrease in mean R-R interval, NN50 and pNN50 was noted as compared to "pre-" values. The decrease in mean RR intervals was extremely significant (p-value 0.0001<0.05). The decrease in NN50 was also found to be very significant (p-value 0.0046 <0.05).

3.2 Frequency-domain Analysis

Table 1 shows mean ± Standard Error values (n = 20) of normalized LF power, pre, during and post-*kapalbhati*. It is noted that there is an increase in LF power during-*kapalbhati* when compared with the pre-values. When the difference was checked for statistical significance between pre- and during-*kapalbhati* LF power, the p-value was less than 0.0001 which is < 0.05 suggesting extremely significant difference between the two LF power. Similarly, a reduction in HF values was observed during-*kapalbhati* as compared to the pre-values. When the difference was checked for statistical significance between pre- and during-*kapalbhati* HF power, the p-value was less than 0.0001 which suggests extremely significant difference. The increase in LF and reduction in HF band power lead to a significant increase in the LF/HF

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ratio during-*kapalbhati* as compared to the baseline.

4. Discussion

The aim of the present study is to understand the control of autonomic nervous system on heart during kapalbhati practice. Heart rate variability (HRV) analysis is used to study the aim. A HRV analysis involves calculations on beat-to-beat intervals (R-R intervals) data using time- and frequency-domain methods. Time-domain methods (i.e., the mean R-R interval, NN50 and pNN50) which mainly reflect parasympathetic or vagal tone were calculated pre, during and post-*kapalbhati* practice. We observed a statistically significant decrease in the mean heart rate, mean RR interval, mean NN50 and mean pNN50 values during-*kapalbhati* as compared to the "pre-values" which suggest that during-*kapalbhati* there is decrease of parasympathetic autonomic tone. Frequency-domain measures (LF power, HF power and LF/HF nu power ratio) were also calculated. LF/HF nu ratio which is an indicator of balance between sympathetic and parasympathetic activity (also known as sympatho-vagal balance) is also calculated. Increase in LF power indicates sympathetic activation although its physiological interpretation is still controversial; it is thought both sympathetic and parasympathetic contributions can be involved in LF power activity. On the other hand, HF power mainly reflects parasympathetic activity. Under control or resting conditions, parasympathetic activity is the main activity which controls the heart rate and sympathetic activity is very low. In this study, we observed a statistically significant increase in the LF power during-*kapalbhati* as compared to the "pre-values" which signifies sympathetic system activation by the act of kapalbhati. Also, LF/HF power ratio increased drastically during-*kapalbhati* from "pre-values" and remained elevated post-*kapalbhati*, this shows that kapalbhati practice leads to sustained sympathetic activation and sympatho-vagal balance is disturbed which can be detrimental to heart if maintained for prolonged intervals. Furthermore, a decrease in the HF component was observed both during and immediately post-*kapalbhati* which suggests sustained inhibition or reduction of the parasympathetic activity to the heart. We propose over-activation of the sympathetic nervous system and withdrawl/inhibition of the parasympathetic activity is responsible for the deleterious cardiovascular effects reported due to kapalbhati practice reported in some individuals [7,8]. Heart rate is controlled by various neuronal and hormonal factors. Heart rate is determined by the rate of depolarization of the cardiac pacemaker. Pacemaker tissue is found in the sinoatrial (SA) node, the atrioventricular (AV) node, and the Purkinje tissue of the heart. But, SA node is considered the main pacemaker as it depolarizes faster than other pacemaker tissues and mainly controls the heart rate. SA node cells receive neuronal input from both sympathetic and parasympathetic (vagal) nerve fibers. Parasympathetic input to SA node decreases heart rate whereas sympathetic input increases it. During exercise, sympathetic activation leads to the release of catecholamines mainly epinephrine and norepinephrine from the post-ganglionic sympathetic nerve fibers innervating the heart which act on the SA node cells adrenergic receptors causing cells depolarization leading to increase of the heart rate. Also, norepinephrine is synthesized in the adrenal medulla which upon stimulation during physical, mental or emotional stress is released in the blood. Norepinephrine while in the blood circulation acts on all the body tissues including the heart. Norepinephrine is also released in the blood as a result of spillover (known as norepinephrine spillover) from the post-ganglionic sympathetic nerve fibers. Thus, norepinephrine exerts its sympathetic effect by acting both as a neurotransmitter and a hormone, thereby, increasing the heart rate during excercise. We think during kapalbhati yoga practice physical stress is induced which release norepinephrine both via the sympathetic nervous system to the heart and also from the adrenal medulla in the blood. In the heart, it acts on sinoatrial node (SA node) increasing the heart rate and in the blood stream it cause constriction of most of the blood vessels leading to increase of blood pressure. In the same way, parasympathetic activity to the heart is primarily mediated by post-ganglionic vagal nerve endings. Vagal nerve endings upon stimulation release acetylcholine neurotransmitter which acts on SA node slowing its conduction thus actively modulating vagal tone and slowing/relaxing the heart rate. This is to note that similar to our results an increase in the LF/HF power ratio and a decrease in the HF power has been reported previously immediately before myocardial ischemic attacks in coronary artery disease patients [15].

The exact mechanism by which doing kapalbhati influences autonomic tone to heart remains a question. It has been proposed earlier that at relatively high respiration rate of 60 $min.^{-1}$, hyperventilation might lead to changes in blood gases or pH which might stimulate central respiratory control. The respiratory and cardiovascular centers are closely associated in the brain stem. Thus, it is very likely that voluntarily changes in respiratory rate like while doing kapalbhati can bring about changes in cardiovascular parameters. As we have observed during kapalbhati there is sympathetic activation which

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can increase heart rate and systolic blood pressure thus if it is performed beyond a certain time and at greater frequency can be detrimental to heart. In medical practice, hyperventilation (high frequency breathing such as kapalbhati) is used as a provocation method for the induction of seizures in suspected epileptic individuals. Thus, hyperventilation procedures are good for health only when performed with proper guidelines and the effect is highly individual-specific. All these factors justify our effort to measure HRV during kapalbhati, thereby helping in standardization of parameters (frequency, duration) for which if kapalbhati is done would provide maximum benefit to a particular individual. There are several limitations to our study like it has been done on healthy, young male individuals although kapalbhati is very popular among obese individuals and also amongst people of all age groups. Thus, this study needs to be repeated in individuals with cardiovascular disorders, obese people and people of all age groups where the results can be very different or even opposite. Furthermore, in this study we have used only HRV analyses as an indicator to study the effect of kapalbhati on cardiovascular function but there are several other cardiovascular parameters as well like baroreflex sensitivity, continuous blood pressure monitoring which should also be studied during kapalbhati practice to understand the complex mechanisms of the cardiac autonomic control from the research perspective.

Conflict of Interest

There is no academic or financial conflict of interest.

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Supplementary Data

Table 2. Mean ± Standard Error values (n = 20) of heart rate (HR) pre-, during- and post-*kapalbhati* yoga

S. No.	Heart Rate pre-Kapalbhati	Heart Rate during-Kapalbhati	Heart Rate post-Kapalbhati
1.	62.86	90.32	70.58
2.	64.28	89.26	60.54
3.	82.91	92.09	81.91
4.	76.91	98.02	81.68
5.	86.26	91.23	75.90
6.	60.20	83.67	75.64
7.	64.48	76.69	61.50
8.	89.45	91.11	91.11
9.	64.95	90.29	82.77
10.	80.15	110.14	80.81
11.	73.54	100.72	76.38
12.	74.91	96.02	79.68
13.	72.42	89.32	75.63
14.	75.95	101.29	80.67
15.	80.91	99.91	79.68
16.	70.26	89.02	75.28
17.	77.50	97.12	83.32
18.	79.12	90.26	83.15
19.	69.52	85.62	75.29
20.	65.71	90.51	68.52
	73.59 ± 1.84	92.63 ± 1.62	77.00 ± 1.64

Table 3. Mean ± Standard error values (n = 20) of ECG RR intervals (in seconds) pre-, during- and post-*kapalbhati* yoga

S. No.	RR intervals (in seconds) Pre-*kapalbhati*	During-*kapalbhati*	Post-*kapalbhati*
1.	0.95	0.63	0.87
2.	0.93	0.67	1
3.	0.72	0.65	0.74
4.	0.78	0.61	0.73
5.	0.74	0.67	0.8
6.	1	0.75	0.84
7.	0.94	0.78	0.97
8.	1.16	0.91	1.21
9.	0.93	0.66	0.87
10.	0.75	0.5	0.77
11.	0.90	0.60	0.82
12.	0.88	0.62	0.96
13.	0.72	0.64	0.74
14.	0.75	0.59	0.68
15.	0.95	0.75	0.79
16.	0.93	0.77	0.96
17.	0.92	0.65	0.86
18.	0.74	0.50	0.76
19.	0.86	0.67	0.85
20.	0.89	0.58	0.82
	0.87 ± 0.02	0.66 ± 0.02	0.85 ± 0.02

Table 4. Mean ± Standard Error values (n = 20) of pNN50 (%) values pre-, during- and post-*kapalbhati* yoga

S. No.	pNN50 (%) Pre-*kapalbhati* values	During-*kapalbhati*	Post-*kapalbhati*
1.	44.3	0	36.9
2.	30.6	0	31.0
3.	6.2	0	11.4
4.	2.7	0	2.5
5.	82.3	45.9	76.1
6.	84.5	13	75.4
7.	29.2	25.7	14.3
8.	1.1	0	1.1
9.	63.9	4.5	46.2
10.	20.5	0	30.3
11.	40.5	0	31.5
12.	35.3	0	34.5
13.	11.2	0	15.4
14.	5.7	0	7.5
15.	32.53	0	30.53
16.	38.21	0	30.89
17.	40.82	10	35.59
18.	36.53	8.91	32.52
19.	37.45	0	33.95
20.	29.9	0	29.8
	33.67 ± 5.23	5.40 ± 2.59	30.36 ± 4.43

Table 5. Mean ± Standard Error values (n = 20) of normalized power of low frequency (LF) component of heart rate variability (HRV) pre-, during- and post-*kapalbhati* yoga

S. No.	LF pre-Kapalbhati	LF during-Kapalbhati	LF post-Kapalbhati
1.	17.9	88.3	63.2
2.	70.3	94.5	90.6
3.	36.6	48.1	35.8
4.	18.3	79.7	74.7
5.	34.2	90.6	40.3
6.	48.3	86.1	22.6
7.	31.1	72.6	59.8

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8.	39.4	89.1	48.5
9.	20.3	63	49.9
10.	40.7	81.3	46.1
11.	84.24	94.50	91.27
12.	14.00	38.51	27
13.	17.7	71.0	63.5
14.	59.26	89.39	85.48
15.	75.5	80.6	69.1
16.	44.2	66.4	55.01
17.	35.98	56.05	40.71
18.	37.87	54.49	51.21
19.	22.7	65.39	50.29
20.	17.7	60.5	49.9
	38.31 ± 4.58	73.50 ± 3.67	55.74 ± 4.31

Table 6. Mean ± Standard Error values (n = 20) of high frequency (HF) power component of heart rate variability pre-, during- and post-*kapalbhati* yoga

S. No.	HF pre-Kapalbhati	HF during-Kapalbhati	HF post-*kapalbhati*
1.	81.7	11.6	36.7
2.	29.7	5.5	9.4
3.	62.6	51.6	63.5
4.	81.4	20.2	25.2
5.	65.7	9.4	59.6
6.	51.6	13.9	77.3
7.	68.9	27.2	40.2
8.	60.5	10.9	51.5
9.	79.1	36.8	49.3
10.	59.1	18.6	53.9
11.	54.49	37.87	51.21
12.	56.05	35.98	40.71
13.	15.73	5.49	8.71
14.	62.6	5.16	43.5
15.	34.38	17.5	33.20
16.	35.94	16.8	22.2
17.	37.89	23.8	36.33
18.	25.00	16.41	15.23
19.	28.52	15.23	27.7
20.	81.7	11.6	36.7
	53.63 ± 4.59	19.57 ± 2.80	39.15 ± 4.02

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