Original Article

Cerebral Hemodynamics and Vagally Mediated HRV Associated with High- and Low-frequency Yoga Breathing: An Exploratory, Randomized, Crossover Study

Chetan Aacharya[1], Shirley Telles[1,2], Sachin Kumar Sharma[2]

[1]Department of Yoga, University of Patanjali, [2]Patanjali Research Foundation, Haridwar, Uttarakhand, India

Abstract

Background: Volitionally modifying respiration leads to changes in middle cerebral arterial (MCA) blood flow. The effect of changes in breath rate on MCA blood flow has not been reported. **Aims and Objectives:** To determine the effect of slow (bumblebee yoga breathing) and fast (high frequency yoga breathing) yoga breathing techniques on MCA blood flow and vagally mediated heart rate variability. **Materials and Methods:** Thirty participants (mean age ± standard deviation, 27.3 ± 4.2 years) were assessed on 2 separate days practicing either high frequency yoga breathing (HFYB, breath frequency 54.2/min) or slow frequency bumblebee yoga breathing (BBYB, breath frequency 3.8/min) in random order to determine the effects of changes in breath frequency on MCA hemodynamics. Assessments included transcranial Doppler sonography, vagally mediated heart rate variability (VmHRV), and respiration. **Results:** Both HFYB and BBYB (i) reduced MCA flow velocities, i.e., peak systolic, end diastolic, and mean flow velocities, and (ii) increased MCA pulsatility indices. There was an increase in VmHRV during BBYB based on increased power in high frequency (HF) and low frequency (LF). LF reflects VmHRV for slow breath frequencies. In BBYB the average breath rate was 3.8 breaths/min. In contrast, VmHRV decreased during HFYB (based on reduced HF power; repeated measures analysis of variance, $P < 0.05$, all cases). **Conclusion:** Hence, irrespective of the differences in breath frequency, both HFYB and BBYB appear to reduce MCA flow velocities and increase the resistance to blood flow bilaterally, although the VmHRV changed in opposite directions. MCA velocity and pulsatility changes are speculated to be associated with low global neural activity during yoga breathing.

Keywords: *Middle cerebral artery blood flow, transcranial Doppler, vagally mediated heart rate variability, volitional yoga breathing*

Introduction

Studies of altered respiration in health and disease demonstrate that changes in respiration modify cerebral circulation.[1] Since experienced yoga practitioners effortlessly practice volitional yoga breathing for several minutes, these practices provide an opportunity to understand the physiological effects of voluntarily changing the breath. Volitionally regulated yoga breathing intentionally alters breath frequency among other characteristics of the breath.[2] A comparison of the effects of fast and slow yoga breathing using functional near infra-red spectroscopy showed that fast breathing was considered effective to improve cerebral brain oxygenation in the frontal areas studied, whereas slow breathing did not have that effect.[3]

Previously, changes in major cerebral vessels based on transcranial Doppler recordings showed that volitional yoga breathing practices changed cerebrovascular hemodynamics from transcranial Doppler monitoring of bilateral middle cerebral arterial (MCA) blood flow.[4-7]

In these studies, differences in study designs and durations of interventions made comparisons across studies difficult; hence, the effects of changes in breath frequency on blood flow in the main cerebral vessels could not be determined. Hence, the present study was designed to compare two yoga breathing practices with opposite effects on breath frequency to determine their impact on cerebrovascular dynamics. High-frequency yoga breathing (HFYB, called *kapalabhati pranayama*) involves volitionally increasing breath frequency, whereas the second practice, bumblebee yoga breathing (BBYB, called *brahmari pranayama*) involves slowing of the breath.

How to cite this article: Aacharya C, Telles S, Sharma SK. Cerebral hemodynamics and vagally mediated HRV associated with high- and low-frequency yoga breathing: An exploratory, randomized, crossover study. Int J Yoga 2024;17:29-36.

Submitted: 16-Oct-2023 **Revised:** 18-Dec-2023
Accepted: 19-Dec-2023 **Published:** 13-May-2024

Address for correspondence:
Dr. Shirley Telles, Patanjali Research Foundation, Patanjali Yogpeeth, Haridwar - 249 405, Uttarakhand, India.
E-mail: shirleytelles@gmail.com

Access this article online

Website: https://journals.lww.com/IJOY

DOI: 10.4103/ijoy.ijoy_197_23

Quick Response Code:

In order to understand possible mechanisms underlying possible differences in cerebrovascular hemodynamics between fast and slow breathing, the vagally mediated heart rate variability (VmHRV) and respiration were recorded simultaneously.

Materials and Methods

Participants

Thirty healthy students of both genders (male: female = 1:1) aged between 20 and 38 years (group mean age ± standard deviation [SD]; 27.3 ± 4.24 years) were recruited from a state university in India. The sample size was not determined *a priori*. The present study, with a sample size of 30, level of significance (α) =0.05, effect size = 0.76 (calculated using Cohen's formula for the change in peak systolic velocity (PSV) following HFYB), had power = 1.000 which was determined using G*power (version 3.1), University of Bonn, Bonn, Germany.[8] Recruitment was by oral announcements in the lecture halls with no incentives to the participants. The criteria for inclusion were: (i) normal health based on a semi-structured interview, (ii) 6 months of experience of yoga breathing practices, and (iii) age range between 20 and 45 years. The participants were to be excluded if they: (i) had recent surgery, (ii) were on medication, (iii) had tinnitus or active ear infection and epilepsy or history of stroke, (iv) have extra systole in electrocardiogram (EKG), (v) were consuming tobacco/alcohol regularly, and (vi) were not having adequate temporal acoustic window as it limits the quality of Transcranial Doppler (TCD) recordings.[9] None of the participants were excluded for any of the above-mentioned exclusion criteria. The study had prior approval from the Institution's Ethics Committee (approval no. PRF/YRD/022/010). The signed informed consent was obtained from each participant. The baseline characteristics of the participants are given in Table 1.

Table 1: The baseline characteristics of the participants

Characteristics (*n*=30)	Values
Age (years)	
Age group, mean±SD	27.3±4.24
Age range	20–38
Gender	
Male:female	1:1
Actual values	15:15
Education (years) (%)	
13–15	10.00
16–17	90.00
Experience of yoga (months) (%)	
6–12	3.00
13–60	30.33
60 and above	66.66
Time spent in yoga practice per week (min) (%)	
<150	10.00
150–300	67.66
300 and above	23.33

SD: Standard deviation

Study design

The study was an exploratory, randomized, crossover trial carried out between June and August 2022. Each participant was assessed for the changes in (i) bilateral cerebrovascular dynamics and (ii) VmHRV and respiration rate during the two yoga breathing sessions (i.e., BBYB and HFYB) carried out on 2 consecutive days, with assessment at the same time of day for each participant. The Nijmegen questionnaire was administered immediately after the yoga breathing sessions to detect if there were any symptoms related to hyperventilation arising from the breathing practices.

The two sessions were conducted in random order on 2 consecutive days at the same time of the day. Random assignment of the order of the two sessions was carried out using the lottery system: A person with no other involvement in the trial prepared thirty identical slips of paper. Of the thirty slips, 15 had the word "HFYB" while the other fifteen had the word "BBYB." The slips of paper were then identically folded and put in an envelope. Each participant drew one slip of paper from the envelope which determined their order of the sessions.[4] The participants who had the word "HFYB" were assigned the HFYB session on day 1 and the BBYB session on day 2 by the person who had no other involvement in the trial, whereas the participants who had the word "BBYB" were assigned the BBYB session on day 1 and the HFYB session on day 2. The words (i.e., "HFYB" or "BBYB" which were written on the slips) were not decoded to the participants. The order of assessments of a session is shown in Figure 1. The total duration of a session was 13.5 min. The assessments were conducted in a well ventilated, dimly lit sound attenuated room. There were two times for the assessments (i.e., forenoon and the other in the evening). However, the time of assessment for each participant was kept constant (e.g., the participants who received BBYB sessions in the forenoon also received the HFYB sessions in the forenoon) given the variation in heart rate variability (HRV) during the day.[10,11] Of the total participants, 14 were assessed in the morning while 16 were assessed in the evening.

The Schematic representation of the study design is given in Figure 1.

Assessments

Cerebral blood flow

The MCAs were monitored bilaterally using transcranial Doppler ultrasonography (Digi-LiteTm-Rimed Ltd, Israel). Two separate 2.0 MHz ultrasound probes (Rimed, SN 17-3681; Rimed, SN 17-3682) were placed over the transtemporal acoustic window just above the zygomatic arch and in front of the tragus of the ear to simultaneously record the insonation of both the MCA arteries.[5] These two probes were firmly fixed in a headframe to stop the

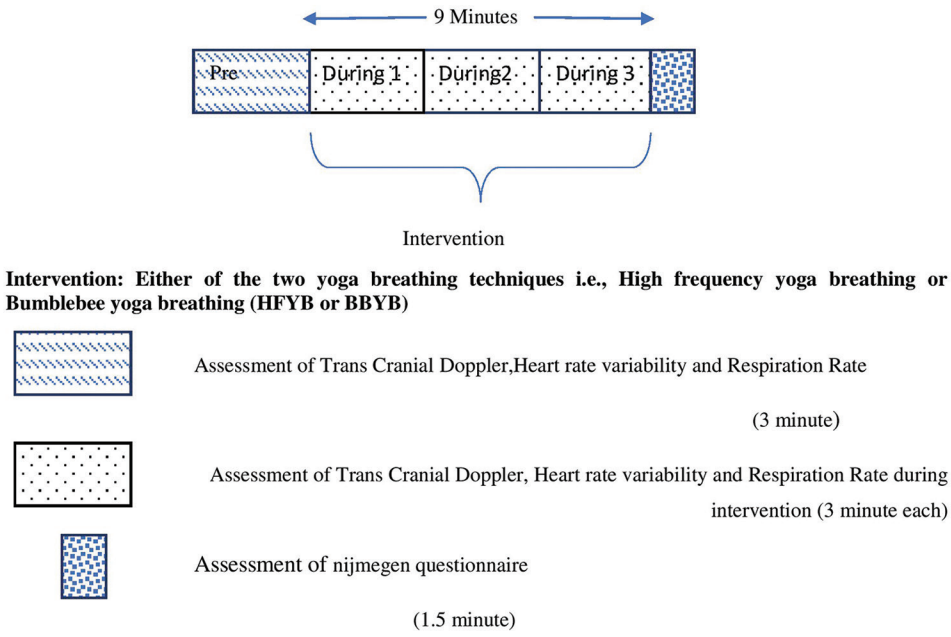

Figure 1: Schematic representation of the study design

motion of the probes and to manage a constant angle of the MCA insonation depth at 40–65 mm from the skull surface where the probes were positioned.[6] The TCD settings were as follows: Intensity spatial-peak temporal average: 420Mw/CM2 Sample volume: 15 mm in length and thermal index: 1.6.[6]

The variables recoded were: (i) PSV (i.e., the first peak on a TCD waveform for each cardiac cycle that shows cerebral blood flow in cm/s at systolic phase), (ii) end diastolic velocity (EDV) (i.e., the second peak on a TCD waveform for each cardiac cycle that shows cerebral blood flow in cm/s at diastolic phase), (iii) mean flow velocities (MFVs) (i.e., the value of EDV plus one-third of the difference of PSV and EDV), and (iv) pulsatility index (PI) (i.e., an indicator of resistance to intracranial blood flow).

Vagally mediated heart rate variability and respiratory characteristics

To reduce variability in the VmHRV recordings, in keeping with the guidelines for VmHRV assessments,[12] sessions were conducted at fixed times (i.e., between 10:30 a.m and 12:30 pm and 3:30 pm to 6:30 pm) and at least 2½ h after the last meal or after the consumption of tea or any caffeinated beverage, in a sound-attenuated and air-conditioned laboratory where participants were assessed while seated with back support.

Assessment of EKG and respiration rate were made using a MP 45 data acquisition system (Biopac student lab, Biopac system Inc, USA). The EKG data were recorded using a standard bipolar limb lead I configuration and Silver/Silver Chloride (Ag/AgCI) pregelled adhesive electrodes (TYCO Healthcare, Germany).[13] Respiration was recorded using a respiratory strain gauge transducer fixed at the abdominal region around 8 cm below the lower coastal margin when the participant sat down erect and steady.[14]

The Nijmegen questionnaire

The Nijmegen questionnaire was used to detect the sign of hyperventilation immediately following the two yoga breathing sessions.[15]

Intervention

For both the interventions (i.e., BBYB and HFYB), participants were instructed to sit in the cross-legged posture (*sukhasan*) comfortably with their spine erect and eyes closed, hands resting on their knees throughout the session.

Bumblebee yoga breath

Participants were asked to breathe slowly and to exhale with the humming sound of a bumblebee, while exhaling through both the nostrils. There were no specific instructions for inhalation. The BBYB practice was for 9 min.

High frequency yoga breathing

Participants were instructed to breathe rapidly performing forceful exhalation followed by passive inhalation through both nostrils. The frequency of the practice was 1.0 Hz (nearly 1 stroke per second) and the total duration of the practice was 9 min.

Data extraction

Cerebral blood flow

The variables of Trans Cranial Doppler, i.e., PSV, EDV, MFV, and PI were assessed and further exported into the Microsoft Excel (2010) file in numeric values.

Vagally mediated heart rate variability

The power in the VmHRV series in the low-frequency (LF) band (0.04–0.15 Hz) and High-frequency (HF) band (0.15–1.50 Hz) was evaluated by applying the Fast Fourier Transformation and was logarithmically transformed to normalize the distribution. The root mean square of successive differences in successive RR intervals (RMSSD),[16] HF VmHRV (high frequency-vagally mediated heart rate variability), LF VmHRV (low frequency-vagally mediated heart rate variability) and LnRMSSD (natural logarithm of root mean square of differences in successive RR intervals) were logarithmically transformed to standardize the data and have been written as LnHF (natural logarithm of high frequency), LnLF (natural logarithm of low frequency), LnRMSSD (natural logarithm of root mean square of successive differences in successive RR intervals).

Respiration

The respiration recording was in the form of a wave with peaks (corresponding to abdominal expansion due to inspiration) and troughs (corresponding to abdominal contraction during expiration. Vertical displacement of respiration sensor during abdominal movements based on inductive plethysmography was calculated for each breath. The respiration rate was calculated as the average number of breaths/minutes in different states, i.e., pre, during 1, during 2, and during 3, expressed as breaths per minute.

Data analysis

The data were analyzed using IBM SPSS (Version 24.0), New York, USA. (i) The data for (a) cerebral blood flow and (b) VmHRV and respiratory characteristics (i.e., were analyzed using the repeated-measures analysis of variance (RM-ANOVA) followed by Bonferroni adjusted *post hoc* tests. The RM-ANOVA had two within-subject factors: These were the two sessions (BBYB and HFYB) and four states (Pre, During 1, During 2, During 3). The statistical significance (α) was set at 0.05.

Results

Thirty participants (15 females) aged between 20 and 38 years (group average age ± SD; 27.3 ± 4.24 years) completed the study. None of the participant reported any sign of hyperventilation following the two breathing sessions based on the scores of the Nijmegen questionnaire (group average score ± SD; 5.1 ± 5.3 for BBYB and 3.8 ± 3.8 for HFYB).

Repeated measures analyses of variance

The ANOVA values for (i) States, (ii) Sessions and (iii) interaction between the Session and States for (a) variables related to cerebral blood flow, and (b) VmHRV (c) height of respiratory waveform and (d) respiration rate are given in Table 2. A significant interaction between the Sessions and States for any variable suggests the interdependence of the two.

Post hoc analysis

The comparisons of during (During 1, During 2, During 3) values were done with the respective pre values.

Cerebral blood flow

BBYB reduced ($P < 0.05$; in all cases) (i) PSV of the left MCA in (a) during 1 and (b) during 3 (ii) PSV of the right MCA in (a) during 1 and (b) during 3 (iii) MFV of the left MCA in (a) during 1 and (b) during 3 (iv) MFV of the right MCA in (a) during 1 and (b) during 3 (v) EDV of the left MCA in (a) during 1 and (b) during 3 (vi) EDV of the right MCA in (a) during 1 and (b) during 3.

BBYB increased ($P < 0.05$; in all cases) (i) PI of the left MCA in during 3 and (ii) PI of the right MCA in during 3.

HFYB reduced ($P < 0.05$; in all cases) (i) PSV of the left MCA in during 1 with (ii) PSV of the right MCA at during 1 (iii) MFV of the left MCA in (a) during 1 and (b) during 3 (iv) MFV of the right MCA in (a) during 1 and (b) during 3 (v) EDV of the left MCA in During 3 (vi) EDV of the right MCA in (a) during 2 and (b) during 3.

HFYB increased ($P < 0.05$; in all cases) (i) PI of the left MCA in (a) during 2 and (b) during 3 (ii) PI of the right MCA increased in (a) during 2 and (b) during 3.

The group mean ± SD values for the variables related to cerebral blood flow are given in Table 3.

Vagally mediated heart rate variability

BBYB increased ($P < 0.05$; in all cases) (i) LnLF in during 1 (ii) LnHF in (a) during 1 and (b) during 3.

HFYB reduced ($P < 0.05$; in all cases) (i) LnLF in (a) during 2 and (b) during 3 (ii) LnHF in (a) during 2 and (b) during 3.

Respiration

BBYB increased ($P < 0.05$; in all cases) height of the respiratory waveform in (a) during 1 and (b) during 3.

BBYB reduced ($P < 0.05$; in all cases) (i) respiration rate in (a) during 1 and (b) during 3.

HFYB increased ($P < 0.05$; in all cases) (i) respiration rate in (a) during 2 and (b) during 3.

The group mean ± SD values for VmHRV and respiration are given in Table 4.

Discussion

During both HFYB and BBYB, there were comparable changes in bilateral MCA velocities despite the differences in breath frequencies during the two practices (i.e., during HFYB average breath frequency was 54.2/min, whereas during

Table 2: Repeated measures analysis of variance

Measurements	Factors	*F*	df	Huynh-feldt ε	*P*
PSV left MCA	States	8.395	1.323, 38.379	0.441	0.003
	Sessions	103.934	1, 29	1	<0.001
	States × sessions	19.42	2.503, 72.593	0.834	<0.001
PSV right MCA	States	10.249	1.3, 37.686	0.433	0.001
	Sessions	131.671	1, 29	1	<0.001
	States × sessions	25.111	2.28, 66.131	0.76	<0.001
MFV left MCA	States	10.408	1.456, 42.21	0.485	0.001
	Sessions	68.02	1, 29	1	<0.001
	States × sessions	22.41	2.378, 68.964	0.793	<0.001
MFV right MCA	States	15.549	1.450, 42.054	0.483	<0.001
	Sessions	91.539	1, 29	1	<0.001
	States × sessions	27.404	2.308, 66.935	0.769	<0.001
PI left MCA	States	13.641	1.843, 53.453	0.614	<0.001
	Sessions	30.246	1, 29	1	<0.001
	States × sessions	10.753	2.011, 58.332	0.67	<0.001
PI right MCA	States	14.395	1.655, 47.995	0.552	<0.001
	Sessions	23.713	1, 29	1	<0.001
	States × sessions	14.296	2.374, 68.842	0.791	<0.001
EDV left MCA	States	11.436	1.552, 44.999	0.517	<0.001
	Sessions	59.108	1, 29	1	<0.001
	States × sessions	17.053	2.804, 40.437	0.695	<0.001
EDV right MCA	States	20.665	1.679, 48.679	0.56	<0.001
	Sessions	69.499	1, 29	1	<0.001
	States × sessions	23.113	2.305, 66.838	0.768	<0.001
LnRMSSD	States	5.046	1.720, 49.892	0.573	0.013
	Sessions	1.142	1, 29	1.000	0.294
	States × sessions	0.582	1.990, 57.711	0.663	0.561
LnLF	States	89.658	1.837, 53.287	0.612	<0.001
	Sessions	22.325	1, 29	1.000	<0.001
	States × sessions	32.207	2.852, 82.710	0.951	<0.001
LnHF	States	21.652	1.754, 50.857	0.585	<0.001
	Sessions	21.652	1, 29	1.000	0.260
	States × sessions	2.080	2.540, 73.672	0.847	0.120
Height of respiratory waveform	States	6.099	1.589, 46.073	0.530	0.008
	Sessions	30.352	1, 29	1.000	<0.001
	States × sessions	14.712	2.784, 80.749	0.928	<0.001
Respiration rate	States	910.051	1.225, 35.513	0.408	<0.001
	Sessions	225.05	1, 29	1	<0.001
	States × sessions	606.208	1.875, 54.381	0.625	<0.001

Level of significance (α) = 0.05. MCA: Middle cerebral artery, PSV: Peak systolic velocity, MFV: Mean flow velocity, EDV: End diastolic velocity, LnRMSSD: Natural logarithm of root mean square of successive differences, LnLF: Natural logarithm of low frequency, LnHF:Natural logarithm of high frequency

BBYB average breath frequency was 3.8/min). These changes included significant decreases in bilateral MCA blood flow velocities (i.e., systolic, end-diastolic and MFV) along with an increase in the PI, indicative of resistance to flow.[17]

HF-VmHRV and RMSSD were used to determine VmHRV for the following reasons: When breath frequency is between 9 (0.15 Hz) and 24 (0.40 Hz) bpm, cardiac vagal activity is reflected in the HF band of the VmHRV.[12] The RMSSD being relatively free from respiratory influences indexes cardiac vagal activity at different breathing frequencies.[12]

There was an increase in vagally mediated HRV during BBYB based on increased LnHF and LnLF (reflecting VmHRV for slow breath frequency of average 3.8/min,[7] with a decrease in LnHF during HFYB. These changes during low and HFYB are comparable to the earlier reports of changes in HF-VmHRV during the yoga breathing practices,[13,18,19] with some differences based on analysis and interpretation of LF-VmHRV and HF-VmHRV.[20,21]

The changes in HF-VmHRV during the two breathing practices support the comparison of the cerebral blood flow

Table 3: Bilateral middle cerebral arterial blood flow velocities of Trans Cranial Doppler

	BBYB				HFYB			
	PRE	D1	D2	D3	PRE	D1	D2	D3
Left MCA								
PSV	78.22±14.18	72.08±12.89*	70.03±12.52	68.91±13.61*	79.58±15.21	71.55±14.87*	68.61±14.92	66.65±15.85
MFV	47.58±8.17	43.75±7.46*	42.44±7.42	42.04±7.6*	48.29±9.27	40.94±10.74*	39.76±10.88	38.61±11.19*
PI	0.95±0.18	0.97±0.19	0.93±0.19	0.96±0.17*	0.97±0.19	1.15±0.35	1.09±0.29*	1.09±0.26*
EDV	32.61±6.53	29.74±6.03*	29.43±5.84	28.69±5.6*	32.89±7.19	26.15±9.65	26.07±9.09	25±9.29*
Right MCA								
PSV	78.49±12.63	71.62±12.37*	69.39±12.74	68.16±12.77*	77.54±11.23	69.32±11.55*	65.93±11.82	64.63±12.57
MFV	48.29±9.01	43.95±8.24*	43.09±8.31	42.15±8.46*	47.91±7.57	40.41±8.6*	38.99±8.43	38.2±9.17*
PI	0.94±0.15	0.96±0.18	0.93±0.16	0.94±0.16*	0.95±0.18	1.12±0.28	1.08±0.26*	1.08±0.25*
EDV	33.41±7.7	30.16±7.06*	30.01±6.79	29.16±7*	32.98±6.74	25.93±8.49	25.68±7.84*	24.91±8.36*

*P<0.05; Repeated measures analysis of variance (RMANOVA), with bonferroni adjusted post-hoc analyses comparing pre and during 1, during 2, during 3. MCA=middle cerebral artery, PSV=Peak systolic velocity, MFV=Mean flow velocity, EDV=End diastolic velocity, PI=pulsatility index, D1=During1, D2=During2, D3=During3, BBYB: Bumblebee yoga breath, HFYB: High frequency yoga breathing

Table 4: Changes in vagally mediated heart rate variability and Respiration. Values are in mean±standard deviation

Variables	BBYB				HFYB			
	PRE	D1	D2	D3	PRE	D1	D2	D3
LnRMSSD	4.01±0.43	4.13±0.66*	4.34±0.56	4.33±0.81*	3.93±0.4	3.76±0.89	3.55±0.76	3.32±0.93*
LnLF	6.77±0.84	8.92±0.97*	9.04±1.02	9.09±1.12	6.52±0.67	6.51±1.28	6.24±1.24*	6.17±1.26*
LnHF	6.89±0.88	7.29±1.18*	7.66±1.24	7.45±1.22*	6.81±0.79	6.3±1.94	5.97±1.5*	5.53±2.05*
Height of respiratory waveform	0.45±0.31	3.83±3.87*	3.76±3.74	4.09±4.09*	0.69±0.52	4.19±4.44	4.13±4.05	4.06±4.49
Respiration rate	18.39±2.84	3.82±1.02*	3.75±0.77	3.93±0.87*	18.25±2.58	53.23±7.48	54.58±8.57*	54.89±9.49*

*P<0.05; Values are in mean±sd. RMANOVA, with Bonferroni adjusted post hoc analyses comparing pre and D1, D2, D3. BBYB: Bumblee bee yoga breathing, RMANOVA: Repeated measures analysis of variance, HFYB: High frequency yoga breathing, D1: During1, D2: During2, D3: During3, SD: Standard deviation, LnRMSSD: Natural logarithm of root mean square of successive differences in successive RR intervals, LnLF: Natural logarithm of low frequency, LnHF: Natural logarithm of high frequency

during the two yoga breathing practices on which the study was based. It was speculated that slow frequency yoga breathing would mimic vagal nerve stimulation effects on cerebral blood flow, leading to an increase in cerebral blood flow,[22] whereas HFYB would have opposite effects on vagally mediated functions and hence on cerebral blood flow. However, while the two breathing practices had opposite effects on VmHRV, both breathing practices decreased Cerebral blood flow (CBF) velocities while increasing the PI. Hence, these changes appear unrelated to vagal mediated effects but appear related to other physiological changes common to both breathing practices.

Previously, altered breath frequencies were reported to cause hyperventilation-related reduced cerebral blood flow.[23] HFYB or BBYB appears unlikely to lead to hyperventilation in the present study since participants' scores in the Nijmegen questionnaire intended to detect hyperventilation, for both HFYB and BBYB sessions were well below the cut-off for symptoms of hyperventilation (i.e., 3.77 for HFYB; 5.07 for BBYB, versus a cut-off value of >19 for hyperventilation in the Nijmegen questionnaire).[24] However in both BBYB and HFYB hypocapnia cannot be ruled out. During both breathing practices there appeared to be an increase in the depth of respiration based on increases in the height of the wave, indicating displacement of the strain gauge transducer with abdominal respiratory movements. While HF deep breathing reduces arterial carbon dioxide,[25] slow breathing with an increase in depth of respiration may also decrease arterial carbon dioxide levels.[26] Increases in the height of the respiration waveform during both HFYB and BBYB reflects deeper respiration which may have led to hypocapnia (without symptoms) and hence have influenced the cerebrovascular hemodynamics, leading to reduced middle cerebral artery velocities, known to occur in voluntary hyperventilation.[27] Simultaneous monitoring of arterial or end-tidal carbon dioxide would have shown whether this did occur.

An earlier study reported increased MCA velocity during breath holding while the carbon dioxide levels were not assessed during the breath holding.[28] In the present study, during BBYB, the MCA velocity decreased. Although carbon dioxide levels were not measured, they may be expected to be greater than during HFYB, since BBYB is much slower in frequency, alhough different from the baseline. However, as compared to breath holding, during breath holding carbon dioxide levels (which were not measured in the study by Settakis *et al*.) may be expected

to increase hence creating hypercapnia, which is known to increase CBF[29] and hence increase MCA velocity. This would not be the case with BBYB.

Another possible explanation for the present decreased MCA velocities is related to flow-activation coupling, an autoregulatory mechanism of cerebral blood flow seen in normal health.[30] With transcranial Doppler sonography, changes in brain activity in areas supplied by the MCA can be measured indirectly with high temporal resolution.[31] Hence, under nonpathological conditions, periods of increased neural activity associated with focused activity,[32] or with rapid eye movement sleep,[33] increase MCA blood flow velocity, whereas periods of low neural activity (such as slow wave sleep) lead to reduced MCA flow velocities.[34] Hence, another possible explanation for the effects of HFYB and BBYB on cerebrovascular hemodynamics could be related to the effects on neural activity. The effects of HFYB on neural activity are complex. Previously, during HFYB participants in normal health had increased slow frequencies in the electroencephalogram (EEG).[23,34] Furthermore, supporting a quiet mental state during HFYB were previous results of a decrease in state anxiety which was reported by participants following HFYB.[35] During BBYB also, previously an increase in theta activity in the EEG during the practice was reported, with the authors attributing this to "*blissful mental quiescence*," described as a state in which thoughts are absent but consciousness remains.[36] Hence, in both HFYB and BBYB, the decrease in MCA blood flow velocities could be due to a decrease in neural activity associated with a quiet mental state, which is the target state aimed to be attained through yoga breathing.[37] In both HFYB and BBYB, the cerebral arterial blood flow velocities and PI during the practices were within the normal range.[38]

Conclusion

In summary during both HFYB and BBYB, the TCD recordings showed decreased MCA flow velocities and increased resistance to cerebral blood flow. VmHRV changes suggested decreased VmHRV during HFYB, whereas VmHRV increased during BBYB. There were no symptoms of hyperventilation with either practice though lower carbon dioxide levels cannot be ruled out. The findings support reduced global neural activity during volitional yoga breathing irrespective of breath frequency, though other changes suggest this may be a "restful alertness." The findings are limited by the absence of a third control session since it was difficult to get the volunteers to participate in a third session. However, without a control session, it is not possible to make comparisons between volitional breathing and regular breathing. Furthermore, additional variables, such as carbon dioxide levels or functional TCD changes would help to understand neural activity and cerebral blood flow changes associated with the breathing practices.

The study is limited by the fact that measures of carbon dioxide levels were not included. Future research should include direct measurements of carbon dioxide levels during the assessment of cerebrovascular hemodynamics. This would be useful to better understand the mechanisms related to the effects of yoga breathing techniques on cerebrovascular hemodynamics. Apart from this, other sources of variation (e.g. phase of the menstrual cycle of female participants) could have contributed to the results. Also, the study was not registered with the Clinical Trials Registry-India, which limits the availability of trial materials, data, and findings where needed.

Ethical statement

The study was approved by the Institutional Ethics Committee of the Patanjali Research Foundation (approval no. PRF/YRD/022/010).

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Ogoh S, Suzuki K, Washio T, Tamiya K, Saito S, Bailey TG, *et al*. Does respiratory drive modify the cerebral vascular response to changes in end-tidal carbon dioxide? Exp Physiol 2019;104:1363-70.
2. Sharma SK, Kala N, Telles S. Volitional yoga breathing influences attention and anxiety: An exploratory randomized crossover study. Complement Med Res 2022;29:120-6.
3. Bellissimo G, Leslie E, Maestas V, Zuhl M. The effects of fast and slow yoga breathing on cerebral and central hemodynamics. Int J Yoga 2020;13:207-12.
4. Nivethitha L, Mooventhan A, Manjunath NK, Bathala L, Sharma VK. Cerebrovascular hemodynamics during pranayama techniques. J Neurosci Rural Pract 2017;8:60-3.
5. Nivethitha L, Mooventhan A, Manjunath NK, Bathala L, Sharma VK. Cerebrovascular hemodynamics during the practice of Bhramari Pranayama, Kapalbhati and Bahir-Kumbhaka: An exploratory study. Appl Psychophysiol Biofeedback 2018;43:87-92.
6. Kumar A, Kala N, Telles S. Cerebrovascular dynamics associated with yoga breathing and breath awareness. Int J Yoga 2022;15:19-24.
7. Laborde S, Allen MS, Borges U, Dosseville F, Hosang TJ, Iskra M, *et al*. Effects of voluntary slow breathing on heart rate and heart rate variability: A systematic review and a meta-analysis. Neurosci Biobehav Rev 2022;138:104711.
8. Erdfelder E, Faul F, Buchner A. GPOWER: A general power analysis program. Behav Res Methods Instrum Comput 1996;28:1.
9. Marinoni M, Ginanneschi A, Forleo P, Amaducci L. Technical limits in transcranial Doppler recording: Inadequate acoustic windows. Ultrasound Med Biol 1997;23:1275-7.
10. Vitale JA, Bonato M, La Torre A, Banfi G. Heart rate variability

in sport performance: Do time of day and chronotype play a role? J Clin Med 2019;8:723.
11. Brockmann L, Hunt KJ. Heart rate variability changes with respect to time and exercise intensity during heart-rate-controlled steady-state treadmill running. Sci Rep 2023;13:8515.
12. Laborde S, Mosley E, Thayer JF. Heart rate variability and cardiac vagal tone in psychophysiological research – Recommendations for experiment planning, data analysis, and data reporting. Front Psychol 2017;8:213.
13. Telles S, Singh N, Balkrishna A. Heart rate variability changes during high frequency yoga breathing and breath awareness. Biopsychosoc Med 2011;5:4.
14. Telles S, Sharma SK, Gupta RK, Bhardwaj AK, Balkrishna A. Heart rate variability in chronic low back pain patients randomized to yoga or standard care. BMC Complement Altern Med 2016;16:279.
15. Courtney R, van Dixhoorn J. Questionnaires and manual methods for assessing breathing dysfunction. In: Recognizing and Treating Breathing Disorders: A Multidisciplinary Approach. London: Churchill Livingstone (Elsevier); 2014. p. 137-46.
16. Shaffer F, Ginsberg JP. An overview of heart rate variability metrics and norms. Front Public Health 2017;5:258.
17. Ghorbani A, Ahmadi MJ, Shemshaki H. The value of transcranial Doppler derived pulsatility index for diagnosing cerebral small-vessel disease. Adv Biomed Res 2015;4:54.
18. Komori T. The relaxation effect of prolonged expiratory breathing. Ment Illn 2018;10:7669.
19. Kuppusamy M, Kamaldeen D, Pitani R, Amaldas J, Ramasamy P, Shanmugam P, *et al*. Effects of yoga breathing practice on heart rate variability in healthy adolescents: A randomized controlled trial. Integr Med Res 2020;9:28-32.
20. Heathers JA. Everything Hertz: Methodological issues in short-term frequency-domain HRV. Front Physiol 2014;5:177.
21. Laborde S, Mosley E, Bellenger C, Thayer J. Editorial: Horizon 2030: Innovative applications of heart rate variability. Front Neurosci 2022;16:937086.
22. Conway CR, Sheline YI, Chibnall JT, George MS, Fletcher JW, Mintun MA. Cerebral blood flow changes during vagus nerve stimulation for depression. Psychiatry Res 2006;146:179-84.
23. Sinha M, Sinha R, Ghate J, Sarnik G. Impact of altered breathing patterns on interaction of EEG and heart rate variability. Ann Neurosci 2020;27:67-74.
24. van Dixhoorn J, Duivenvoorden HJ. Efficacy of Nijmegen questionnaire in recognition of the hyperventilation syndrome. J Psychosom Res 1985;29:199-206.
25. Sehati S, Young JD, Sykes MK, McLeod CN. Monitoring of end-tidal carbon dioxide partial pressure during high frequency jet ventilation. Br J Anaesth 1989;63:47S-52S.
26. Anderson DE, McNeely JD, Windham BG. Device-guided slow-breathing effects on end-tidal CO(2) and heart-rate variability. Psychol Health Med 2009;14:667-79.
27. Duarte J, Markus H, Harrison MJ. Changes in cerebral blood flow as monitored by transcranial Doppler during voluntary hyperventilation and their effect on the electroencephalogram. J Neuroimaging 1995;5:209-11.
28. Settakis G, Lengyel A, Molnár C, Bereczki D, Csiba L, Fülesdi B. Transcranial Doppler study of the cerebral hemodynamic changes during breath-holding and hyperventilation tests. J Neuroimaging 2002;12:252-8.
29. Lucas RA, Pearson J, Schlader ZJ, Crandall CG. Hypercapnia-induced increases in cerebral blood flow do not improve lower body negative pressure tolerance during hyperthermia. Am J Physiol Regul Integr Comp Physiol 2013;305:R604-9.
30. Hartje W, Ringelstein EB, Kistinger B, Fabianek D, Willmes K. Transcranial Doppler ultrasonic assessment of middle cerebral artery blood flow velocity changes during verbal and visuospatial cognitive tasks. Neuropsychologia 1994;32:1443-52.
31. Droste DW, Harders AG, Rastogi E. A transcranial Doppler study of blood flow velocity in the middle cerebral arteries performed at rest and during mental activities. Stroke 1989;20:1005-11.
32. Silvestrini M, Cupini LM, Matteis M, Troisi E, Caltagirone C. Bilateral simultaneous assessment of cerebral flow velocity during mental activity. J Cereb Blood Flow Metab 1994;14:643-8.
33. Droste DW, Berger W, Schuler E, Krauss JK. Middle cerebral artery blood flow velocity in healthy persons during wakefulness and sleep: A transcranial Doppler study. Sleep 1993;16:603-9.
34. Stancák A Jr., Kuna M, Srinivasan, Vishnudevananda S, Dostálek C. Kapalabhati – Yogic cleansing exercise. I. Cardiovascular and respiratory changes. Homeost Health Dis 1991;33:126-34.
35. Telles S, Gupta RK, Gandharva K, Vishwakarma B, Kala N, Balkrishna A. Immediate effect of a yoga breathing practice on attention and anxiety in pre-teen children. Children (Basel) 2019;6:84.
36. Vialatte FB, Bakardjian H, Prasad R, Cichocki A. EEG paroxysmal gamma waves during Bhramari Pranayama: A yoga breathing technique. Conscious Cogn 2009;18:977-88.
37. Kala N, Telles S, Sharma SK, Balkrishna A. P300 following four voluntarily regulated yoga breathing practices and breath awareness. Clin EEG Neurosci 2023;54:117-29.
38. Moppett IK, Mahajan RP. Transcranial Doppler ultrasonography in anaesthesia and intensive care. Br J Anaesth 2004;93:710-24.