Original Article

Cerebrovascular Dynamics Associated with Yoga Breathing and Breath Awareness

Ankur Kumar, Niranjan Kala, Shirley Telles

Division of Yoga and Clinical Neurophysiology, Patanjali Research Foundation, Patanjali Yogpeeth, Haridwar, Uttarakhand, India

Abstract

Aims: Breath frequency can alter cerebral blood flow. The study aimed to determine bilateral middle cerebral arterial hemodynamics in high-frequency yoga breathing (HFYB) and slow frequency alternate nostril yoga breathing (ANYB) using transcranial Doppler sonography. **Methods:** Healthy male volunteers were assessed in two separate trials before, during, and after HFYB (2.0 Hz for 1 min, n = 16) and ANYB (12 breaths per minute for 5 min, n = 22). HFYB and ANYB were separately compared to breath awareness (BAW) and to control sessions. **Statistical Analysis:** The data were analyzed using repeated-measures ANOVA with Bonferroni adjusted *post hoc* tests. **Results:** During HFYB there was a decrease in end-diastolic velocity (EDV) and mean flow velocity (MFV) ($P < 0.01$ for left and $P < 0.05$ for right middle cerebral arteries; MCA) with an increase in pulsatility index (PI) for the right MCA ($P < 0.05$). During ANYB, there was a bilateral decrease in peak systolic velocity ($P < 0.05$ for left and $P < 0.01$ for right MCA), EDV ($P < 0.01$) and MFV ($P < 0.01$ for left and $P < 0.001$ for right MCA) and an increase in PI ($P < 0.01$). During BAW of the two sessions there was a decrease in lateralized flow and end-diastolic velocities ($P < 0.05$) and an increase in PI ($P < 0.05$). **Conclusions:** Changes in peak flow velocities and pulsatility indices during and after HFYB, ANYB, and BAW suggest decreased cerebrovascular blood flow and increased flow resistance based on different mechanisms.

Keywords: *Breath awareness, cerebrovascular hemodynamics, transcranial Doppler sonography, yoga breathing*

Introduction

Changes in cerebral blood flow (CBF) are recorded through functional near-infrared spectroscopy (FNIRs) and transcranial Doppler sonography (TCD).[1,2] A transcranial Doppler sonography recording showed a decrease in right middle cerebral artery diastolic blood flow velocity after high-frequency yoga breathing (HFYB) (2.0 Hz practiced for 1 min; *kapalabhati pranayama*) and hyperventilation, whereas after hyperventilation alone there was a decrease in left systolic blood flow velocity.[3] The results were attributed to carbon dioxide washout and hypocapnia following high-frequency yoga breathing and hyperventilation.

Yoga-regulated breathing can also be practiced at a slower than resting respiratory frequency. *Anulom-vilom pranayama* (alternate nostril yoga breathing [ANYB]) is a practice which involves breathing through the left and right nostrils alternately with a decrease in respiratory rate.[4] Slow breathing with a decrease in breath depth is associated with hypoventilation resulting in hypercapnia.[5] Hence in ANYB, the arterial carbon dioxide levels may be expected to increase depending on breath frequency, depth, and duration of breathing.[6]

Hence, the present study was planned to determine the effects of (i) HFYB and (ii) slow frequency ANYB on bilateral middle cerebral artery blood flow in volunteers with normal health. Both practices were compared to breath awareness (BAW) as a control since BAW is a part of all yoga breathing practices,[7] but does not involve changing the breath pattern.

Methods

Participants

Trial-1 assessed HFYB (2.0 Hz) and in the second trial (Trial-2) ANYB (<10

How to cite this article: Kumar A, Kala N, Telles S. Cerebrovascular dynamics associated with yoga breathing and breath awareness. Int J Yoga 2022;15:19-24.

Submitted: 29-Nov-2021 **Revised:** 17-Feb-2022
Accepted: 18-Feb-2022 **Published:** 21-Mar-2022

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: www.ijoy.org.in

DOI: 10.4103/ijoy.ijoy_179_21

Quick Response Code:

breaths/min) was assessed. In Trial-1 sixteen healthy male volunteers (group mean age ± Standard deviation [SD], 23.7 ± 4.5 years) and in Trial-2 twenty healthy male volunteers (group mean age ± SD, 22.9 ± 5.4 years) were recruited. Two trials differed by three factors: (i) the yoga breathing, (ii) duration of yoga breathing, i.e., 1 min in Trial-1 and 5 min in Trial-2; these durations were determined based on comparable durations used in previously published research[8,9] and (iii) number of participants (i.e., 16 in Trial-1 and 22 in Trial-2). Other methodological details were the same for both trials. The participants were recruited based on: (i) normal health based on a semi-structured examination, (ii) a minimum of 3 months experience of yoga breathing practices (iii) right-hand dominance based on the Edinburgh handedness inventory[10] and (iv) willingness to take part in the study. Hand dominance was recorded to determine which hand would be used to regulate nostril airflow during ANYB, as this motor activity could influence cerebral blood flow contralaterally. The exclusion criteria were: (i) consumption of alcohol and tobacco, (ii) taking any conventional or nonconventional medication, (iii) epilepsy for Trial-1, and (iv) nasal septal deviation for Trial-2.

Institutional ethical committee approval (YRD-018/016-17) was obtained. Participants' signed informed consent was obtained.

Study design

Participants were assessed in three separate sessions in random order on three consecutive days. For Trial-1 the three sessions were: (i) HFYB, (ii) BAW, and (iii) quiet sitting (QS). For Trial-2 the sessions were: (i) ANYB, (ii) BAW, and (iii) QS. There were baseline assessments for 5 min, followed by yoga breathing (in Trial-1 HFYB for 1 min; in Trial-2 ANYB for 5 min)[8,9] and postassessment for 5 min. Figure 1 shows a schematic representation of the study design.

Assessment of cerebral blood flow

Transcranial Doppler sonography was used to monitor the middle cerebral arteries (MCA) bilaterally (Digi-Lite™-Rimed Ltd, Israel). Separate 2.0 Mega Hz ultrasound probes (RIMED, SN 17-3681) were placed at the transtemporal acoustic window to simultaneously record the insonation of both MCA. A headframe was used to reduce motion and to secure a constant angle of the middle cerebral artery insonation depth at 40–65 mm from the skull surface. The sample volume was adjusted to get an acoustically optimal and visually stable ultrasound signal.

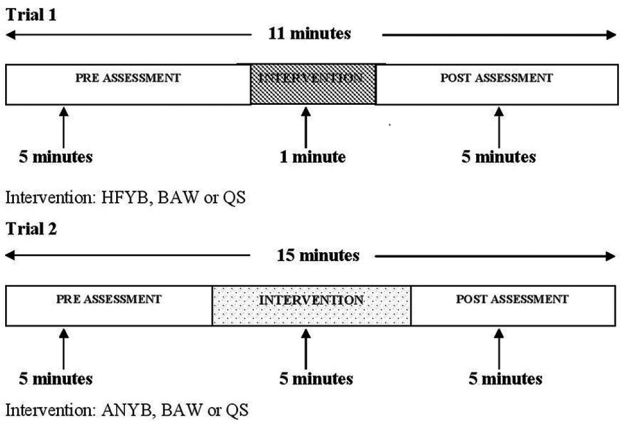

Figure 1: Schematic representation of the study design

The TCD settings were as follows: intensity spatial-peak temporal-average: 420 mW/cm^2; sample volume: 15 mm in length and thermal index: 1.6. The average peak systolic velocity (PSV) in cm/s, end-diastolic velocity (EDV) in cm/s, mean flow velocity (MFV) in cm/s, and pulsatility index (PI) were recorded in pre, during, and post states of each session.

Qualitative self-assessment of practice

Visual analog scales were used for volunteers to self-rate their quality of practice. These were 10 cm horizontal lines to rate the quality of practice from 0 (the worst possible) to 10 (excellent) at the end of each session. A minimum reading of 7.0 cm was considered to indicate a satisfactory practice. No sessions needed to be excluded for a reading <7.0.

Interventions

During each session, participants were seated comfortably on a chair with their hands resting on their knees and eyes closed. Since the participants were yoga practitioners, no training of breathing practices was given to them before data recording. However, the breath rate for the two yoga breathing practices was observed before the recordings. Participants were observed during a single trial before recording and their breath rate was counted by visual inspection of thoracoabdominal movements, approximately 11 cpm (range 9–12 cpm) for ANYB and 120 cpm (range 110–124 cpm) for HFYB. Furthermore, at the end of a session participants were asked if they were able to maintain their breath rate constant during the practice, and indicate this on a 10 cm, visual analog scale.

High-frequency yoga breathing

HFYB included voluntary rapid breathing with forceful contraction of the anterior abdominal wall during expiration. The respiratory rate was 2.0 Hz and the total duration of practice was 1 min.

Alternate nostril yoga breathing

Participants were instructed to inhale through the left nostril and exhale through the right nostril, followed by inhalation through the right nostril and exhalation through the left nostril. Nostril patency was regulated with gentle pressure from either the ring finger (to occlude the left nostril) or the thumb (to occlude the right nostril) of the

right hand, while the index and middle finger were kept flexed against the palm of the right hand. The right hand was noted as the dominant hand for all participants.[10] When breathing through the left or right nostril, pressure of the ring finger (for the left nostril) or the thumb (for the right nostril) of the right hand were released keeping the finger or thumb close to the nose but not touching it. This sequence was repeated during the 5 min practice. Participants were instructed to be aware of the passage of air through the nasal passage as well as other sensations during the practice.

Breath awareness

Throughout the practice, participants were asked to consciously maintain their awareness on the movement of air in the nasal passage and other sensations related to respiration. They were instructed not to alter the respiration in any way and only to pay attention to the breathing process. BAW was selected as an intervention because being aware of the breath is an inherent part of all yoga breathing techniques.

Quiet sitting

Participants were instructed to allow their thoughts to wander freely without any attempt to direct their thoughts. They were told not to alter their breathing in any way and let breathing be spontaneous. Quiet sitting was different from yoga breathing as it did not involve voluntary changes in respiration and it was different from BAW as it did not involve conscious awareness on breathing.

Data analysis

The data were analyzed using repeated-measures analysis of variance (RM-ANOVA) followed by Bonferroni adjusted *post hoc* tests using SPSS (version 24.0, IBM SPSS, New York, USA). The RM-ANOVA had two within-subjects factors: States, with three levels, i.e., pre, during, and post; and Sessions, with three levels, i.e., yoga breathing (HFYB in Trial-1 and ANYB in Trial-2), BAW and quiet sitting.

Results

The group mean values ± SD for PSV, EDV, MFV, and PI are given in Table 1.

Repeated measures analyses of variance

Trial-1

In Trial-1, MFV differed significantly between states for the left and the right MCA ($F = 8.81$; df = 1.88, 28.15; Huynh-Feldt $\varepsilon = 0.94$; $P < 0.001$). A significant difference between states was noted for the left MCA ($F = 11.92$; df = 1.58, 23.72; Huynh-Feldt $\varepsilon = 0.79$; $P < 0.001$) and the right MCA ($F = 12.71$; df = 1.58, 23.76; Huynh-Feldt $\varepsilon = 0.79$; $P < 0.001$) in EDV as well as for the right MCA in PI. A significant interaction between sessions x states was found in MFV ($F = 8.09$; df = 1.55, 23.30; Huynh-Feldt $\varepsilon = 0.39$; $P < 0.004$) and EDV for the left MCA ($F = 11.13$; df = 1.57, 23.56; Huynh-Feldt $\varepsilon = 0.39$; $P < 0.001$) while for the right MCA the EDV ($F = 5.25$; df = 1.91, 28.67; Huynh-Feldt $\varepsilon = 0.48$; $P = 0.012$) and PI ($F = 6.16$; df = 2.05, 30.71; Huynh-Feldt $\varepsilon = 0.51$; $P = 0.005$) showed a significant interaction between sessions x states suggesting interdependence between factors.

Trial-2

In Trial-2, for the left MCA as well as for the right MCA, there was a significant difference in states in PSV ($F = 0.814$; df = 2.00. 42.00; Huynh-Feldt $\varepsilon = 1.00$; $P < 0.001$ in left MCA); ($F = 8.96$; df = 2.00. 42.00; Huynh-Feldt $\varepsilon = 1.00$; $P < 0.001$ in right MCA), EDV ($F = 15.62$; df = 1.61, 33.71; Huynh-Feldt $\varepsilon = 0.80$; $P < 0.001$ in left MCA); ($F = 19.39$; df = 1.40, 29.47; Huynh-Feldt $\varepsilon = 0.70$; $P < 0.001$ in right MCA), MFV ($F = 13.31$; df = 1.85, 38.75; Huynh-Feldt $\varepsilon = 0.92$; $P < 0.001$ in left MCA); ($F = 15.97$; df = 1.63, 34.28; Huynh-Feldt $\varepsilon = 0.82$; $P < 0.001$ in right MCA), and PI ($F = 16.07$; df = 1.64, 34.44; Huynh-Feldt $\varepsilon = 0.82$; $P < 0.001$ in left MCA); ($F = 14.57$; df = 1.24, 26.02; Huynh-Feldt $\varepsilon = 0.62$; $P < 0.001$ in right MCA)., Also there was a significant interaction between sessions x states suggesting interdependence of these factors in PSV ($F = 4.11$; df = 3.42, 71.75; Huynh-Feldt $\varepsilon = 0.85$; $P = 0.007$ in left MCA); ($F = 4.33$; df = 3.69, 77.56; Huynh-Feldt $\varepsilon = 0.92$; $P = 0.004$ in right MCA), EDV ($F = 4.67$; df = 2.04, 42.91; Huynh-Feldt $\varepsilon = 0.51$; $P = 0.014$ in left MCA); ($F = 6.04$; df = 2.63, 55.22; Huynh-Feldt $\varepsilon = 0.66$; $P = 0.002$ in right MCA), MFV ($F = 4.64$; df = 2.40, 50.33; Huynh-Feldt $\varepsilon = 0.60$; $P < 0.01$ in left MCA); ($F = 5.62$; df = 3.29, 69.18; Huynh-Feldt $\varepsilon = 0.82$; $P < 0.001$ in right MCA), and PI ($F = 5.40$; df = 2.07, 43.46; Huynh-Feldt $\varepsilon = 0.52$; $P = 0.007$ in left MCA); ($F = 5.60$; df = 2.05, 43.01; Huynh-Feldt $\varepsilon = 0.51$; $P = 0.007$ in right MCA), for both left and right MCA. In addition, there was a significant difference between sessions in PSV, EDV, and MFV for the right MCA only ($F = 3.58$; df = 2.00,42.00; Huynh-Feldt $\varepsilon = 1.00$; $P = 0.037$ in PSV), ($F = 2.73$; df = 2.00,42.00; Huynh-Feldt $\varepsilon = 1.00$; $P = 0.077$ in EDV), and ($F = 3.36$; df = 2.00,42.00; Huynh-Feldt $\varepsilon = 1.00$; $P = 0.04$ in MFV of right MCA

Post hoc analyses

Trial-1

MFV reduced significantly during the practice of HFYB for the left ($P = 0.001$; Cohen's d = 0.55; 95% confidence interval [CI] = 1.70, 13.25) and for the right MCA ($P = 0.04$; Cohen's d = 0.50; 95% CI = 0.19, 10.05). EDV also decreased during the practice for the left ($P = 0.003$; Cohen's d = 0.82; 95% CI = 2.800, 14.10) and for the right MCA ($P = 0.016$; Cohen's d = 0.75; 95% CI=-1.03, 10.79). PI increased during HFYB for the right MCA ($P = 0.018$; Cohen's d = 0.83; 95%

Table 1: Bilateral middle cerebral arterial flow velocities and pulsatility index

Session	State	Left MCA				Right MCA			
		Peak velocity	Diastolic velocity	Mean velocity	Pulsatility index	Peak velocity	Diastolic velocity	Mean velocity	Pulsatility index
					Trial-1				
HFYB	Pre	68.23±20.20	29.13±9.32	42.17±12.39	0.94±0.24	63.71±16.21	25.25±7.43	38.07±9.88	1.02±0.18
	During	64.23±18.71	21.61±9.00†	35.82±10.84†	1.26±0.49	60.17±18.80	19.34±8.29*	32.95±10.39*	1.29±0.42*
	Post	67.34±19.98	28.20±9.99	41.25±13.01	0.98±0.20	62.20±14.10	24.86±7.39	37.31±9.16	1.03±0.18
BAW	Pre	67.69±14.20	28.63±8.59	41.65±9.77	0.96±0.21	65.21±18.54	26.64±7.97	39.50±10.95	0.98±0.19
	During	67.32±13.76	26.68±8.49†	40.23±9.49*	1.04±0.24	64.65±16.99	24.59±7.43†	37.94±9.80	1.07±0.22*
	Post	67.65±15.17	28.48±9.10	41.53±10.59	0.97±0.20	65.35±17.51	26.45±7.80	39.42±10.49	1.00±0.20
QS	Pre	67.70±16.23	27.85±6.94	41.13±9.65	0.98±0.16	63.05±17.94	25.70±9.10	38.15±11.78	1.00±0.16
	During	67.84±16.37	27.62±7.01	41.02±9.71	0.99±0.17	61.40±20.48	24.97±10.36	37.11±13.46	1.03±0.21
	Post	67.50±16.08	27.51±6.88	40.84±9.58	0.99±0.16	60.93±21.96	24.73±10.55	36.80±14.08	1.06±0.30
					Trial-2				
ANYB	Pre	66.88±11.95	27.85±5.01	40.86±6.72	0.95±0.15	65.27±14.22	27±6.68	39.75±8.97	0.97±0.12
	During	62.21±14.85*	23.66±7.07†	36.51±9.47†	1.08±0.15†	59.72±16.32†	22.45±8.21†	34.88±10.7‡	1.12±0.19†
	Post	65.68±12.35	27.34±5.41	40.12±7.08	0.96±0.17	63.93±14.68	26.6±6.8	39.04±9.1	0.97±0.15
BAW	Pre	69.33±15.17	27.76±6.45	41.62±9.14	1.01±0.1	69.52±16.5	27.82±7.21	41.72±10	1±0.12
	During	66.52±15.42*	25.3±6.99*	39.04±9.53*	1.07±0.14	66.02±16.15†	24.93±7.43†	38.63±10.02†	1.08±0.18
	Post	67.46±14.81	27.47±6.61	40.8±9.07	0.99±0.12	68.28±18.5	28.03±8.06	41.46±11.25	0.98±0.13
QS	Pre	68.4±10.36	28.22±5.11	41.61±6.43	0.97±0.13	70.67±13.16	28.59±6.52	42.61±8.26	1±0.18
	During	68.16±10	27.81±5.39	41.26±6.46	0.98±0.15	70.93±12.91	28.55±6.72	42.68±8.3	1.01±0.19
	Post	67.5±10.37	27.49±5.31§	40.82±6.51	0.99±0.15	70.66±13.31	28.36±6.61	42.46±8.36	1.01±0.19

*P<0.05, †P<0.01, ‡P<0.001; *post hoc* analyses with Bonferroni adjustment, during compared with pre, §P<0.01; *post hoc* analyses with Bonferroni adjustment, post compared with pre. Values are group mean±SD. SD=Standard deviation, MCA=Middle cerebral arteries, HFYB=High frequency yoga breathing, BAW=Breath awareness, QS=Quiet sitting, ANYB=Alternate nostril yoga breathing

CI=-0.495,-0.043). There was no significant difference between pre- and post-values in any of the four variables.

BAW caused a reduction in MFV (P = 0.025; Cohen's d = 0.14; 95% CI = 0.157, 2.693), as well as EDV (P = 0.003; Cohen's d = 0.23; 95% CI = 2.796, 14.10) during the practice for the left MCA. At the right MCA BAW increased PI (P = 0.003; Cohen's d = 0.44; 95% CI=-0.127,-0.042) and decreased EDV (P = 0.010; Cohen's d = 0.27; 95% CI = 0.475, 3.638) during the practice. No significant differences were observed between pre and post values. There were no significant changes during and after QS.

Trial-2

Five minutes of ANYB caused the following changes. For the left MCA, there was a significant decrease in PSV (P = 0.013; Cohen's d = 0.35; 95% CI = 0.854, 8.477), MFV (P = 0.008; Cohen's d = 0.54; 95% CI = 1.026, 7.673) and EDV (P = 0.009; Cohen's d = 0.69; 95% CI = 0.949, 7.435) as well as an increase in PI (P = 0.004; Cohen's d = 0.87; 95% CI=-0.207,-0.035) during the practice of ANYB. The same changes were also apparent for the right MCA, with a significant decrease in PSV (P = 0.002; Cohen's d = 0.36; 95% CI = 2.015, 9.080), MFV (P = 0.001; Cohen's d = 0.49; 95% CI = 1.890, 7.863) and EDV (P = 0.002; Cohen's d = 0.61; 95% CI = 1.652, 7.433) as well as an increase in PI (P = 0.009; Cohen's d = 0.97; 95% CI=-0.259,-0.033) during ANYB. There were no significant differences when pre and post values were compared.

For the left MCA, there was a significant decrease in PSV (P = 0.036; Cohen's d = 0.18; 95% CI = 0.152, 5.480), MFV (P = 0.021; Cohen's d = 0.28; 95% CI = 0.331, 4.826) and EDV (P = 0.020; Cohen's d = 0.37; 95% CI = 0.333, 4.583) during BAW. For the right MCA, there was a significant decrease in PSV (P = 0.002; Cohen's d = 0.21; 95% CI = 1.236, 5.777), MFV (P = 0.002; Cohen's d = 0.31; 95% CI = 1.070, 5.123) and EDV (P = 0.003; Cohen's d = 0.39; 95% CI = 0.918, 4.862) during BAW. No significant changes were observed between pre- and post-values. QS caused a significant decrease in EDV (P = 0.008; Cohen's d = 0.06; 95% CI = 0.176, 1.293) after the practice for the left MCA.

Visual analog scale

For all trials, participants had visual analog scales' scores for maintaining the breath rate constant during the intervention, which was required to be greater than 7.0 on the 10.0 cm scale, for inclusion in the trial.

Discussion

Healthy volunteers showed a decrease in bilateral middle cerebral artery MFV and EDV during 5 min of ANYB, 1 min of HFYB, and during equal durations of BAW. During 1 min of HFYB alone, PSV decreased. In both

ANYB and HFYB as well as the corresponding BAW practice, the PI was higher compared to the respective preceding period. The changes did not persist in the post periods.

A decrease in cerebral blood flow velocity is correlated with decreased cerebral blood flow[11] while PI assesses resistance to intracranial blood flow.[12] Hence lower flow velocities and higher pulsatility indices of bilateral MCA during both yoga breathing practices are suggestive of reduced cerebrovascular flow volume and higher flow resistance, respectively.[13,14]

In HFYB the increased breath rate (around 2.0 Hz) and forceful exhalation are believed to increase the volume of carbon dioxide exhaled.[15] This is supported by a previous study on 47 healthy participants, where 15 min of HFYB resulted in a decrease in PaCO2 from 35.79 ± 2.23 mmHg (Pre) to 26.80 ± 2.40 mmHg (During) and 35.05 ± 2.14 mmHg (Post).[16] Hence HFYB appears to produce hypocapnia similar but of a lesser magnitude than hyperventilation-induced hypocapnia, which is relevant since hyperventilation decreases cerebral blood flow, cerebral blood volume and changes intracranial pressure.[17] The magnitude of hypocapnia may be speculated to be lesser during HFYB compared to frank hyperventilation since participants in the present study did not report symptoms of hyperventilation. This is consistent with previous assessments on 140 participants who performed HFYB but showed no evidence of hyperventilation discomfort.[18] Discomfort was determined using the Nijmegan Discomfort Questionnaire designed to detect symptoms of over-breathing.[19] Hence hypocapnia-induced changes may explain some, but possibly not all changes in CBF associated with HFYB. Other mechanisms are postulated below.

During ANYB, the changes in flow velocity and PI could be related to ventilation if the depth of breathing was increased. Previously, it was demonstrated that slow-paced breathing at 0.1 Hz can exert different effects on end-tidal CO2 based on the depth of breathing.[20] Hence, in the present study, during 5 min of slow breathing achieved during ANYB, the decrease in middle cerebral artery flow velocities and increase in the PI could be a consequence of hyperventilation-induced hypocapnia, only if ANYB practice was both slow as well as deep in excess of tidal breathing when it could be associated with hyperventilation rather than the speculated normoventilation. However, as for HFYB, after ANYB the participants in the present study did not report symptoms of hyperventilation, suggesting that the depth of breathing was not increased during ANYB. Future research could determine if maintaining yoga breathing during ANYB close to tidal breathing, would make a difference to cerebral blood flow changes.

An alternative explanation for the decreased cerebral blood flow velocity during ANYB, BAW and also during HFYB could be a decrease in the level of neural activity. Based on recognized principles of autoregulation of cerebral blood flow, the blood flow to the brain decreases when neural activity is low.[21] This kind of decrease in cerebral blood flow occurs during deep sleep (i.e., Slow Wave Sleep stages 3 and 4), when neural activation is considerably lower than during wakefulness, leading to reduced cerebral blood flow velocities compared to the wakeful state.[22] In the present study, all three practices (i.e., HFYB, ANYB, and BAW) may be associated with lower neural activation than the preceding state, based on previously published EEG studies.[23-25] Previously, increased relative power in the theta band and decreased beta activity were reported after ANYB practice,[23] increased alpha activity was reported during HFYB[24] and (iii) during BAW, alpha increased despite the performance of mentally engaging tasks.[25] These previous reports supported a mental state with low neural activity during the three practices (i.e., ANYB, HFYB and BAW).

Hence, the mechanisms involved in cerebrovasular hemodynamic changes during HFYB, ANYB and BAW appear to be complex. The findings during HFYB may be mediated partially by hyperventilation and also by reduced neural activity. The explanation for the effects of ANYB appears less likely to be due to hyperventilation, but more likely to be due to reduced neural activity. In BAW, reduced levels of neural activity could explain the changes in cerebral blood flow. Hence, chemical as well as changes in neural activity levels appear to influence cerebral blood flow in the MCA during these practices.

Yoga breathing was originally intended to develop control over the mental state as a training for meditation and more advanced spiritual practice. In a traditional yoga text (i.e., Hatha Yoga Pradipika Chapter II, Verse 2 (*Circa* 1350 CE), it is stated that ‘When the breath wanders, or is irregular, the mind is also unsteady, but when the breath is still, so also is the mind steady’. These descriptions are based on the experiences of the ancient sages.[26] In the ancient texts, there is also a description of spontaneous breath suspension described as *Kewala kumbhaka*, considered challenging to attain (Hatha Yoga Pradipika Chapter II, Verse 71–77).[26] This breath suspension could be attained through practices which are intended to induce hyperventilation. Hence, it is possible that the ancient sages did experience a degree of hypocapnia in advanced yoga breathing practice. Such a practice would have been motivated by an interest in regulating the mental state as a part of spiritual practice. Nowadays, yoga breathing is practiced for health benefits. While these breathing practices may not be harmful to healthy individuals, understanding the physiological effects of these practices can promote effective and safe practice by persons of different ages and baseline health levels. The findings are limited by the omission of training to the participants in the breathing techniques, exclusion of assessment of respiration rate and blood gas analysis

during the intervention sessions, and different durations of interventions. Restricting the participants to males with prior experience of yoga limits the generalizability of the findings.

Conclusions

Changes in peak flow velocities and pulsatility indices during and after HFYB, ANYB, and BAW suggest decreased cerebrovascular blood flow and increased flow resistance based on different mechanisms.

Acknowledgment

The authors are gratefully acknowledge the help of Mr. Vikas Upadhyay for contributing in data collection and data analysis.

Ethical clearance

Institutional ethical committee approval (YRD-018/016-17) was obtained.

Financial support and sponsorship

The authors gratefully acknowledge Patanjali Research Foundation for funding the study.

Conflicts of interest

There are no conflicts of interest.

References

1. Cipolla MJ. The Cerebral Circulation. San Rafael (CA): Morgan & Claypool Life Sciences; 2009.
2. Lipnick MS, Cahill EA, Feiner JR, Bickler PE. Comparison of transcranial Doppler and ultrasound-tagged near infrared spectroscopy for measuring relative changes in cerebral blood flow in human subjects. Anesth Analg 2018;126:579-87.
3. Naveen KV, Nagendra HR, Telles S, Garner C. Transcranial Doppler studies of middle cerebral artery blood flow following different test conditions. Neurol India 1999;47:249.
4. Ghiya S, Lee CM. Influence of alternate nostril breathing on heart rate variability in non-practitioners of yogic breathing. Int J Yoga 2012;5:66-9.
5. Gorini M, Misuri G, Corrado A, Duranti R, Iandelli I, De Paola E, *et al.* Breathing pattern and carbon dioxide retention in severe chronic obstructive pulmonary disease. Thorax 1996;51:677-83.
6. Patel S, Miao JH, Yetiskul E, Anokhin A, Majmundar SH. Physiology, carbon dioxide retention. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2021.
7. Telles S, Singh N. The psychophysiology of yoga regulated breathing (pranayamas). In: Research-Based Perspectives on the Psychophysiology of Yoga. Pennsylvania, USA: IGI Global; 2018.
8. 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.
9. Telles S, Verma S, Sharma SK, Gupta RK, Balkrishna A. Alternate-Nostril yoga breathing reduced blood pressure while increasing performance in a vigilance test. Med Sci Monit Basic Res 2017;23:392-8.
10. Ransil BJ, Schachter SC. Test-retest reliability of the Edinburgh Handedness Inventory and Global Handedness preference measurements, and their correlation. Percept Mot Skills 1994;79:1355-72.
11. Chan KH, Dearden NM, Miller JD. The significance of posttraumatic increase in cerebral blood flow velocity: A transcranial Doppler ultrasound study. Neurosurgery 1992;30:697-700.
12. Gosling RG, King DH. Arterial assessment by Doppler-shift ultrasound. Proc R Soc Med 1974;67:447-9.
13. Gdovinová Z. Blood flow velocity in the middle cerebral artery in heavy alcohol drinkers. Alcohol Alcohol 2001;36:346-8.
14. Harris S, Reyhan T, Ramli Y, Prihartono J, Kurniawan M. Middle cerebral artery pulsatility index as predictor of cognitive impairment in hypertensive patients. Front Neurol 2018;9:538.
15. Ansari RM. Kapalabhati pranayama: An answer to modern day polycystic ovarian syndrome and coexisting metabolic syndrome? Int J Yoga 2016;9:163-7.
16. Telles S, Singh N, Balkrishna A. Metabolic and ventilatory changes during and after high-frequency yoga breathing. Med Sci Monit Basic Res 2015;21:161-71.
17. Curley G, Kavanagh BP, Laffey JG. Hypocapnia and the injured brain: More harm than benefit. Crit Care Med 2010;38:1348-59.
18. Telles S, Singh N, Yadav A, Balkrishna A. Effect of yoga on different aspects of mental health. Indian J Physiol Pharmacol 2012;56:245-54.
19. van Doorn P, Folgering H, Colla P. Control of the end-tidal PCO2 in the hyperventilation syndrome: Effects of biofeedback and breathing instructions compared. Bull Eur Physiopathol Respir 1982;18:829-36.
20. Szulczewski MT. An anti-hyperventilation instruction decreases the drop in end-tidal CO_2 and symptoms of hyperventilation during breathing at 0.1 Hz. Appl Psychophysiol Biofeedback 2019;44:247-56.
21. Armstead WM. Cerebral blood flow autoregulation and dysautoregulation. Anesthesiol Clin 2016;34:465-77.
22. 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.
23. Telles S, Gupta RK, Yadav A, Pathak S, Balkrishna A. Hemisphere specific EEG related to alternate nostril yoga breathing. BMC Res Notes 2017;10:306.
24. 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.
25. Bing-Canar H, Pizzuto J, Compton RJ. Mindfulness-of-breathing exercise modulates EEG alpha activity during cognitive performance. Psychophysiology 2016;53:1366-76.
26. Muktibodhananda S. Hatha Yoga Pradipika. Munger, Bihar India: Yoga Publications Trust; 2002.