THE EFFECT OF CONSISTENT PRACTICE OF YOGIC BREATHING EXERCISES ON THE HUMAN CARDIORESPIRATORY SYSTEM

by

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Abstract

Yogic breathing exercises (YBE) are complex breathing patterns that can include hyperventilation, hypoventilation, and apnea. Some YBE can significantly alter blood gases and result in hypoxic hypercapnia. The consequence of consistent practice of these breathing exercises is unknown. Thus, the purpose of this Master's thesis was to quantify the cardiovascular, respiratory, and cerebrovascular effects of two common YBE: *bhastrika* and *chaturbhuj*; and, to determine the effect of their consistent practice on chemosensitivity. The first study was cross-sectional and compared experienced yogic breathers (YB) with matched controls in the above categories. It determined three things. First, *bhastrika* and *chaturbhuj* result in significant hypoxic hypercapnia. Second, the increase in blood pressure during their practice was higher in experienced yogic breathers. Third, experienced YB had reduced chemosensitivity compared to controls. The second was a controlled, longitudinal training study where experimental subjects practiced yogic breathing exercises for 6 weeks. This study had three major findings. First, after 6 weeks of training, *bhastrika* and *chaturbhuj* produced hypercapnia and mild hypoxia. Second, *chaturbhuj* resulted in cyclic oscillation of cardiovascular variables including blood pressure, heart rate, stroke volume, and cerebral blood flow velocity with inspiration and expiration. Third, post intervention there was no change in chemosensitivity measures. The findings from these two studies demonstrate that YBE significantly alter end-tidal gases, resulting in complex oscillations of cardiovascular and cerebrovascular variables, and if practiced for the long term, may reduce chemosensitivity.

Preface

Study 1 received ethical approval from the UBC Clinical Research Ethics Board (#H09-03202). A version of Study 1 has been previously published in Abstract form as: J.A.A. McKay, K. Stewart, J.S. Querido, G.E. Foster, M.S. Koehle, A.W. Sheel (2010). Long-term practice of yogic breathing exercises may be associated with increased blood pressure during breath-holds. Applied Physiology Nutrition and Metabolism. 35:S68. I identified the research question, designed the study, analyzed the data, and wrote the abstract for publication. Data collection was shared between K. Stewart and myself. M.S. Koehle, G.E. Foster, and J.S. Querido assisted with study setup and data analysis. All coauthors provided editorial feedback on the manuscript.

Study 2 received ethical approval from the UBC Clinical Research Ethics Board (#H10-02374).

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List of Abbreviations

BMI	Body mass index
bpm	Breaths per minute or Beats per minute
CBFV	Cerebral blood flow velocity
cm	Centimeter (s)
CO	Cardiac output
CO_2	Carbon dioxide
COPD	Chronic obstructive pulmonary disease
DBP	Diastolic blood pressure
DD	Dyspnea related distress
DI	Dyspnea index
EBT	Eucapnic Butekyo breathing technique
*f*b	Breathing frequency
FEV_1	Forced expiratory volume in 1 second
FYB	Full yogic breath
HCVR	Hypercapnic ventilatory response
HR	Heart rate
HVR	Hypoxic ventilatory response
kg	Kilogram (s)
L	Litre (s)
m	Meter (s)
MAP	Mean arterial blood pressure
MCAv	Middle cerebral arterial blood velocity
min	Minute (s)
mmHg	Millimeters of mercury
O_2	Oxygen
OSA	Obstructive sleep apnea
$PaCO_2$	Arterial partial pressure of carbon dioxide
PaO_2	Arterial partial pressure of oxygen
PCLE	Pink city lung exerciser
PEF	Peak expiratory flow rate
$P_{ET}CO_2$	End-tidal partial pressure of carbon dioxide
$P_{ET}O_2$	End-tidal partial pressure of oxygen
s	Seconds
SaO_2	Oxyhaemoglobin saturation with pulse oximetry
SBP	Systolic blood pressure
SD	Standard deviation
SV	Stroke volume
TCD	Trans-cranial doppler
TPR	Total peripheral resistance
V_E	Minute ventilation
V_t	Tidal volume

YB	Yogic breather (s)
YBE	Yogic breathing exercise (s)

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Chapter 1: Introduction

Control of breathing (*pranayama*) is one of the central teachings in classical yoga (Hari Dass, 1999; Muktibodhananda, 2006). To focus the human mind, traditional yoga texts instruct students to practice yogic breathing exercises (YBE). A single breath can be partitioned into four parts: inspiration, internal breath-hold, expiration, and external breath-hold. These four parts are combined, varied, and emphasized in different combinations in all YBE (Saraswati, 2006). For example, in the YBE *dirgha* rechak: the inspiration, internal breath-hold, and external breath-hold are the same as in a normal breath, but the expiration is as long and slow as possible. In addition, YBE are designed around four themes: hyperventilation, hypoventilation, breath-holds, and specific respiratory muscle recruitment patterns. Arguably, the most important theme in YBE is breath-holds (Muktibodhananda, 2006; Saraswati, 2006).

Recent research on YBE has shown that their practice can increase blood pressure (McKay *et al.*, 2010), and decrease the ventilatory response to low oxygen (hypoxic) and high carbon dioxide (hypercapnic) environments (Spicuzza *et al.*, 2000; Shannahoff-Khalsa *et al.*, 2004). However, there are significant gaps in the YBE literature. To date, the majority of published studies on YBE are observational in nature and lack quantification of type and amount (in days per week and hours per day) of YBE their subjects practiced. Of the published YBE training studies, many do not include precise interventions or control groups (Gokal *et al.*, 2007; Donesky-Cuenco *et al.*, 2009). For example, Donesky-Cuenco *et al.* (2009) found that a 12 week yoga intervention significantly increased 6 minute walk distance in chronic obstructive pulmonary disease

patients by 71.7 ± 21.8 feet. However, because the intervention included both YBE and physical postures, there was no way to determine which part of the intervention was associated with the change in walk distance. Furthermore, Gokal *et al.* (2007) followed participants in a 1 week yoga retreat and claimed that the decreases in weight, blood pressure, blood glucose, and blood cholesterol they reported were due to yoga and YBE practice. However, they did not include a control group.

Much like experienced breath-hold divers, experienced yogic breathers (YB) can endure extremely low levels of O_2, and high levels of CO_2. In a case study, Miyamura et al. (2002) observed an experienced YB perform the YBE *ujjayi* at a breathing frequency (*f*b) of 1 breath/minute for 1 hour. The subject's end-tidal O_2 ($P_{ET}O_2$) and CO_2 ($P_{ET}CO_2$) decreased to 62 mmHg, increased to 48 mmHg, respectively. *Ujjayi* is considered a novice YBE in which students are instructed to breathe slowly and deeply through the nose. During both the inspiration and expiration the glottis is slightly constricted so that the breath can be heard passing along the back of the throat.

Experienced YB have also been shown to have lower hypercapnic ventilatory sensitivity compared to control subjects. In the aforementioned case study, the experienced YB hypercapnic ventilatory sensitivity was measured using the Read re-breathing method and found to be 0.26 L/min/mmHg (Miyamura *et al.*, 2002). This is 85% lower than the hypercapnic ventilatory sensitivity reported for control subjects in another study (1.73 ± 0.84 L/min/mmHg) (Stanescu *et al.*, 1981).

Stanescu et al. (1981) measured hypercapnic ventilatory sensitivity in 8 experienced YBE practitioners and 8 well matched controls. They found that experienced YB were significantly less sensitive to CO_2 than controls (0.70 ± 0.29

L/min/mmHg vs. 1.73 ± 0.84 L/min/mmHg, $p < 0.05$). Similarly, Spicuzza et al. (2000) noted a reduction in hypercapnic ventilatory sensitivity and hypoxic ventilatory sensitivity in a separate group of experienced yoga practitioners, however, specific sensitivities were not provided in the research letter. These cross-sectional data suggest that there may be a dose-response relationship among the total amount of YBE practiced, and ventilatory sensitivity to hypoxia and hypercapnia. A well-controlled longitudinal YBE training study may help to quantify the influence YBE have on hypoxic and hypercapnic chemosenstivities.

During hypoxia and hypercapnia, the cardiovascular system operates in an attempt to maintain homeostasis. The cardiovascular system responds to apnea by reducing heart rate (bradycardia) and vasoconstricting the peripheral arteries (Grassi *et al.*, 1994; Daly, 1997). This response increases the amount of time before hypoxic damage occurs by conserving oxygen for the brain and heart (Foster & Sheel, 2005). To supply the brain with an adequate amount of oxygen in response to a hypoxic insult, there is an increase in CBFV (Querido *et al.*, 2008). CBFV also increases in response to hypercapnia (Ivancev *et al.*, 2007). However, in some pathological models, this response is blunted. In obstructive sleep apnea (OSA) patients, the increase in CBFV in response to hypercapnia is reduced (Foster *et al.*, 2009).

OSA patients suffer repeated bouts of hypoxic hypercapnia throughout sleep due to airway occlusion (Dempsey *et al.*, 2010). During a normal inspiration, upper airway dilators are activated to keep the upper airways open against rising inspiratory pressures. During sleep in OSA patients, there is a reduction in muscular tone of the upper airway dilators, specifically in the oropharynx. This results in a collapse of the airways when the

negative inspiratory pressure generated by the diaphragm, external intercostals, and other recruited inspiratory muscles is greater than the tone keeping the airways open. When this occurs, and in extreme cases it occurs 80 times an hour, despite repeated inspiratory efforts, OSA patients become progressively hypoxic and hypercapnic (Dempsey *et al.*, 2010). In a healthy individual, hypoxic hypercapnia results in an increase in CBFV, but in many OSA patients, this response is blunted (Placidi *et al.*, 1998).

The reason the CBFV response is blunted in OSA patients is not fully understood. Originally, it was thought that it was due to chronic intermittent exposure to hypoxic hypercapnia, however, now there is evidence that the blunted CBFV response in OSA patients is due to co-morbidities associated with the condition (Selim *et al.*, 2008). This theory is supported by research on breath-hold divers (Joulia *et al.*, 2009). Breath-hold divers are exposed to hypoxia and hypercapnia on a regular basis and their CBFV response to hypercapnia is unchanged (Ivancev *et al.*, 2007). However, comparisons between pathological reductions in O_2, and increases in CO_2, and voluntary apnea are difficult to make.

The literature lacks studies that have quantified the acute effects of YBE on human physiology. The physiological changes that occur during specific YBE need to be measured to determine if consistent practice of YBE correlates with measureable changes in cardio-respiratory physiology. Further study in this research area is necessary to further our understanding of the effects of YBE. The effect YBE may have on cerebrovascular reactivity to hypoxia and hypercapnia has not yet been investigated. By learning the cardiorespiratory and neurological effects of each YBE, it may be possible to

determine a prescription of YBE for a desired physiological effect such as reducing HR, blood pressure, or increasing CBFV in OSA patients.

Purpose

The purpose of this Master's thesis was four-fold. First, to measure the changes in cardiovascular, cerebrovascular, and respiratory variables during the practice of two YBE, *bhastrika*, and *chaturbhuj*. Second, to determine if long term practice of YBE affected hypercapnic ventilatory threshold and sensitivity. Third, to determine if regular practice of YBE for 6 weeks was a sufficient stimulus to decrease hypercapnic ventilatory sensitivity and increase hypercapnic ventilatory threshold. Fourth, to determine if the practice (either long term or 6 weeks) of YBE changed the response of cardiovascular, cerebrovascular, and respiratory variables during the same YBE.

Hypothesis

The hypothesis was five-fold. First, it was hypothesized that during both *bhastrika*, and *chaturbhuj*, $P_{ET}O_2$ would decrease and $P_{ET}CO_2$ would increase, resulting in a hypoxic, hypercapnic stimulus. Second, it was hypothesized that subjects experienced in YBE would have decreased hypercapnic ventilatory sensitivity and increased hypercapnic ventilatory threshold when compared to controls. Third, it was hypothesized that a 6 week intervention of 5 times weekly practice of YBE would result in decreased hypercapnic ventilatory sensitivity and increased hypercapnic ventilatory threshold – both measured by the Duffin re-breathing method – and increased breath-hold time. Fourth, it was hypothesized that the CBFV response to hypercapnia would be

unchanged after the intervention. Fifth, based on data from Study 1 (Chapter 3, Figure 2), it was hypothesized that MAP would increase more in response to *bhastrika* and *chaturbhuj* after the 6 week intervention.

Chapter 2: Literature Review

Introduction

For many years in India, and currently in the Western world, yogic breathing exercises (YBE) have been used with varying success for the treatment of chronic physiological and psychological disorders such as asthma, chronic obstructive pulmonary disease and depression (Cooper *et al.*, 2003; Brown & Gerbarg, 2005; Pomidori *et al.*, 2009). People practice YBE with different goals in mind; some for the purpose of relaxation and spiritual gain, others with the hope that YBE will provide physiological or psychological benefit. YBE are designed around four themes: hyperventilation, hypoventilation, breath-holds, and specific respiratory muscle recruitment patterns. Though comparatively little research has been done on the physiological effects of specific YBE, there has been much research on the physiological effects of both hypoventilation and apnea in other populations. Breath-hold divers, underwater hockey players, and sleep apnea patients regularly experience repeated bouts of apnea and hypoventilation. This review will use these, and other well-studied populations to provide insight into the physiological adaptations that may occur with long-term practice of YBE.

Breath-hold divers can withstand breath-holds to arterial oxygen (PaO_2) levels as low as 28 mmHg and arterial carbon dioxide ($PaCO_2$) levels above 55 mmHg. In comparison, most non-divers can only endure to a PaO_2 of 45 mmHg and $PaCO_2$ of 50 mmHg (Ferretti, 2001). Physiologically, this 17 mmHg difference in PaO_2 corresponds to two very different arterial oxyhaemoglobin saturations (SaO_2: ~ 53%, and 81% respectively) because of the shape of the oxygen-hemoglobin dissociation curve

(Severinghaus, 1979; West, 2005). In addition to extreme levels of mental fortitude, enduring a breath hold until SaO_2 falls below 60% requires many physiological changes that this review will discuss.

The purpose of this review is fourfold. First, to provide a complete definition of YBE. Second, to examine the physiological effects of YBE on the human cardiorespiratory system and provide insight into their clinical effectiveness or absence thereof. Third, to examine the effects of other repeated apnea models such as breath-hold diving and obstructive sleep apnea on hypoxic (HVR) and hypercapnic (HCVR) ventilatory response and threshold, blood pressure and heart rate regulation, and middle cerebral artery blood velocity (MCAv). Finally, this review aims to provide a clear direction for further research on YBE.

Methods

YBE research for this review was compiled from the MEDLINE database (1950 - 2010), the American Physiological Society (1948 – 2010), and the *Journal of Alternative and Complimentary Medicine* (1998 – 2010). Searches were performed using both MeSH (Medical Subject Headings) terms, and text keywords. MeSH terms included: yoga, respiration, and breathing exercises. Keyword searches were combined with Boolean operators and included the truncated keywords: yog*, breath*, and pranayam*. Traditional information and theories were researched in English translations of Indian texts where YBE are described (Muktibodhananda, 2006) and in more recent publications (Hari Dass, 1981; Saraswati, 2006).

Defining yogic breathing exercises

The traditional name for YBE, *pranayama*, comes from the Sanskrit language and can be broken down into three parts: *prana*, *yama,* and *ayama*. *Prana* is translated in the yoga sutras as the "vital energy" that animates all things (Hari Dass, 1999). *Yama* means "control" or "restraint," and *ayama* means "expansion." Together, *pranayama* means "the control and expansion of vital energy" (Saraswati, 2006). The traditional purpose of *pranayama* practice was not to simply increase breath hold time. The yoga sutras – the original Indian text outlining the practices of yoga – explain that by developing control of the breath, one develops control of the mind (Hari Dass, 1999). This section will explain the foundations of YBE and provide their inclusive definition.

The full yogic breath (FYB)

The respiratory muscle recruitment pattern during a full yogic breath (FYB) is important to understand because the majority of YBE use the FYB. The FYB is most simply described as a full, deep inspiration followed by a full deep expiration. Though it is not possible for someone to choose which part of the lung they ventilate, it may be possible for them to choose the respiratory muscles activation pattern. For example, by moving the chest up and keeping the abdomen in the same place during inspiration, the majority of the inspiratory work would be done by the external intercostal muscles and the sternocleidomastoid. When taught by a yoga instructor, the inspiratory portion of the FYB is separated into three phases. First, subjects are instructed to activate the diaphragm with the cue "Inhale into the bottom of the lungs by pushing the stomach out." Next, subjects are instructed to "Continue the inhale into the middle portion of the torso

by expanding the lower rib cage." Finally, subjects are instructed to "Fill the top of the lungs by expanding the chest up and out." After a short pause, subjects expired with the instructions "Relax the upper chest, then the ribcage, and finally pull the abdomen in slightly." A common analogy that was used when teaching the FYB was the filling and emptying of a jar (lungs) with water (air). The jar always fills from the bottom to the top, and always empties from the top to the bottom. The FYB is done through the nose. Though students are taught to visualize a FYB cycle in this manner, we have yet to find published data that confirms their ability to selectively ventilate the upper and lower sections of the lung.

Three traditional YBE

This section will describe three common traditional YBE: *chaturbhuj, bhastrika,* and *ujjayi*. *Chaturbhuj* is a hypoventilation breathing pattern that uses the FYB. During which, the breath is divided into four equal parts: inspiration, breath-hold, expiration, breath-hold. Each part is performed for equal amounts of time, resulting in an inspiration:breath-hold:expiration:breath-hold ratio of 1:1:1:1. Preliminary data on the cardiorespiratory and cerebrovascular changes that occur during *chaturbhuj* and *bhastrika* from this lab are shown above (Figures 2, 3).

Bhastrika, translated as "breath of fire," or "bellows breath," is one of the most common YBE used today (Muktibodhananda, 2006; Saraswati, 2006). It is important to note that the term *bhastrika* is used in the published literature to describe more than one YBE. For the purposes of this review, the term *bhastrika* will be used to describe a YBE that is: one to two minutes of "abdominal hyperventilation" through the nose followed by

an inspiration to TLC and maximal breath-hold. The breathing pattern used in *bhastrika* is different than the FYB described above. *Bhastrika's* "abdominal hyperventilation" breath is simply the first step of the FYB inspiration followed by a forceful exhale through the nose.

Ujjayi is a beginner YBE that doesn't require a specific inspiration:expiration ratio or a FYB. Students are instructed to inspire through the nose and to expand the stomach by activating the diaphragm. Additionally, during both the inspiration and expiration, the glottis is closed slightly so that the breath can be heard passing along the back of the throat. *Ujjayi* is a very common YBE that is most often used in *asana* (yoga posture) classes to generate heat in the body. Though data on the effect of *ujjayi* on body temperature has not been published, based on other research it seems possible to raise body temperature while sitting still. Previously, three advanced Tibetan monks were able to increase their body temperature (+ 8°C measured at the toe) during a Tibetan meditation practice (Benson *et al.*, 1982).

Defining YBE

YBE have previously been defined as breathing practices where the time spent expiring is twice the duration of the time spent inspiring (inspiratory:expiratory ratio of 1:2) (Singh *et al.*, 1990; Cooper *et al.*, 2003). This definition encompasses only a small portion of the many different YBE. YBE are designed around four themes: hyperventilation, hypoventilation, breath-holds, and specific respiratory muscle recruitment patterns (Muktibodhananda, 2006; Saraswati, 2006). Additionally, each breath can be subdivided into four parts: inspiration, internal breath-hold, expiration, and

external breath-hold. These four parts are combined, varied, and emphasized in different combinations in all YBE (Saraswati, 2006). With the exception of *ujjayi*, which can be practiced during yoga postures, YBE are done in a seated position.

Lessons learned from other models of apnea and timed breathing patterns

This section will focus on apnea models and timed breathing exercises other than YBE. It will also outline the cardiovascular and cerebrovascular responses to apnea in underwater sportsmen. Examination of these models will provide insight into the potential physiological effects of apnea-focused, and hypoventilatory YBE.

The Pink City Lung Exerciser (PCLE) and The Eucapnic Butekyo Breathing Technique (EBT)

Most of the research that examines the effect of breathing exercises on symptom reduction in mild asthmatics focuses on inhaler devices that claim to mimic YBE, such as the PCLE. The PCLE is a plastic mouthpiece with an adjustable aperture. There is a one-way valve that opens at the onset of inspiration and closes at the onset of expiration, halving the diameter of the tube during expiration.

Previous researchers found that after using the PCLE twice a day for 2 weeks, mildly asthmatic patients increased the dose of histamine necessary to provoke a 20% reduction in their forced expiratory volume in 1 second (FEV_1), a reduction comparable to a low-dose of inhaled cortical steroids (Singh *et al.*, 1990). To specify, the asthmatics in this study were not classified as sufferers of exercise-induced or allergenic asthma. They all had FEV_1 values greater than 60% of predicted, and the dose of histamine

required to decrease FEV_1 by 20% was less than 4μmol. Following the PCLE intervention, resting FEV_1, peak expiratory flow rate, symptom score, and inhaler use were not different between groups, but the authors suggest that this may be because the subjects were only mildly asthmatic.

Another explanation focuses on the device itself. Halving the expiratory diameter does not guarantee a doubling of the time spent expiring. In fact, Poiseuille's law states that flow through a tube is proportional to radius to the 4^{th} power (West, 2005). It follows then, that if the radius of the PCLE is halved, and expiratory pressure does not increase, then the flow rate would be 16 times slower during expiration – not two times. Additionally, if the intervention claims to reduce minute ventilation (V_E) through breathing frequency (*f*b), then both *f*b and tidal volume (V_t) need to be measured to confirm that subjects are not compensating for the smaller airway diameter by breathing deeper or more frequently.

A more recent PCLE 6-month intervention study used both the PCLE and the EBT. This study found no difference in asthma outcome measures between the PCLE and placebo group. However, they did notice a reduction of β_2-agonist use in the EBT group vs. both the placebo and PCLE groups respectively (Cooper *et al.*, 2003). The PCLE does not accurately recreate YBE for two reasons. First, breath-holds are arguably the most important part of any YBE routine and practice with the PCLE does not include apnea. Second, increasing expiratory duration is part of many YBE, however, expiratory resistance rarely changes. More research needs to be done on the PCLE to first determine the exact ratio of time spent inspiring to time spent expiring.

The EBT is a breathing technique very similar to YBE that includes slow, shallow breathing and breath-holds. It was developed by Konstantin Buteyko to help reduce asthma symptoms through a reduction in V_E. He theorized that chronic hyperventilation led to hypocapnia, which caused the airways to constrict in order to conserve CO_2 (Bruton & Lewith, 2005). In a study by (Cooper *et al.*, 2003), subjects were instructed to use the EBT twice a day, and as a tool to relieve asthma symptoms instead of an inhaler. Subjects recorded breath-hold time and practiced breathing exercises that reduced both their *f*b and V_t. Additionally, subjects were instructed to tape their mouth shut at night to reduce mouth breathing, avoid strenuous exercise, and to avoid highly processed foods. All of these changes resulted in the reduced β_2-agonist use noted above. In contrast, the PCLE group used the device twice daily for 15 minutes with no significant difference in any measure after the intervention (Cooper *et al.*, 2003).

Cardiovascular and cerebrovascular responses to apnea

As the duration of apnea or hypoventilation increases, the human body becomes increasingly hypoxic and hypercapnic. During these practices, the cardiovascular system works to keep the human body in homeostasis. The purpose of all the cardiovascular responses to apnea have the same purpose – to conserve oxygen for the brain and the heart while increasing the amount of time before hypoxic damage occurs (Foster & Sheel, 2005). The two major cardiovascular changes that occur during apnea to accomplish this goal are a reduced heart rate (bradycardia) and peripheral vasoconstriction, (Grassi *et al.*, 1994; Daly, 1997). Bradycardia preserves O_2 stores by effectively decreasing the amount of work done by the heart and therefore decreasing its oxygen uptake (VO_2) (Foster &

Sheel, 2005). Cardiac output is the product of heart rate and blood pressure, and it follows that peripheral vasoconstriction serves two purposes. First, it preserves oxygenated blood and reduces whole body VO_2 by reducing blood flow to the skeletal muscles, skin, and visceral system. Second, it preserves cardiac output during bradycardia. Both of these cardiovascular responses result in a larger pool of O_2 available for the heart and brain, which would increase breath-hold time and the time before tissue damage occurs.

During static apnea, the HR of trained breath-hold divers dropped an average of 25% (80 bpm to 60 bpm) during the first 2 and a half minutes of apnea (Lindholm *et al.*, 2006). Another study compared elite breath-hold divers with matched controls in their cardiovascular response to maximal static apnea and found similar results. Elite divers' HR dropped from 78 bpm at rest to 61 bpm after 2 minutes of static apnea and remained unchanged until inspiration, whereas the HR of controls did not change. Additionally, cerebral blood flow, measured at the carotid artery, increased almost 3-fold in the divers (approximately 0.2 L/min to 0.6 L/min from rest to end apnea), and did not change in the control group (Joulia *et al.*, 2009). This study found that at the same apnea duration, breath-hold divers had larger increases in cerebral blood flow with smaller decreases in SaO_2. No data on CO_2 levels during apnea was presented (Joulia *et al.*, 2009). Although CO_2 is thought to be the primary determinant of changes in cerebral blood flow (Przybylowski *et al.*, 2003), the vascular response to apnea, mental state, and sympathetic nerve activity may also play a role in cerebrovascular regulation (Joulia *et al.*, 2009).

In healthy subjects, hypercapnic re-breathing tests resulted in increased middle cerebral artery blood velocity (MCAv) (52.52 ± 11.84 to 86.45 ± 16.02, $P < 0.05$)

(Ivancev *et al.*, 2009). This response is preserved in breath-hold divers (Ivancev *et al.*, 2007). Interestingly, obstructive sleep apnea (OSA) patients who also endure hypoxia and hypercapnia for extended periods of time, tend to have decreased cerebrovascular reactivity – a smaller increase in cerebral blood velocity in response to hypoxia and hypercapnia (Placidi *et al.*, 1998; Foster *et al.*, 2009). Though both groups endure hypoxic hypercapnia, breath-hold divers endure a second stimulus when diving to depth; they experience hyperoxic hypercapnia due to hydrostatic pressure. This environmental difference may explain part of the difference in cerebrovascular reactivity. However, more plausible is the difference in body mass index (BMI) between these groups. OSA is highly correlated with obesity (BMI > 30), whereas breath-hold divers are typically not obese (BMI 20 – 25) (Gold *et al.*, 1993; Grassi *et al.*, 1994; Delapille *et al.*, 2001). A recent study in rats demonstrated that obesity and hypertension were associated with decreased lumen diameter in the middle cerebral artery (MCA) due to increased cerebrovascular myogenic tone and inward remodeling (Osmond *et al.*, 2009). In humans, obesity, independent of hypertension was associated with decreased cerebral blood velocity, and increased cerebrovascular resistance (Selim *et al.*, 2008). There have yet to be any studies examining cerebrovascular reactivity in experienced yogic breathers (YB).

Physiological control of ventilation

The physiological effects of YBE cannot be explained without considering ventilatory control. Originally, it was thought that ventilation was controlled by two separate systems – the peripheral and central chemoreflexes – which responded

independently to oxygen (O_2) and carbon dioxide (CO_2) respectively. It is now evident that the chemoreflex systems are mutually dependent (Duffin & Mahamed, 2003; Day & Wilson, 2007). As a whole, the human chemoreflex system is made up of three parts: sensors, effectors, and the central controller. The peripheral chemoreceptors are located at the bifurcation of the carotid artery in the carotid body, and in the aortic arch. Their primary job is to measure the amount of O_2 in arterial blood. Animal denervation studies have shown that the hypoxic ventilatory drive originates solely from the peripheral chemoreceptors (Izumizaki *et al.*, 2004). Recent work has shown that the carotid peripheral chemoreceptors also contribute approximately one third of the total HCVR while the central chemoreceptors contribute the other two thirds (Forster *et al.*, 2008). The peripheral chemoreceptors relay information through the 9th cranial nerve to the respiratory centres in the medulla for integration (West, 2005).

The central chemoreceptors are surrounded by the brain extracellular fluid and are anatomically located just below the ventral surface of the medulla in the brainstem, adjacent to the cerebrospinal fluid (CSF). They are sensitive only to CO_2. As blood levels of CO_2 rise, CO_2 diffuses through the blood brain barrier and brain extracellular fluid, into the CSF causing increased H^+ concentration and decreased pH in the CSF. The central chemoreceptors respond to decreased pH with increased firing (West, 2005).

There are two systems of "higher control," the brainstem and the cortex. The neurons in the brainstem are responsible for the involuntary, periodic nature of inspiration and expiration. Based on information from the chemoreceptors, they can increase or decrease their firing rate through the phrenic nerve to regulate ventilation. To a point, signals from the cortex can override the brainstem to produce voluntary changes

in ventilation such as hyper/hypoventilation and prolonged apnea (West, 2005) (Ferretti, 2001). This is important in YBE because ventilation is voluntarily controlled.

Duffin and colleagues have extensively researched human ventilatory sensitivity to $PaCO_2$ and PaO_2. Though complex and quite variable (up to 27% within subjects (Koehle *et al.*, 2005)), the relationship between $PaCO_2$ and ventilation is linear past the hypercapnic ventilatory threshold. This threshold is well defined in many review articles (Duffin *et al.*, 2000; Duffin & Mahamed, 2003). Briefly, the hypercapnic ventilatory threshold is the $PaCO_2$ above which, there is a measurable increase in ventilation. This threshold is not static: in hypoxia it decreases, and in hyperoxia it increases (Duffin *et al.*, 2000). The same relationship occurs with hypercapnic ventilatory sensitivity.

Hypercapnic ventilatory sensitivity can be modified with apnea or intermittent hypoxia (IH) (repeated bouts of exposure to low oxygen levels) training. Underwater hockey players and breath-hold divers have reduced hypercapnic ventilatory sensitivities when compared to above water athletes (55% and 77% vs. controls, respectively (Davis *et al.*, 1987; Delapille *et al.*, 2001)). Lacking in the literature is measurement of the hypercapnic ventilatory threshold in underwater sportsmen and YBE practitioners. Intuitively, repeated exposure to hypoxia and hypercapnia should result in acclimation to the stimuli, and a reduced physiological response. However, following one week of IH exposure for 60 minutes at 12% O_2 per day, the hypercapnic ventilatory threshold decreases – subjects began to increase ventilation at lower levels of $PaCO_2$ (Mahamed & Duffin, 2001; Koehle *et al.*, 2007).

Research has shown that there are two breaking points during a breath-hold: the physiological break point, and termination of the breath-hold (Lin *et al.*, 1974), which

may be psychological rather than physiologic. The physiological break point occurs when the subject begins to make involuntary ventilatory movements and continuation of the breath-hold requires increasing inhibitory input from the cortex. This process is common in YBE that incorporate breath holds and it would be interesting to learn which breaking point adapts more as breath-hold time increases.

The physiological effects of YBE and other breathing patterns

This section will examine the intervention studies that have used traditional YBE, and other breathing exercises, as stimuli, and measured their effect on human physiology. A summary of relevant studies is shown in Table 1. Though there are many studies that have found YBE to be effective in the treatment of stress, depression, and other mental disorders ((Shannahoff-Khalsa & Beckett, 1996; Naga Venkatesha Murthy *et al.*, 1998; Brown & Gerbarg, 2005), the remainder of this review will focus on the proposed physiological benefits of YBE.

Table 1. Characteristics of published YBE studies

Study	Design	Subjects	Stimulus/Test	Control group	Outcome measures
Stanescu et al. 1981	Cross Section	YB	HCVR	Yes	↓ Hypercapnic ventilatory sensitivity vs. controls (0.70 ± 0.29 L/min/mmHg vs. 1.73 ± 0.84 L/min/mmHg)
Spicuzza et al. 2000	Cross Section	YB	HVR, HCVR	Yes	↓ Hypoxic and hypercapnic ventilatory sensitivity vs. controls
Miyamura et al. 2002	Case Study	YB	*Ujjayi* YBE @ 1 bpm	No	↓ Hypercapnic ventilatory sensitivity (0.26 L/min/mmHg)
Shannahoff-Khalsa et al. 2004	Case Study	YB	(1:1:1 YBE @ 1 bpm)	No	MAP and HR cycle with inspiration and expiration
Gokal et al. 2007	Intervention	Obese/Diabetic/ Hypertensive	YBE, yoga postures, diet	No	↓Weight, MAP, blood glucose and cholesterol
Pramanik et al. 2009	Cross Section	Sedentary people (25-35 yrs old)	5 minutes YBE: (1:1.5 inspire:expire ratio)	Yes	↓MAP - No change in MAP during YBE with PNS blocking drug
Bernardi et al. 1998	Intervention	CHF patients	1 month, 1 hr/day FYB practice (~6 bpm)	Yes	↑SaO_2 @ same V_E during FYB ↑VO_2 in cycle test
Bernardi et al. 2001	Cross Section	YB	Hypobaric hypoxia (Chamber @ 5000 m)	Yes	↑SaO_2 @ same V_E ↓SNS outflow

The FYB

People who practice YBE tend to have slower and deeper resting breathing patterns than controls – matched for age, weight, and body surface area – because they use the FYB muscle recruitment pattern (Spicuzza *et al.*, 2000). Following one month of respiratory training, the FYB has been shown to increase SaO_2 at *f*b of 6 bpm, compared to spontaneous breathing in chronic heart failure (CHF) patients. In this study, V_E was unchanged between spontaneous breathing and FYB at a *f*b of 6 bpm. The reason is reduced deadspace; the dead space V_E : V_t ratio decreased from 38.7±2.0 to 25.9±1.5, $P < 0.05$. The FYB effectively ventilated more of the lung while moving the same amount of air. This was confirmed with alveolar ventilation data (Bernardi *et al.*, 1998). These effects are significant on both a statistical and practical level. The experimental group

also increased peak wattage achieved, and whole-body VO_2 in a cycle ergometer exercise test (Bernardi *et al.*, 1998). This is promising for patients who are affected by respiratory disorders such as chronic obstructive pulmonary disease (COPD) and asthma.

Another study (Pomidori *et al.*, 2009) that looked at the effect of FYB in patients who suffered from moderate to severe COPD supports the above CHF study (Bernardi *et al.*, 1998). The authors found that without changing minute ventilation, the FYB increased resting SaO_2 and reduced *f*b; confirming that the FYB produces a deep, efficient, and slow breathing pattern. This group also noted an interesting correlation; the patients who desaturated the most during the 6-minute walk distance test had the largest increase in SaO_2 during the FYB ($r = 0.78$). These data suggest that COPD patients who experience the most arterial O_2 desaturation and dyspnea during daily activities may experience the largest benefit from using the FYB breathing pattern.

These findings are consistent with the literature on pursed lip breathing. Pursed lip breathing is commonly used with COPD patients. Patients inspire through the nose, and then expire slowly through pursed lips. Several studies have shown that this breathing pattern results in increased SaO_2, decreased *f*b, unchanged V_E, and significant dyspnea relief in some COPD patients with varying degrees of severity (Dechman & Wilson, 2004). Positive effects of pursed lip breathing are thought to be due to decreased *f*b, while the pursing of the lips just a method for the reduction (Thoman *et al.*, 1966). The patients who were unrelieved did not experience tracheobronchial collapse during expiration whereas the relieved patients did (Ingram & Schilder, 1967). The pursed lip breathing pattern most likely improves dyspnea by reducing airway constriction during expiration because of decreased expiratory flow rate.

Interventions using both YBE & yoga postures

Another study on COPD patients found that a 12 week intervention of yoga postures (stretching, twisting, balance and strengthening poses) and simple YBE – inspiration:expiration ratio of 1:2 - had no effect on feelings of breathlessness (dyspnea index (DI) and dyspnea related distress (DD)) (Donesky-Cuenco *et al.*, 2009). DI and DD were assessed using the questions "how short of breath are you right now?" and "how bothersome or worrisome is your shortness of breath to you right now?" respectively, and rated numerically using a modified Borg scale during cycle ergometer exercise. Similar to the study by Pomidori *et al*., (2009) study, a significant change noted in the treatment group was an increased distance covered in the 6-minute walk test (Donesky-Cuenco *et al.*, 2009). Whether the increase in physical capacity was due to the YBE, the postures, or both, cannot be determined because the control group practiced neither the YBE nor the postures. The authors postulate that the reason no changes were seen in DI or DD may be because their intervention was too mild.

With a large study population of patients with diabetes, hypertension, obesity, and dyslipidemia, (Gokal *et al.*, 2007) reported that a one week long yoga retreat where participants practiced YBE, postures, and meditation, resulted in statistically significant positive changes in BMI, mean arterial pressure (MAP), blood glucose, and cholesterol levels. Though positive, these results are not conclusive and need to be carefully interpreted. A one-week yoga retreat is very much a short-term lifestyle change. Food is sometimes provided, and participation in classes is voluntary. The 'intervention' experienced by each person would be unique. Also, lacking a control group, it was not

possible for the authors to determine which part of the intervention was responsible for the positive outcome (Gokal *et al.*, 2007).

Cardiorespiratory adaptations to traditional YBE

The limited research on the effects of YBE on arterial blood gasses suggest that much like experienced breath-hold divers, experienced YBE practitioners can endure extremely low levels of O_2, and high levels of CO_2. In a case study, (Miyamura *et al.*, 2002) observed an experienced YBE practitioner perform *ujjayi* at a *f*b of 1 breath/minute continuously for 1 hour. End-tidal O_2 ($P_{ET}O_2$) and CO_2 ($P_{ET}CO_2$) reached extreme values of 62 and 48 mmHg, respectively. Contrary to the diving response to hypoxia and hypercapnia, the subjects HR increased from 69 to 75 bpm, perhaps due to the length of the practice. The subjects HCVR was measured using the Read re-breathing method and found to be quite low (0.26 l/min/mmHg). Similar results were found by (Stanescu *et al.*, 1981), who measured HCVR in 8 experienced YBE practitioners and 8 controls. He found that experienced yogic breathers were significantly less sensitive to CO_2 than controls (0.70 ± 0.29 l/min/mmHg vs. 1.73 ± 0.84 l/min/mmHg, $p < 0.05$). The difference in HCVR between Miyamura's and Stanescu's experienced practitioners is most likely due to their experience levels. Although, Miyamura's experiment was a case study, the subject had reportedly been practicing YBE for 19 years, 2 to 2.5 hours per day. Stanescu's experienced practitioners were reported to have been practicing YBE for 4 to 12 years, but the duration per day was unclear, Stanescu reports "up to 2 hours per day." Similarly, (Spicuzza *et al.*, 2000) noted a reduction in HCVR and HVR in experienced yoga practitioners compared to controls, however, specific slopes were not

provided in the research letter. Though these data suggest that there may be a dose response relationship between the length of time and amount of YBE practiced, and hypercapnic chemosensitivity, studies examining the physiology of extreme YBE practitioners such as the one in Miyamura's study are rare; most likely due to the scarcity of such advanced practitioners.

VO_2 can change during YBE, though the direction and magnitude depend on the specific YBE. In a recent study it was found that a 1:4:2 inspiration:breath-hold:expiration ratio resulted in increased VO_2 compared to both rest and meditation (Danucalov *et al.*, 2008). However, a major limitation of this study was that they did not report V_t, *f*b, or V_E data. Without that data, it is impossible to know the degree to which subjects hyper-or-hypoventilated during the YBE.

Altitude is a hypobaric, hypoxic stimulus and acute exposure usually results in increased ventilation (Bernardi *et al.*, 2001). A study comparing yoga trainees to matched controls examined the cardiorespiratory changes that accompanied simulated altitude to 5000m (Bernardi *et al.*, 2001). At 5000m and the same or lower V_E, the yoga trainees had higher SaO_2 in three cases: spontaneous breathing, controlled breathing at 15 bpm, and during the FYB. Interestingly heart rate and blood pressure variability were measured as indices of SNS activation. SNS activity increased at altitude, but the response was blunted in the yoga trainees and during controlled breathing and the FYB in both groups.

Another study (Pramanik *et al.*, 2009) looked at the possible effects of "*bhastrika*" on the parasympathetic nervous system (PNS) by measuring cardiovascular variables during the YBE with and without ingestion of hyoscine-*N*-butyl bromide – a

proven parasympathetic blocking drug (Bittiner & Smith, 1986). It is important to note that the YBE used in this study is different than the *bhastrika* described in section 3.2. The YBE described in the cited study is a slow inhale of 4 seconds, followed by a slow exhale of 6 seconds, resulting in a *f*b of 6 bpm. To refresh, *bhastrika* is a YBE that begins with a 1 minute hyperventilation, followed by a maximal inhale to TLC, and then a maximal breath-hold. They hypothesized that YBE decreased blood pressure through increased PNS output. Under normal conditions, they found that 5 minutes of 6 bpm breathing lowered MAP. After a dose of the PNS blocking drug, the same YBE was not accompanied by a reduction in MAP (Pramanik *et al.*, 2009). This data shows if you remove PNS activity, MAP does not change in response to this YBE. It does not however, prove that under normal conditions, this YBE reduces MAP through increased PNS output. Sympathetic activity was not measured though it directly affects MAP through arterial vasoconstriction (Mohrman David E., 2006). This would have been beneficial as earlier research found that SNS activity is blunted during slow breathing (Bernardi *et al.*, 2001). Further study needs to examine both nervous systems to determine the degree to which both play a role in blood pressure regulation in YBE.

(Shannahoff-Khalsa *et al.*, 2004) conducted a case study on the hemodynamic changes that occurred during an intermediate, 1 bpm YBE. The pattern was 20 seconds of inspiration followed by a 20 second breath-hold, and then a 20 second expiration, repeated for 31 minutes. They found that MAP, and HR cycled with the breath during the YBE and the cycling pattern continued for 10 minutes post-test. This continuation suggests that the pattern generators in the cardiorespiratory control centre of the brainstem were altered by the 20:20:20 breath. Further research is needed to determine if

this adaptation is first, consistent between subjects, second, occurs at breathing frequencies higher than 1 breath per minute, and third, if it would be beneficial for patients with cardiac arrhythmias.

Recently, a case report was published describing spontaneous pneumothorax in an otherwise healthy 29 year-year-old woman one day after performing *kapal bhati* YBE (Johnson *et al.*, 2004). This introductory YBE is a shallow, rapid, repeated breath through the nose. The pattern begins with a forceful expiration from about half of TLC. During the expiration, the abdomen is pulled in towards the spine. At the end of the expiration, the abdomen is relaxed which results in an inhalation, back to about half of TLC. The case report does not offer any hypotheses to the method of this pneumothorax. Contra-indications for the practice of *kapal bhati* include: heart disease, high blood pressure, vertigo, epilepsy, stroke, hernia, or gastric ulcer (Saraswati, 2006). This is the only published case associating YBE with clinical problems.

Future directions

Primary to continued research on the effect of YBE on human physiology is quantification of the stimulus. The physiological changes that occur during specific YBE need to be measured to determine if consistent practice of YBE correlates with measureable changes in cardio-respiratory physiology. Further study in this research area is crucial to our understanding of the effects of YBE. By learning the exact cardiovascular, respiratory, and neurological effects of each YBE, it may be possible to determine a prescription of YBE for a desired physiological effect such as reducing dyspnea, HR, blood pressure or increasing cerebrovascular reactivity in OSA patients.

Conclusion

Though the practice of YBE has been associated with decreased ventilatory sensitivity to hypoxia (Spicuzza *et al.*, 2000) and hypercapnia (Miyamura *et al.*, 2002), preliminary research using YBE in clinical populations has yet to meet with great success. Due to limited research on the physiological changes that occur during specific YBE, the reliance on small groups, cross-sectional study designs, and a lack of suitable control groups, the effects of YBE on human physiology are still quite unclear. Further study and refinement of the interventions may help to determine specific combinations of YBE that are effective interventions for asthmatics, COPD sufferers, and OSA patients. Additionally, to be able to prescribe these practices as alternatives or in addition to conventional treatment, more research needs to focus on the physiological changes that occur during specific YBE. To learn more about the mechanism by which YBE affect human physiology, the stimulus of individual YBE must be quantified.

Chapter 3: Methods

In this Master's thesis two separate studies were carried out. The first study was observational and compared experienced YB with matched controls. The second study was a 6 week YBE training study which compared experimental and control groups.

Study 1

Overview

All experimental procedures were approved by the Clinical Research Ethics Board (#H09-03202) at the University of British Columbia. After signing the informed consent form, baseline anthropometirc and spirometric measures were taken. Next, subjects were tested during two YBE, *bhastrika*, and *chaturbhuj*, which are explained in detail in Study 2, and during two Duffin re-breathe tests. To explain briefly, in *bhastrika*, subjects hyperventilated through their nose for one minute, then inspired to total lung capacity and held their breath as long as they could. In *chaturbhuj*, subjects inspired, then held their breath, then expired, then held their breath out, each for the same amount of time at a rate that they could maintain for 10 minutes. Following 15 minutes of rest, two Duffin re-breathe tests were performed. The Duffin re-breathe tests are explained in detail below.

Subject characteristics

Height (cm), body mass (kg) and age were determined in order to characterize all subjects. These measurements were performed before any testing. Forced expiratory volume in 1 second (FEV_1) was determined using a stand-alone spirometer (Spirolab II;

Medical International Research, Roma, Italy). Nine male subjects (yogic breather (YB), *n*=3; control, *n*=6) free from any history of cardiorespiratory disease consented to participate in the study. Status as a YB required 1 year of 5 times weekly practice of YBE that included breath-holds and low frequency breathing for a minimum of 20 minutes per session. All subjects were non-smokers, and free from all cardiorespiratory diseases or ailments, control subjects were age and BMI matched and new to the practice of YBE. Additional exclusion criteria included travel to altitude above 2300 m within 6 months of testing, experience in breath-hold sports and competitive swimming.

Cardiorespiratory measures

Arterial oxyhaemoglobin saturation (SaO_2) was measured at the finger using photoplethysmography (Model 3740; Ohmeda, Louisville, CO, USA). $P_{ET}O_2$ and $P_{ET}CO_2$, were measured at the mouth and analyzed through calibrated O_2 and CO_2 analyzers (models S-3A/I and CD-3A, respectively; Applied Electrochemistry, Pittsburgh, PA). Inspiratory flow data was collected using a heated pneumotach (Hans-Rudolph HR800; Kansas City, MO, USA), connected to the inspired side of a mouth and nasal breathing facemask (Mirage NV 16709; ResMed, San Diego, CA, USA). Inspiratory flow signals were integrated to determine tidal volume (V_t) and fb, and then multiplied together to obtain minute ventilation (V_E).

Beat-by-beat systolic, diastolic, and mean blood pressure were obtained non-invasively throughout testing using finger pulse photoplethysmography (Finometer; FMS, Arnhem, Netherlands). Before testing with the blood pressure device, a return-to-flow systolic calibration occured and the hydrostatic height sensor system was zeroed.

Beat-by-beat blood pressure was calibrated against an automated blood pressure measurement (BPM-100; VSM Medtech Ltd, Vancouver, Canada) taken from the right arm at the level of the heart every 3 min. During this time, heart rate was obtained from electrocardiographic analysis.

Cerebral blood flow velocity

CBFV measurements were made using a Neurovision transcranial doppler system (TCD) at the middle cerebral artery (Neurovision 500 M; Multigon Industries, Yonkers, NY, USA). A probe was fixed to the zygomatic arch of the subject which directed ultrasound waves at a frequency of 2 Mhz to a depth of 3.5-5.5 cm. CBFV was determined approximately at the midpoint of the middle cerebral artery upstream from the bifurcation to optimize the ultrasound waveform. The ultrasound probe was held in place using a Marc 600 TCD fixation headframe to ensure the validity of the measurements. For each subject, the same depth and gain of the Doppler signal were used in all three testing sessions. Both peak CBFV and mean CBFV were taken and used as indicies of cerebral blood flow. Mean CBFV was calculated using an algorithm which averages CBFV over 3 second intervals.

Duffin re-breathe

The re-breathe protocol used was based on the Read re-breathe (Read, 1967) as modified by Duffin (Mohan & Duffin, 1997; Duffin & Mahamed, 2003). To start each re-breathe, subjects hyperventilated room air through a three-way rebreathing valve (Three-Way Y-Shape™ 4000; Hans-Rudolph, Shawnee, KS, USA) and were coached for

5 minutes to lower their $P_{ET}CO_2$ to between 18 – 25 mmHg. Next, subjects were switched to a plastic re-breathe bag containing 42 mmHg CO_2 and either 50 mmHg or 200mmHg O_2 (for the hypoxic and hyperoxic tests, respectively). Subjects were instructed to take three deep breaths and then relax, and breathe however was comfortable. This procedure slowly raised the subject's $P_{ET}CO_2$ while the re-breathe bag was kept iso-oxic using a gas solenoid valve controlled by a specifically designed computer program (LabVIEW 7.0, National Instruments, Austin, TX). The re-breathe test was terminated when the subject's $P_{ET}CO_2$ reached 60 mmHg, when V_E reached 100 L/min, when the researchers noted strong discomfort by the subject, or when the subject wished to stop.

The same specifically designed software used to collect the data calculated hypercapnic ventilatory threshold and sensitivity. For each subject, this software fitted a straight line to the $P_{ET}CO_2$ vs. time relationship and derived a predicted $P_{ET}CO_2$ for each breath. Next, predicted $P_{ET}CO_2$ was plotted against V_E. Average baseline ventilation, hypercapnic ventilatory threshold, and sensitivity were determined from this plot. Hypercapnic ventilatory threshold was the $P_{ET}CO_2$ above which, there was a rise in V_E for a rise in $P_{ET}CO_2$. Hypercapnic ventilatory sensitivity was the slope of the line after the threshold. In some subjects, there were two distinct slopes and ventilatory thresholds. When this occurred, the first slope was gradual, and the second was steep. The first slope was taken as the hypercapnic ventilatory sensitivity, while the second slope and threshold, if present, were not analyzed as per previously published instructions (Koehle *et al.*, 2007). Increases in ventilation following the first threhold are generally due to increases in tidal volume, while increases after the second ventilatory threshold are

generally due to increasing breathing frequency (Duffin & Mahamed, 2003). I fitted and analyzed all of the Duffin curves and was blinded to each subject's group.

Data and statistical analysis

All data were acquired using an analog-to-digital converter (Powerlab/16SP ML 795; ADInstruments, Colorado Springs, CO, USA) interfaced with a computer. Data were sampled at 200 Hz and stored for subsequent analysis. In addition, a 16-bit analog-to-digital converter (National Instruments, AT-MIO-16XE-50) was used to collect ventilatory data during the Duffin re-breathe tests (Duffin *et al.*, 2000). Hypercapnic ventilatory threshold and sensitivity analysis was completed using the ventilatory data collected by the 16-bit analog-to-digital converter in a specifically designed program (National Instruments, LabVIEW) in accordance with previously published guidelines (Duffin *et al.*, 2000). Parameters were expressed as means ± SD. Differences in the parameters were detected between subjects using independet sample *t*-tests. Relationships among the parameters were derived by simple linear regression analysis. Statistical significance was set at $P < 0.05$.

Study 2

Overview

All experimental procedures and protocols were approved (#H10-02374) by the Clinical Research Ethics Board at the University of British Columbia which conforms to the Declaration of Helsinki. Each subject's first visit to the lab included signing of the

informed consent form, baseline anthropometric and spirometric measures, and a hypoxic Duffin re-breathe test for the purpose of familiarization. In week 2, subjects returned to the lab for baseline measurement of hypoxic and hypercapnic ventilatory sensitivitiy and threshold, and breath-hold time. Weeks 3 and 4 were dedicated to training in introductory YBE. Week 5 was in the lab and included all tests from week 2 and additional measurement of cardiorespiratory variables during two YBE. In weeks 6 to 9, subjects learned 3 intermediate YBE and were instructed to practice them 5 times weekely, 20 minutes per time. Once per week subjects attended a YBE class, reported their progress, and received further technical instruction. Subjects returned to the lab in week 10 for the same testing protocol as week 5. Refer to Figure 1 for a detailed explanation of the study design.

	Week 1: Familiarization	Week 2: Baseline test # 1	Week 3: Class # 1 & intro training	Week 4: Class # 2 & intro training	Week 5: Baseline test # 2	Weeks 6 - 9: YBE training *	Week 10: Post-training test
Description	• Consent form •Anthropometry • Spirometery • Hypoxic Duffin re-breathe test	• Hypoxic and hyperoxic Duffin re-breathe tests to measure: Hypercapnic ventilatory threshold and sensitivity • Breath-hold time	• In Class 1, Subjects learn the FYB, the locks, and the four purifications • On their own, Subjects practice the locks and four purifications, 4 times, 20 minutes per time for a total of 5 times per week	• In Class 2, Subjects receive further instruction in the practices learned in Class 1 • Training at home during week 4 is the same as during week 3	• All the tests from Week 2 • Subjects learn *bhastrika, chaturbhuj,* and *sahit kumbhak* YBE •Cardiovascular, respiratory, and cerebrovascular variables measured during *bhastrika* and *chaturbhuj* YBE	• Once per week classes where subjects practice *bhastrika, chaturbhuj,* and *sahit kumbhak* YBE • On their own, Subjects practice the above YBE 4 times, 20 minutes per time for a total of 5 times per week	•All the tests from Week 2 • Spirometery •Cardiovascular, respiratory, and cerebrovascular variables measured during *bhastrika* and *chaturbhuj* YBE
Experimental group	✔	✔	✔	✔	✔	✔	✔
Control group	✔	✔			✔**		✔***

* Subject Requirements: While practicing at home, subjects will use a metronome for counting during *chaturbhuj* and *sahit kumbhak*, and a stopwatch to record breath hold time during *bhastrika*. Subjects will record the counts used in *chaturbhuj* and *sahit kumbhak*, and their breath-hold time during *bhastrika* each practice session in a provided log book.
** Control group will only perform tests from Week 2
*** Control group will only perform tests from Week 2 and Spirometery

Figure 1. A visual representation of the study design. Week 1 was for familiarization. Weeks 2 and 5 were for baseline tests before and after beginning YBE practice during Weeks 3 and 4. Weeks 6 – 9 were for practice of YBE. Week 10 was for post-testing. Checkmarks for the experimental and control groups indicate which sessions were attended by each group.

Subject characteristics

Seventeen subjects (7 female and 10 male) consented to participate in the study. After providing written informed consent, subjects were randomly assigned into two groups; (n = 9) experimental group and (n = 8) control group. All subjects were new to the practice of YBE and non-smokers. 15 subjects were free from all cardiorespiratory diseases or ailments, 1 subject had asthma, and 1 subject had exercise induced asthma. Additional exclusion criteria included travel to altitude above 2300m within 6 months of

the first visit to the research lab, experience in breath-hold sports and competitive swimming. Height (cm), body mass (kg) and age were determined in order to characterize all subjects. These measurements were be performed before any subsequent testing. Forced expiratory volume in 1 second (FEV_1) was determined using a stand-alone spirometer (Spirolab II; Medical International Research, Roma, Italy).

Cardiorespiratory measures

Cardiorespiratory measures were collected and analyzed in the same way as Study 1.

Cerebral blood flow velocity

CBFV measurements were made in the same manner as Study 1. However, to ensure exact placement of the ultrasound probe in subsequent testing sessions, a trace was made of each subjects head, ear, and crucial facial features in relation to the ultrasound probe. This trace was made by holding a transparency to the left side of the subject's head and tracing the above features with a felt pen. Specific notes were made to ensure correct position and aim of the probe.

Duffin re-breathe

The procedures used for the Duffin re-breathes were the same as those in Study 1.

Breath-holds

Following 10 minutes of baseline data collection, each subject performed three maximal breath-holds. The breath-hold protocol we chose was based on a previously published study (Schagatay *et al.*, 1999) which separated repeated apneas with 2 minutes of rest. Subjects were immediately stopped if they took an uncharacteristically large inspiration or expiration before the breath-hold (Schagatay *et al.*, 1999). Subjects were cued as follows: "At the end of a normal breath out, inhale as deep as you can, and hold in as long as you can." Breath-hold time was calculated from the end of the final inspiration to the beginning of the first expiration. The longest of the three attempts was used in analysis. During both the normal breath-hold and the *bhastrika* breath-hold, subjects were given no encouragement to continue the hold, or information on time. This was done so that there was no external influence on breath-hold time.

Data and statistical analysis

All data were acquired in the same manner as Study 1. Differences in all parameters were detected between subjects, and between tests using 2x3 repeated measures ANOVA and post hoc (Fisher's least significant difference) tests. Relationships among the parameters were derived by simple linear regression analysis. Statistical significance was set at $P < 0.05$.

Yogic breathing exercises

All YBE were taught by this author; an experienced and certified yoga teacher (200-hour certification, 2007; Salt Spring Centre of Yoga, Salt Spring Island, BC). All YBE instructions are from (Hari Dass, 1981; Muktibodhananda, 2006; Saraswati, 2006)

Beginner YBE

Before the subjects were taught the three YBE that were practiced during weeks 6 to 9, they were taught three preliminary practices: the full yogic breath (FYB), the three locks, and the four purifications.

The FYB

The FYB is most simply described as a full, deep breath. First, subjects were instructed to activate the diaphragm with the cue "Inhale into the bottom of the lungs by pushing the stomach out." Next, subjects were instructed to "Continue the inhale into the middle portion of the torso by expanding the lower rib cage." Finally, subjects were instructed to "Fill the top of the lungs by expanding the chest up and out." After a short pause, subjects expired with the instructions "Relax the upper chest, then the ribcage, and finally pull the abdomen in slightly." A common analogy that was used when teaching the FYB was the filling and emptying of a jar (lungs) with water (air). The jar always fills from the bottom to the top, and always empties from the top to the bottom. The FYB was done through the nose.

The three locks

There are three locks that are incorporated into many YBE. First, the root lock is a contraction of the pelvic floor that is applied and held during inspiration and relaxed upon expiration. Second, the throat lock is engaged at the beginning of a breath-hold by bringing the chin down towards the upper chest. The throat lock is released upon expiration at the end of the breath-hold. Third, the stomach lock is engaged at the end of an expiration by bringing the abdomen in towards the spine and up towards the chest. The stomach lock is released upon inspiration.

The four purifications

The four purifications are a set of beginner YBE that develop common breathing patterns that are used in intermediate YBE. In traditional teaching of YBE, the four purifications and the three locks should be practiced regularly for a minimum of 3 months before a student progresses to intermediate YBE (Hari Dass, 1981).

i. Nadi shodhan

This practice is also known as alternate nostril breathing. While closing the right nostril with the right thumb, the subject inspires through the left nostril. Then, the left nostril is closed with the right ring finger and the subject expires through the right nostril. The next inspiration is through the right nostril. The subject then closes the right nostril with their right thumb and expires through the left nostril. That constitutes one round. This practice is done with the FYB.

ii. Kapal bhati

This is a shallow, rapid, repeated breath through the nose. The pattern begins with a forceful expiration from about half of total lung capacity. During the expiration, the abdomen is pulled in towards the spine. At the end of the expiration, the abdomen is relaxed which results in an inhalation. That constitutes one round. Subjects were instructed to keep their chest still during this practice.

iii. Agnisar dhauti

This practice is done while holding the breath out and is most simply described as a repeated "stomach lock." As the breath is held out, the abdomen is brought in towards the spine and up towards the chest and then relaxed. This process is repeated for 10-15 seconds with the breath held out.

iv. Ashvini mudra

This practice is done while holding the breath in and is most simply described as a repeated "root lock." As the breath is held in, the pelvic floor is rapidly contracted and relaxed. This process is repeated for 10-15 seconds.

Intermediate YBE

Bhastrika

Bhastrika, translated as "breath of fire," or "bellows breath," is one of the most common YBE used today. Though *bhastrika* is taught differently among yogic systems,

the basic structure of the practice is the same; one to two minutes of abdominal hyperventilation through the nose followed by an inspiration to total lung capacity and maximal breath-hold. The breathing pattern used in *bhastrika* is different than the FYB described above. *Bhastrika's* abdominal hyperventilation breath is simply the first step of the FYB inspiration (filling only the bottom of the jar) followed by a forceful exhale through the nose where the abdomen is pulled in.

Chaturbhuj

Chaturbhuj is a hypoventilation breathing pattern, done through the nose, that uses the FYB and no locks. Each breath is timed and divided into four equal parts: inspiration, breath-hold, expiration, breath-hold. During this YBE, students started with a count of 6 (minimum *f*b of 2-4 breaths per minute) for 10 consecutive minutes. Each week when the students returned for class, they were encouraged to increase the count if it was comfortable. Many experienced practitioners can tolerate a count of 15-20 (*f*b of 0.75 - 1 breath per minute) for the same duration. In week 5, all subjects performed *chaturbhuj* for ten minutes at a count of 6 (*f*b = 2.5 bpm). In week 10, subjects were instructed to perform chaturbhuj at the highest count (lowest *f*b) they could maintain for 10 minutes.

Sahit kumbhak

Sahit kumbhak is more complicated than *bhastrika* and *chaturbhuj*. It brings together all of the elements that the students learned with the previous YBE. It is an alternate nostril, hypoventilation breathing pattern that uses the FYB, apnea, and the three

locks. Each breath is timed and the inspiration:breath-hold:expiration ratio is 1:4:2. Subjects began by closing the right nostril, and inspiring with a FYB through the left nostril for 4 seconds. During the inspiration, the root lock was applied. Next, the breath was held in for 16 seconds. At the beginning of the breath hold, the throat lock was applied and held for the duration of the breath hold. At the end of the breath hold, the subjects released both locks and began their expiration by closing the left nostril with their ring finger, and expiring through the right nostril for 8 seconds. At the end of the expiration, subjects applied the stomach lock for 1 second and then began the cycle again with a 4 second inspiration through the right nostril. The same as in *nadi shodhan*, each round starts with an inspiration through the left nostril and ends with an expiration through the left nostril (two inspiration and two expirations per round).

Classes

Supervised YBE classes were held twice a week in a quiet classroom in the Osborne building to accommodate the subjects' schedules. Each subject was required to attend one of the two classes per week.

Practice at home

In addition to attending one class per week, subjects were instructed to practice on their own 4 times per week, 20 minutes each time. Additionally, subjects were asked to measure breath-hold time during *bhastrika* and note the counts used during *chaturbhuj* and *sahit kumbhak* each week.

Chapter 4: Results

Study 1

Experienced yogic breathers (YB) and control groups were matched for age (control: 28.8 ± 6.2 years; YB: 30.3 ± 8.5 years), and BMI (control: 24.0 ± 1.5 Kg/m^2; YB: 22.6 ± 1.5 Kg/m^2). YB had been practicing YBE for an average of 4.1 ± 2.5 years for more than 20 minutes per day. Representative physiological changes during *bhastrika* are shown below in Figure 1.

Note the increases in MAP (39.8 %) and MCAv (102 %) throughout the breath-hold. Maximal breath-hold time during *bhastrika* was similar between YB and controls (control: 84.8 ± 21.5 s; YB: 98.0 ± 21.4 s, $P = 0.43$). The levels of $P_{ET}O_2$ (control: 76.3 ± 11.6 mmHg; YB: 78.7 ± 9.4 mmHg) and $P_{ET}CO_2$ (control: 46.7 ± 5.0 mmHg; YB: 45.8 ± 2.7 mmHg) reached at end breath-hold were also similar between groups. However, peak MAP tended to be greater in YB at end breath-hold (control: 108.6 ± 15.9 mmHg; YB: 23.5 ± 7.8 mmHg, $P = 0.10$) (Figure 2A).

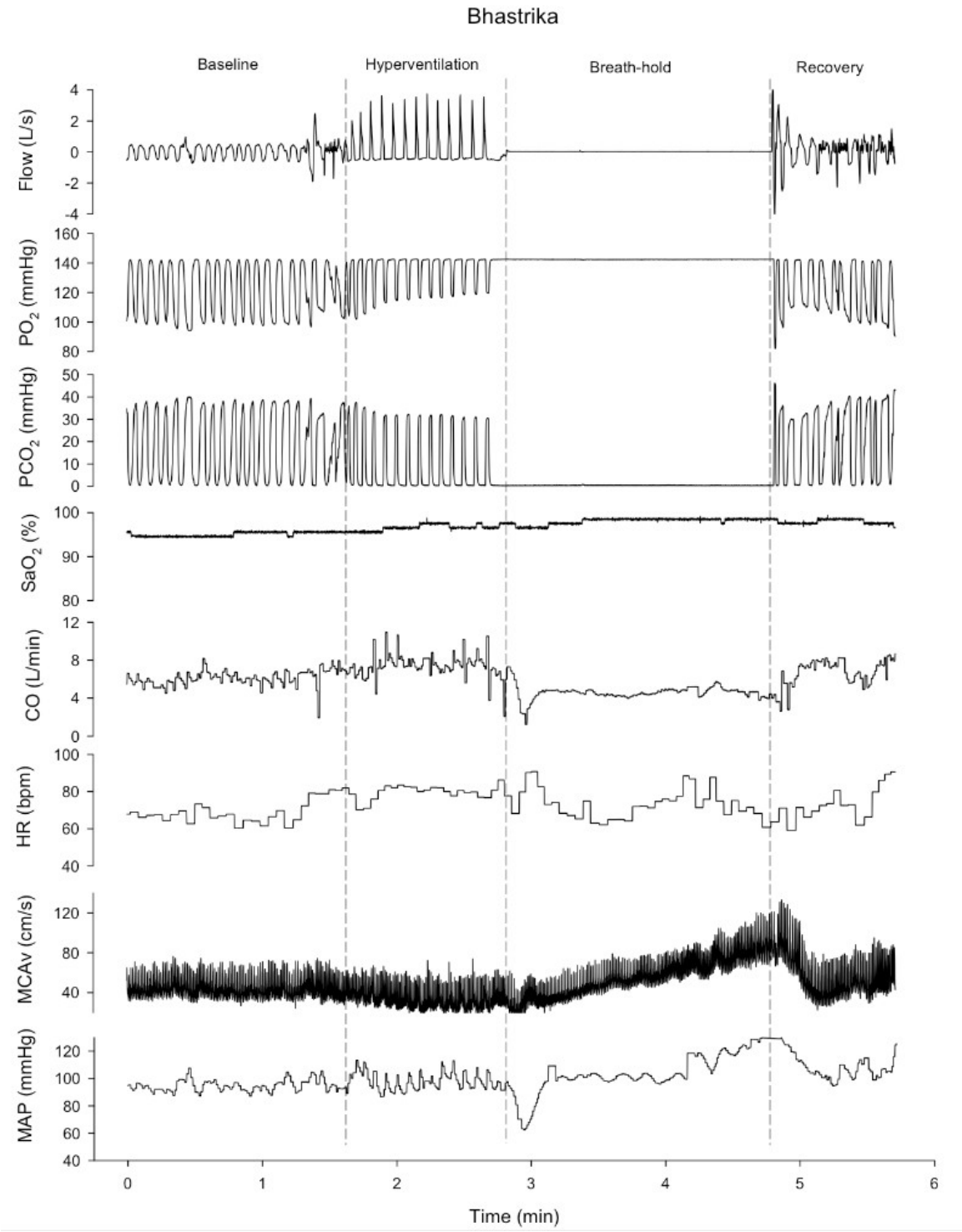

Figure 2. Cardiorespiratory responses to *bhastrika* in an experienced YB.

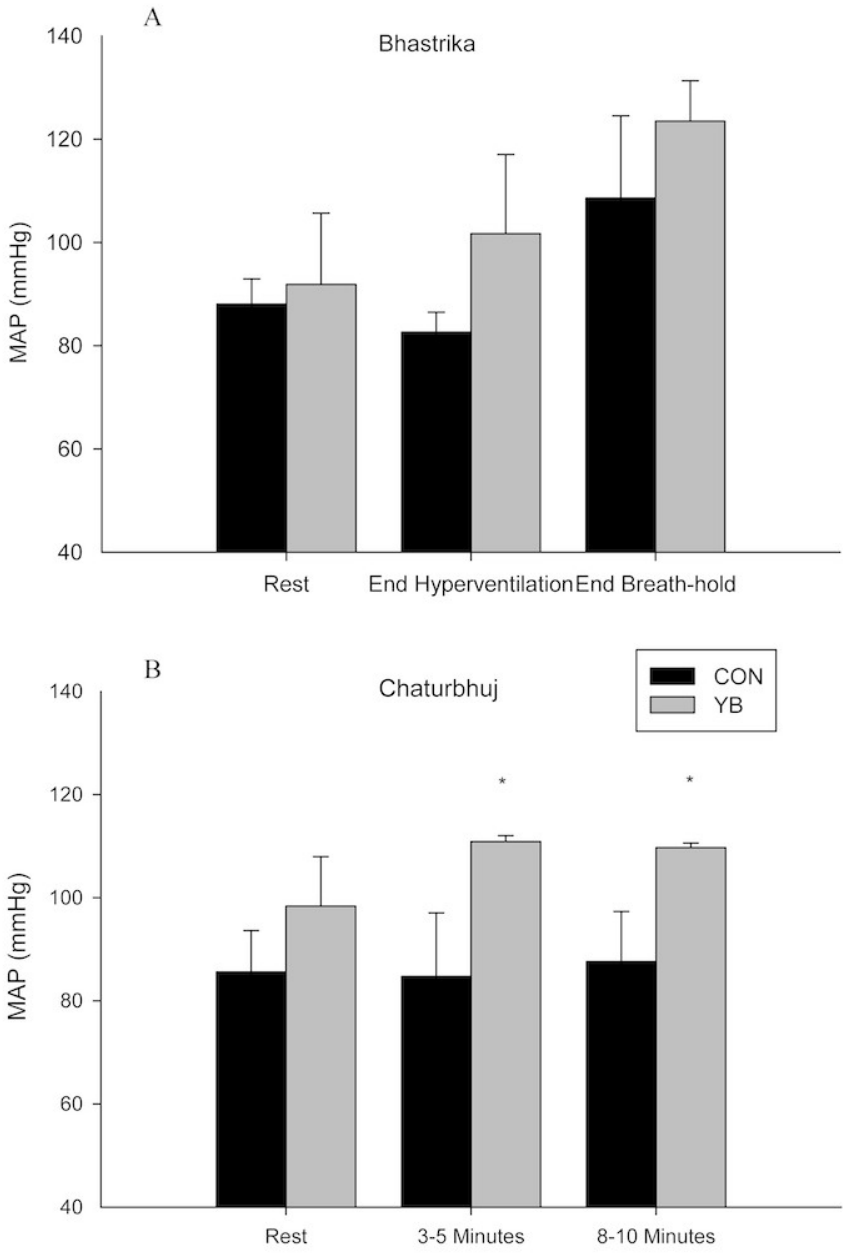

Figure 3. Graphical illustration of the MAP response to *bhastrika* (A) and *chaturbhuj* (B) in YB and CON. Note the increased BP in YB throughout *chaturbhuj*. * $P < 0.05$ compared to control.

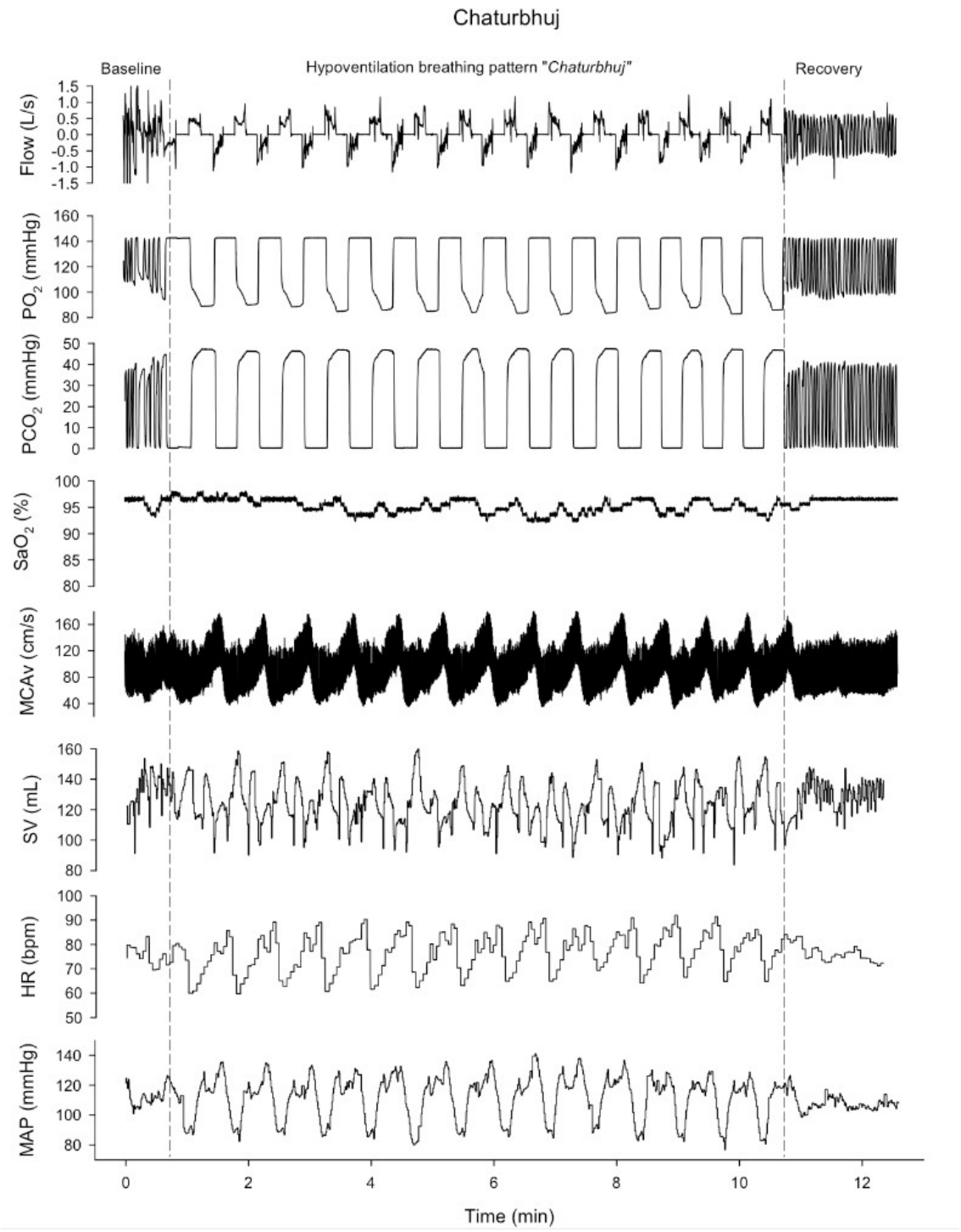

Figure 4. Cardiorespiratory responses to *chaturbhuj* in an experienced YB.

Representative physiological changes during *chaturbhuj* are shown in Figure 3. MAP and MCAv cycled with the phases of respiration in the YB, both decreased with inspiration and increased with expiration. During *chaturbhuj*, YB had lower *f*b (control: 2.7 ± 0.7 bpm; YB: 1.4 ± 0.2 bpm, $P < 0.05$) and higher tidal volume (control: 2.9 ± 0.9 L; YB: 4.1 ± 0.5 L, $P < 0.05$) resulting in slightly lower minute ventilation (control: 7.9 ± 3.3 L/min; YB: 5.9 ± 1.3 L/min, $P = 0.11$). During *chaturbhuj* mean $P_{ET}O_2$ was lower in YB (control: 94.3 ± 12.0 mmHg; YB: 77.5 ± 5.7 mmHg, $P < 0.05$) and $P_{ET}CO_2$ seemed greater, (control: 41.3 ± 8.7 mmHg; YB: 47.3 ± 1.9 mmHg, $P = 0.15$), but not significantly different. MAP was similar at rest between groups (control: 85.6 ± 8.0 mmHg; YB: 98.4 ± 9.6 mmHg $P = 0.13$), however, during *chaturbhuj*, MAP was higher in the YB (control: 87.6 ± 9.8 mmHg; YB: 110.0 ± 0.9 mmHg, $P < 0.05$) (Figure 2B).

The hypercapnic ventilatory threshold was the same between groups in both the hypoxic and hyperoxic tests (hypoxic: control: 43.7 ± 4.4 mmHg; YB: 43.8 ± 1.2 mmHg $P = 0.80$; hyperoxic: control: 48.1 ± 5.7 mmHg; YB: 47.9 ± 1.3 mmHg, $P = 0.70$). Hypercapnic ventilatory sensitivity was lower in the YB only in hyperoxia (Hypoxic: control: 6.6 ± 3.3 L/min/mmHg; YB: 5.3 ± 0.7 L/min/mmHg, $P = 0.85$; Hyperoxic: control: 4.6 ± 1.2 L/min/mmHg; YB: 3.4 ± 0.4 L/min/mmHg, $P < 0.05$).

Study 2

Anthropometric and spirometeric measures were similar between groups at baseline and did not change following the intervention ($P > 0.05$, Table 2). Eight out of nine experimental subjects attended each instructional class, reported completing their practice at home, and completed each testing session. One subject withdrew from the

study before the final testing session due to non-compliance with home practice during the 4 weeks prior to testing. There was no change in spirometric measures following the intervention. BMI was not different between groups. This was important because obesity affects blood pressure and cerebrovascular reactivity to hypoxia. Independent of co-morbidities, obesity is associated with increased cerebrovascular resistance, and reduced CBFV both at rest, and in response to hypoxia (Selim *et al.*, 2008; Dempsey *et al.*, 2010).

Table 2. Anthropometric and Spirometric data (means ± SD) at baseline and following the intervention**.**

	Baseline		6 Weeks	
	Control	**Experimental**	**Control**	**Experimental**
Age, yr	25.1 ± 3.1	23.9 ± 2.5		
Height, cm	176.3 ± 10.7	173.6 ± 7.1		
Weight, Kg	74.3 ± 15.8	70.1 ± 9.7		
BMI, Kg/m^2	23.7 ± 3.1	23.3 ± 2.9		
FVC, L	5.1 ± 1.4	4.9 ± 1.1	4.8 ± 1.6	5.1 ± 0.7
PEF, L/s	9.1 ± 2.0	10.2 ± 2.4	8.8 ± 2.2	9.9 ± 2.4
FEV 1, L	4.2 ± 0.9	4.2 ± 1.0	4.1 ± 1.1	4.2 ± 0.5
FEV 1, %	83.2 ± 7.4	85.5 ± 4.4	87.8 ± 5.9	82.4 ± 2.6

Bhastrika can be broken down into two physiologically different parts. First, the hyperventilation; and second, the breath-hold. As expected, the hyperventilation was achieved primarily by an increase in V_t (rest: 0.81 ± 0.1 L; hyperventilation: 1.7 ± 0.52 L, $P < 0.05$). During the hyperventilation, $P_{ET}O_2$ increased an average of 25.5 ± 8.5 mmHg, and $P_{ET}CO_2$ decreased an average of 10.0 ± 3.5 mmHg. The change in $P_{ET}O_2$ increased SaO_2 (rest: 95.8 ± 0.97 %; hyperventilation: 97.2 ± 0.75 %, $P < 0.05$). In the

hyperventilation portion of *bhastrika*, MCAv decreased, heart rate was slightly elevated, and there was no change in MAP (Table 2).

At end breath-hold, *bhastrika* resulted in a decrease in $P_{ET}O_2$ from 125.6 ± 8.5 mmHg to 90.8 ± 12.1 mmHg (9.3 mmHg lower than rest, $P < 0.05$). However, this decrease did not cause a significant drop in SaO_2. At end breath-hold, $P_{ET}CO_2$ increased from 27.4 ± 3.5 mmHg to 40.6 ± 2.7 mmHg (3.2 mmHg higher than rest, $P < 0.05$). MCAv more than doubled from end hyperventilation to end breath-hold, and MAP and HR were elevated significantly at end breath-hold (Table 3).

Table 3. Cardiorespiratory variables (means ± SD) at rest and during Bhastrika**.**

	Rest	Hyperventilation	Breath-hold
$P_{ET}O_2$ (mmHg)	100.1 ± 7.4	125.6 ± 8.5*	90.8 ± 12.1*
$P_{ET}CO_2$ (mmHg)	37.4 ± 3.6	27.4 ± 3.5*	40.6 ± 2.7*
Inspired V_E (L/min)	11.4 ± 1.9	27.1 ± 7.8*	
SaO_2 (%)	95.8 ± 0.97	97.2 ± 0.75*	93.3 ± 3.9
V_t (L)	0.81 ± 0.1	1.7 ± 0.52*	
***fb* (bpm)**	14.2 ± 1.4	16.4 ± 3.1	
MCAv (cm/s)	55.8 ± 26.3	40.2 ± 18.3*	87.4 ± 23.0*
SV (mL)	90.5 ± 21.4	88.2 ± 19.1	
TPR (CGS)	1332 ± 247	1200 ± 502	
CO (L/min)	5.8 ± 1.3	7.2 ± 2.6	
SBP (mmHg)	110.1 ± 9.5	112.2 ± 15.1	124.0 ± 16.6*
DBP (mmHg)	71.1 ± 7.9	70.9 ± 10.9	83.2 ± 15.4*
MAP (mmHg)	83.0 ± 6.6	84.4 ± 11.6	96.7 ± 13.0*
HR (bpm)	65.3 ± 9.8	74.5 ± 9.5*	80.8 ± 15.1*

* Different compared to resting values ($P < 0.05$)

Chaturbhuj was performed for 10 minutes by each experimental subject. Though *f*b was reduced (rest: 14.2 ± 1.4 bpm; *chaturbhuj*: 2.6 ± 0.16 bpm, , $P < 0.05$), mean values from week 5 show that SaO_2 did not change during *chaturbhuj*. $P_{ET}CO_2$ increased

2.3 mmHg ($P < 0.05$), and HR increased 2.9 bpm ($P < 0.05$) (Table 4). However, these mean values do no illustrate the cyclical changes shown in the individual traces (*bhastrika*: Figures 5 – 7; *chaturbhuj*: Figures 8 – 10).

Table 4. Cardiorespiratory variables (means ± SD) at rest and during Chaturbhuj.

	Rest	Chaturbhuj
$P_{ET}O_2$ (mmHg)	100.1 ± 7.4	99.8 ± 5.3
$P_{ET}CO_2$ (mmHg)	37.4 ± 3.6	39.7 ± 2.7*
Inspired V_E (L/min)	11.4 ± 1.9	6.6 ± 1.4*
SaO_2 (%)	95.8 ± 1.0	95.5 ± 1.2
V_t (L)	0.81 ± 0.1	2.6 ± 0.6*
***f*b (bpm)**	14.2 ± 1.4	2.6 ± 0.16*
MCAv (cm/s)	55.8 ± 26.3	51.6 ± 25.4
SV (mL)	90.5 ± 21.4	85.1 ± 14.6
TPR (CGS)	1332 ± 247	1345 ± 226
CO (L/min)	5.8 ± 1.3	5.7 ± 1.1
SBP (mmHg)	110.1 ± 9.5	107.4 ± 13.2
DBP (mmHg)	71.1 ± 7.9	71.6 ± 9.2
MAP (mmHg)	83.0 ± 6.6	85.2 ± 10.8
HR (bpm)	65.3 ± 9.8	68.2 ± 8.0*

* Different compared to resting values ($P < 0.05$).

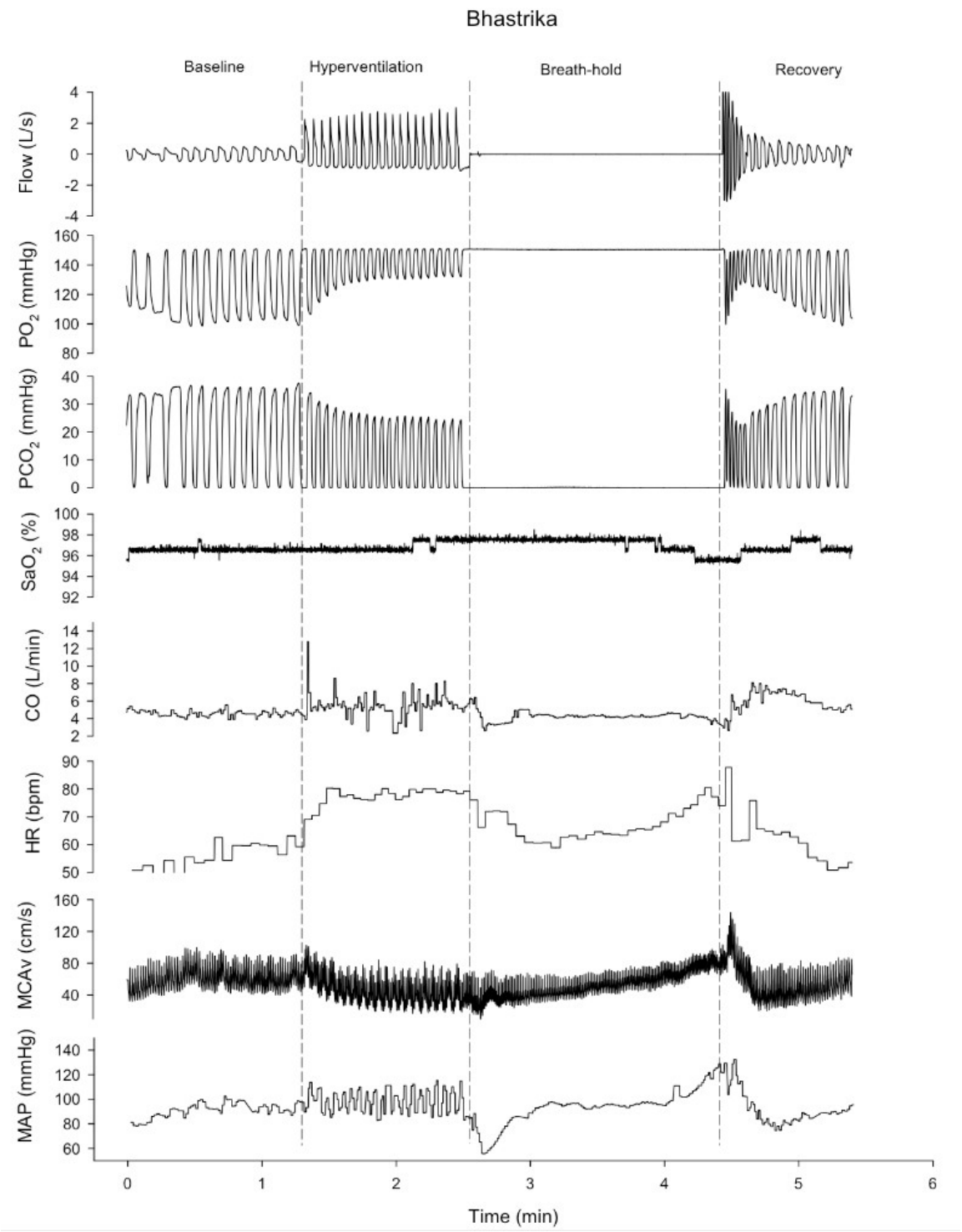

Figure 5. Cardiorespiratory, cerebrovascular, and blood pressure responses to *bhastrika* in an experimental subject. Notice the similar increases in MCAv and MAP during the breath-hold.

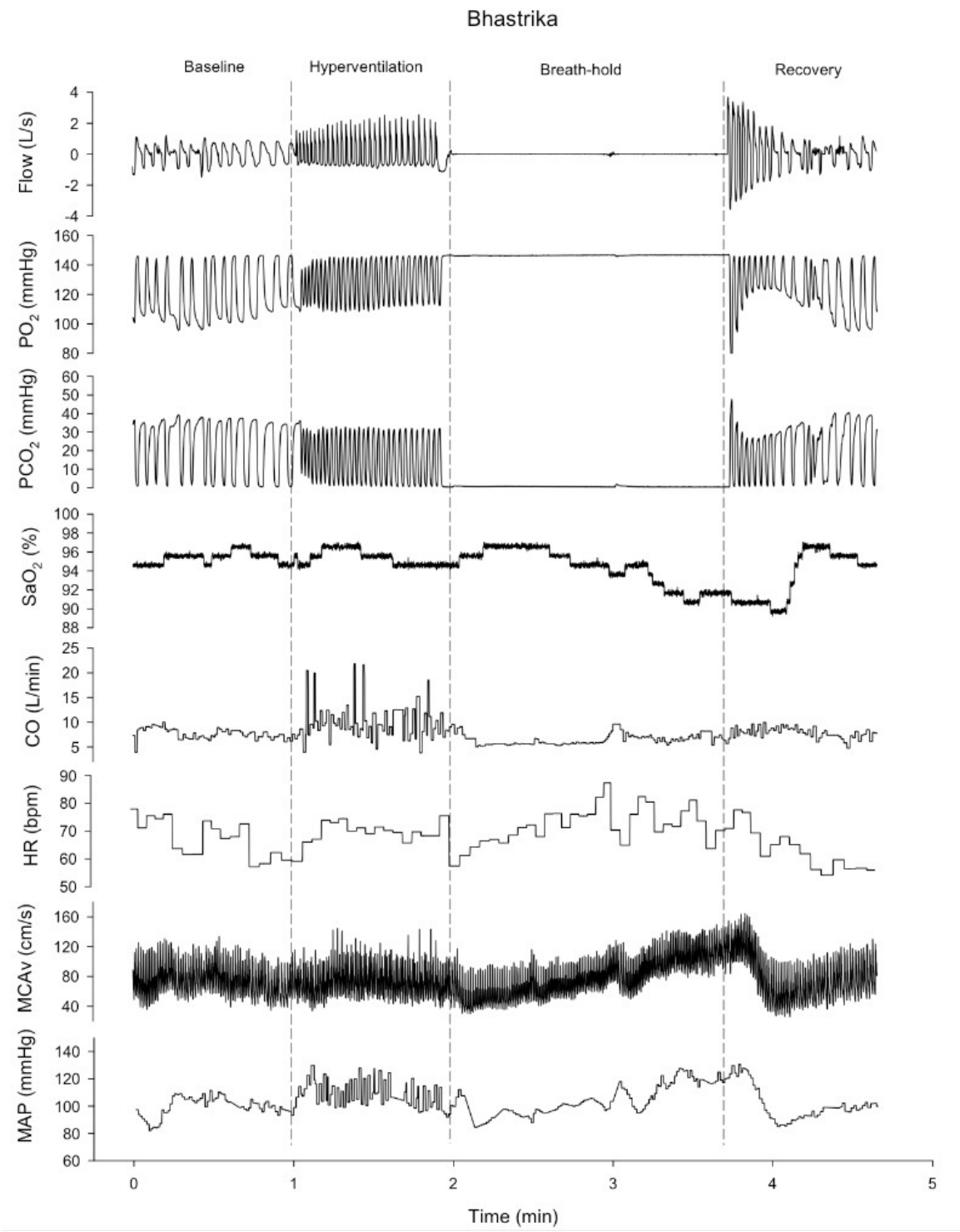

Figure 6. Cardiorespiratory, cerebrovascular, and blood pressure responses to *bhastrika* in an experimental subject. Notice the similar increases in MCAv and MAP during the breath-hold.

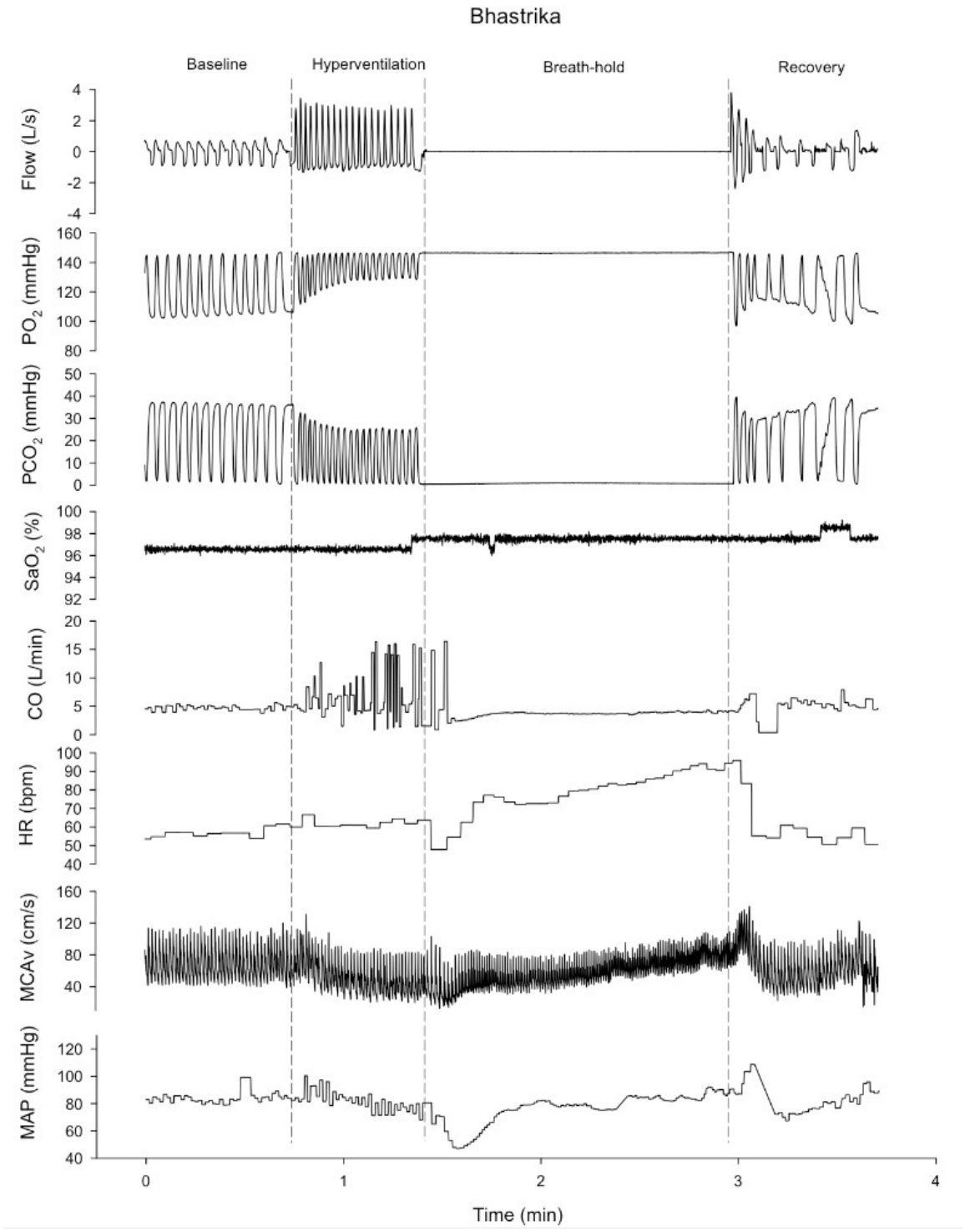

Figure 7. Cardiorespiratory, cerebrovascular, and blood pressure responses to *bhastrika* in an experimental subject. Notice the similar increases in MCAv and MAP during the breath-hold.

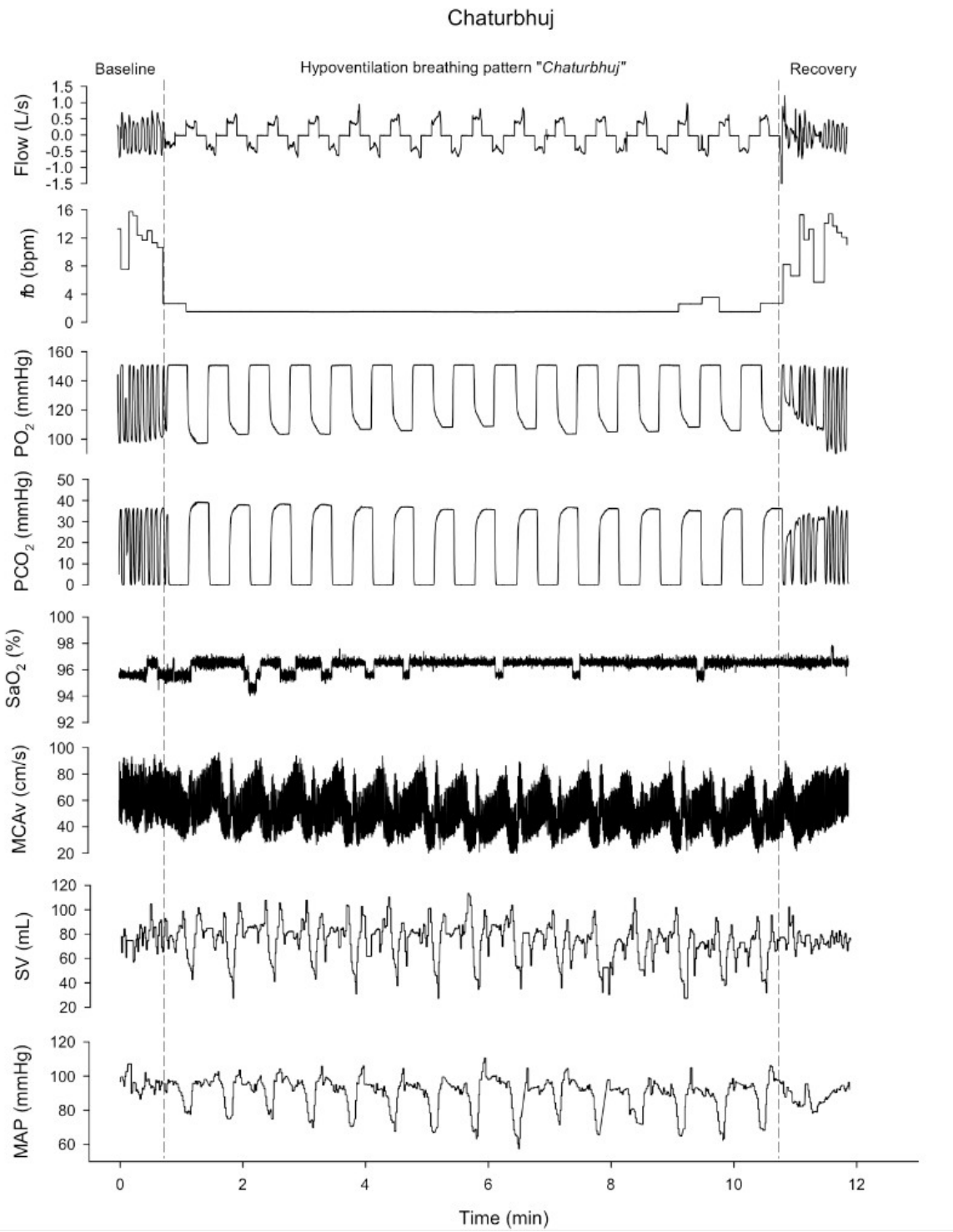

Figure 8. Cardiorespiratory, cerebrovascular, and blood pressure responses to *chaturbhuj* in an experimental subject. Note how SaO_2, MCAv, SV, and MAP oscillate with ventilation**.**

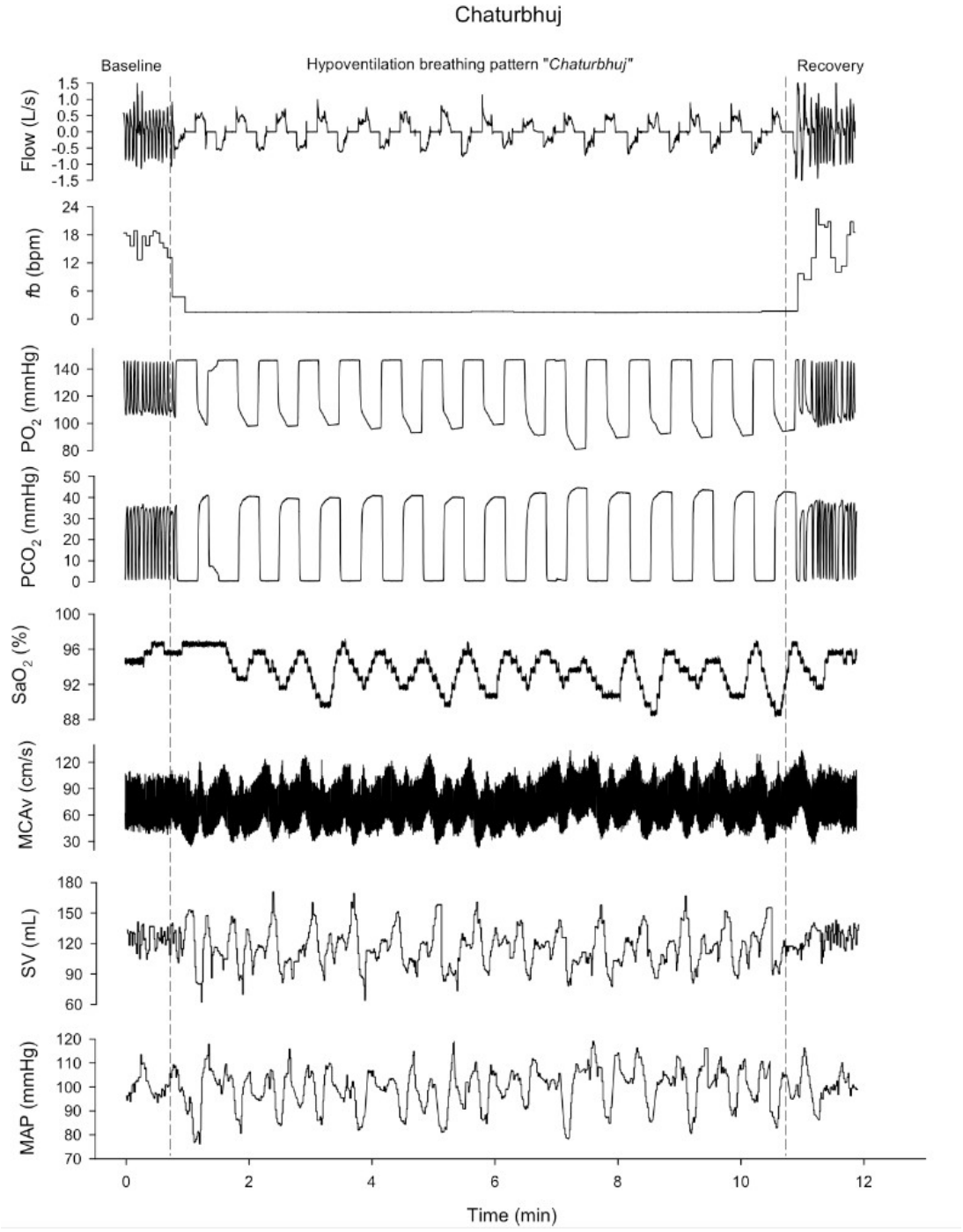

Figure 9. Cardiorespiratory, cerebrovascular, and blood pressure responses to *chaturbhuj* in an experimental subject. Note how SaO_2, MCAv, SV, and MAP oscillate with ventilation**.**

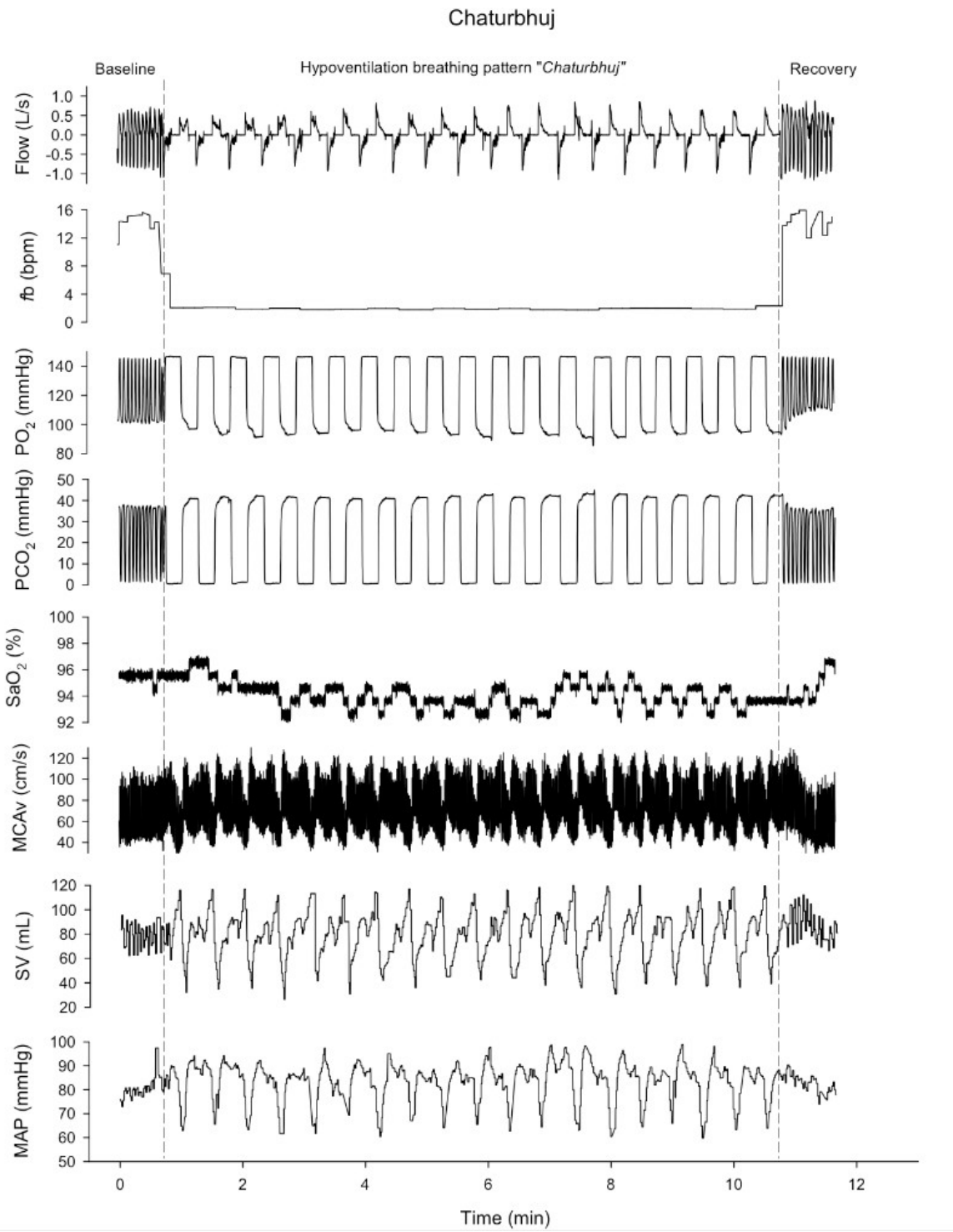

Figure 10. Cardiorespiratory, cerebrovascular, and blood pressure responses to *chaturbhuj* in an experimental subject. Note how SaO_2, MCAv, SV, and MAP oscillate with ventilation**.**

Cardiorespiratory data were collected during *bhastrika* and *chaturbhuj* at two time points. The first collection period was in week 5, after the subjects had practiced the beginner YBE for two weeks. The second collection period was in week 10, after the subjects had practiced the intermediate YBE for four weeks (Figure 1). In the *bhastrika* hyperventilation, there were no changes in any cardiorespiratory variables from week 5 to week 10. In the *bhastrika* breath-hold, there was no change in breath-hold time (Week 5: 90.6 ± 33.6 s; Week 10: 104.7 ± 9.4 s, $P = 0.48$), however, there was a change in SaO_2 at end breath-hold (Week 5: 91.4 ± 3.9 %; Week 10: 94.7 ± 3.1 %, $P < 0.05$). There were no other differences to report between weeks 5 and 10 during *bhastrika*.

Baseline ventilation (L/min) during the Duffin re-breathe tests was measured following the 5 minutes of hyperventilation and three equilibration breaths from the re-breathing bag, and ended at each subject's hypercapnic ventilatory threshold. In the hypoxic Duffin re-breathe, there were no differences in baseline ventilation between time points or between groups, however, there was a time*group interaction ($P < 0.05$, Figure 10). In the hyperoxic Duffin re-breathe there was a difference between time points ($P < 0.05$), and a time*group interaction ($P < 0.05$, Figure 12), however, there was no difference between groups.

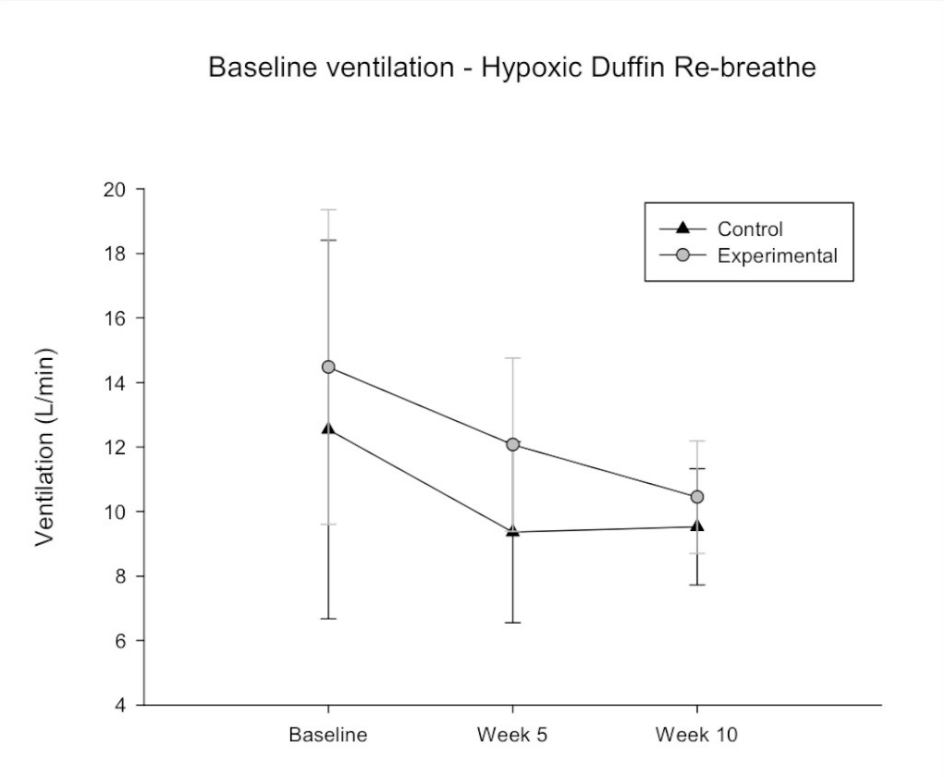

Figure 11. Mean baseline V_E (L/min) ± SD for control and experimental groups at the beginning of the hypoxic Duffin re-breathe test. This data was averaged following the 5 minutes of hyperventilation and 3 equilibration breaths from the re-breathing bag, until the hypercapnic ventilatory threshold. Notice the trend in both figures of decreased V_E with each successive test in the experimental group.

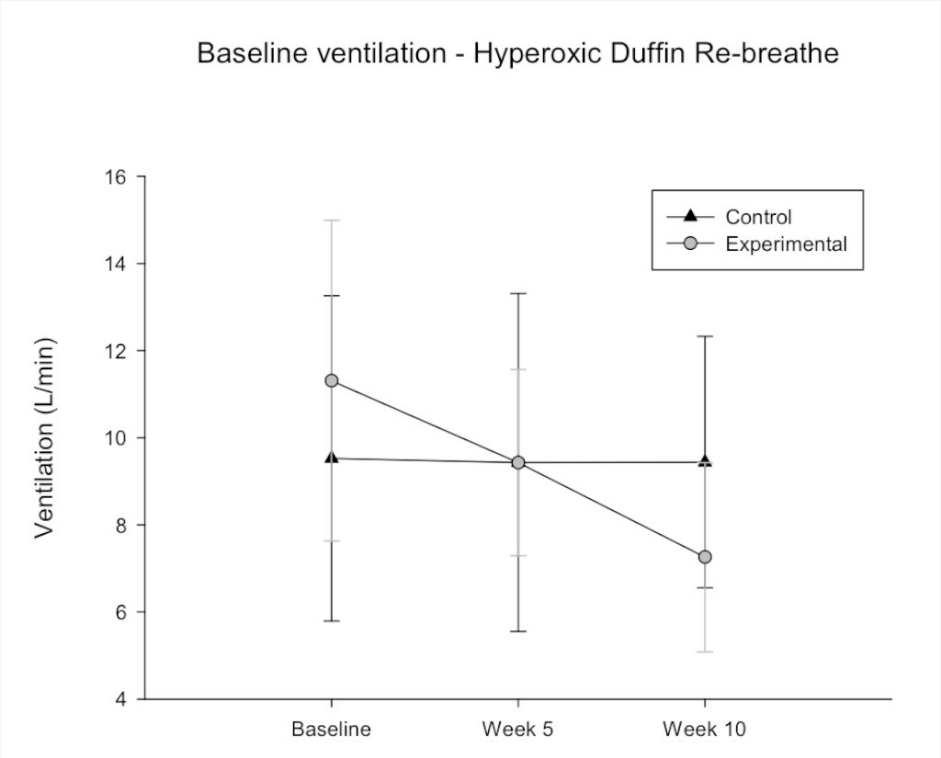

Figure 12. Mean baseline V_E (L/min) ± SD for control and experimental groups at the beginning of the hyperoxic Duffin re-breathe test. This data was averaged following the 5 minutes of hyperventilation and 3 equilibration breaths from the re-breathing bag, until the hypercapnic ventilatory threshold. Notice the trend in both figures of decreased V_E with each successive test in the experimental group.

The hypercapnic ventilatory threshold (Hypoxic: 40.7 ± 2.25 mmHg; Hyperoxic: 43.5 ± 2.23 mmHg, $P < 0.01$) and sensitivity (Hypoxic: 5.64 ± 2.33 L/min/mmHg; Hyperoxic: 3.61 ± 1.46 L/min/mmHg, $P < 0.01$) were different between the hypoxic and hyperoxic Duffin re-breathe tests (Figures 13,14). There was no difference in hypercapnic ventilatory threshold or sensitivity between groups or between time points.

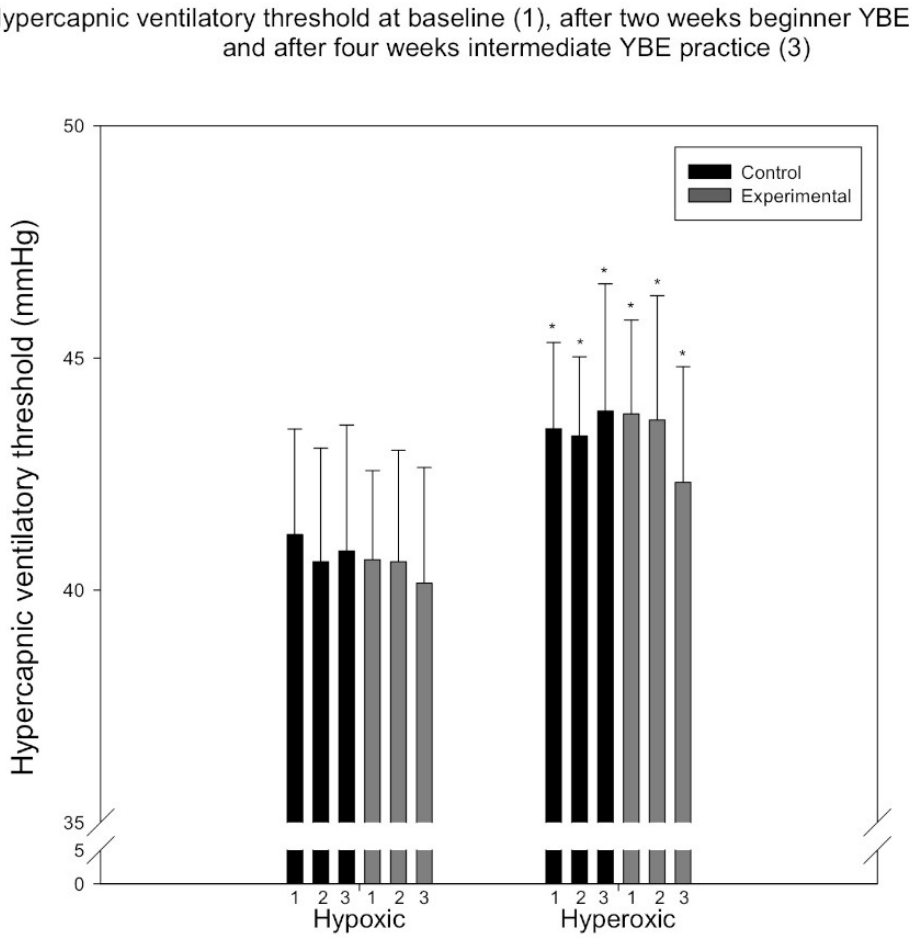

Figure 13. * Different from hypoxic test $P < 0.05$. Hypercapnic ventilatory threshold in hyperoxia was significantly lower than in hypoxia. There was no difference between groups or between time points.

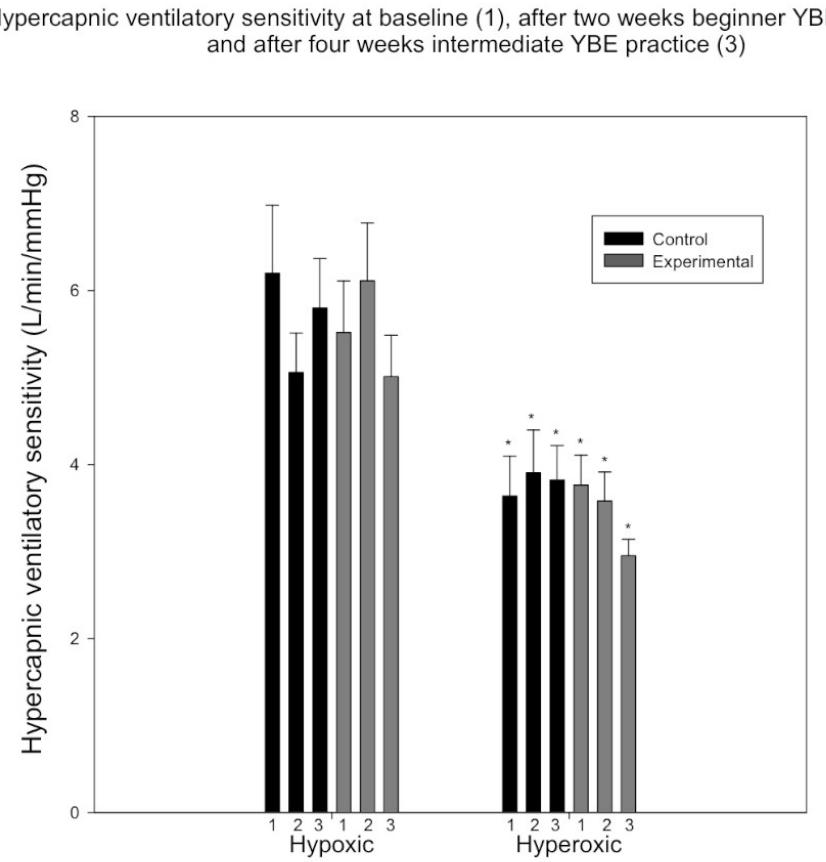

Figure 14. * Different from hypoxic test P < 0.05. Hypercapnic ventilatory sensitivity in hyperoxia was significantly lower than in hypoxia. There was no difference between groups or between time points.

Normal breath-hold time between groups was the same at baseline (control: 58.8 ± 17.3 s; experimental: 65.9 ± 14.9 s, $P = 0.38$) and in week 10 (control: 75.0 ± 14.3 s; experimental: 81.3 ± 8.9 s, $P = 0.54$). Comparing the normal breath-hold with the *bhastrika* breath-hold in week 5, we found that breath-hold time was not different (normal: 71.0 ± 25.4 s; *bhastrika*: 84.6 ± 24.8 s, $P = 0.07$), however, $P_{ET}CO_2$ at end breath-hold was different (normal: 45.7 ± 3.9 mmHg; *bhastrika*: 40.6 ± 2.7 mmHg, $P < 0.01$). After 4 weeks of intermediate YBE practice, in week 10, breath-hold time was longer in *bhastrika* (normal: 81.3 ± 8.9 s; bhastrika: 104.7 ± 9.4 s, $P < 0.01$), but there

was no difference in $P_{ET}CO_2$ (normal: 43.0 ± 3.7 mmHg; *bhastrika*: 40.9 ± 6.2 mmHg, $P = 0.08$).

There was no difference in the MAP or MCAv response when comparing the *bhastrika* breath-hold to the normal breath-hold. There was no significant correlation between change in breath-hold time and change in hypercapnic ventilatory sensitivity in either oxygen environment (hypoxia: $r = 0.26$, $P = 0.57$; hyperoxia: $r = 0.08$, $P = 0.87$). The response of MCAv to hypercapnia in both Duffin re-breathe environments was not different between groups, and did not change following either part of the intervention (Figure 15).

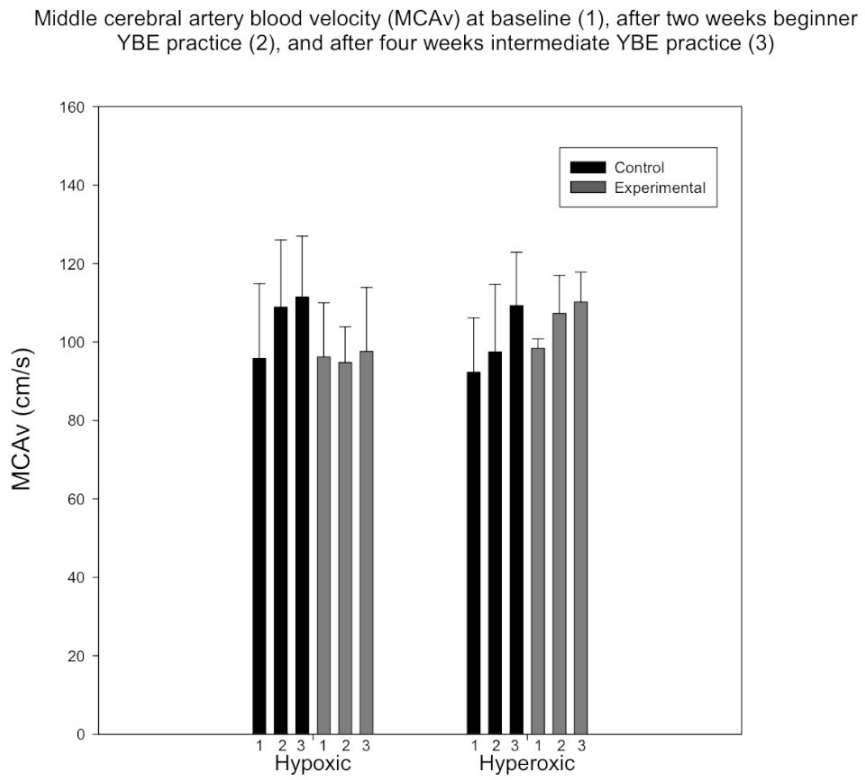

Figure 15. Average MCAv (cm/s) ± SD during the last 15 seconds of the Duffin re-breathe tests. There was no difference between tests, groups, or time points.

The MAP responses to *bhastrika* (week 5: 109.6 ± 4.8 mmHg; week 10: 96.7 ± 18.9 mmHg, $P = 0.40$) and *chaturbhuj* (week 5: 88.4 ± 8.2 mmHg; week 10: 83.4 ± 9.4 mmHg, $P = 0.62$) were not different following 4 weeks of intermediate YBE practice (Figure 16).

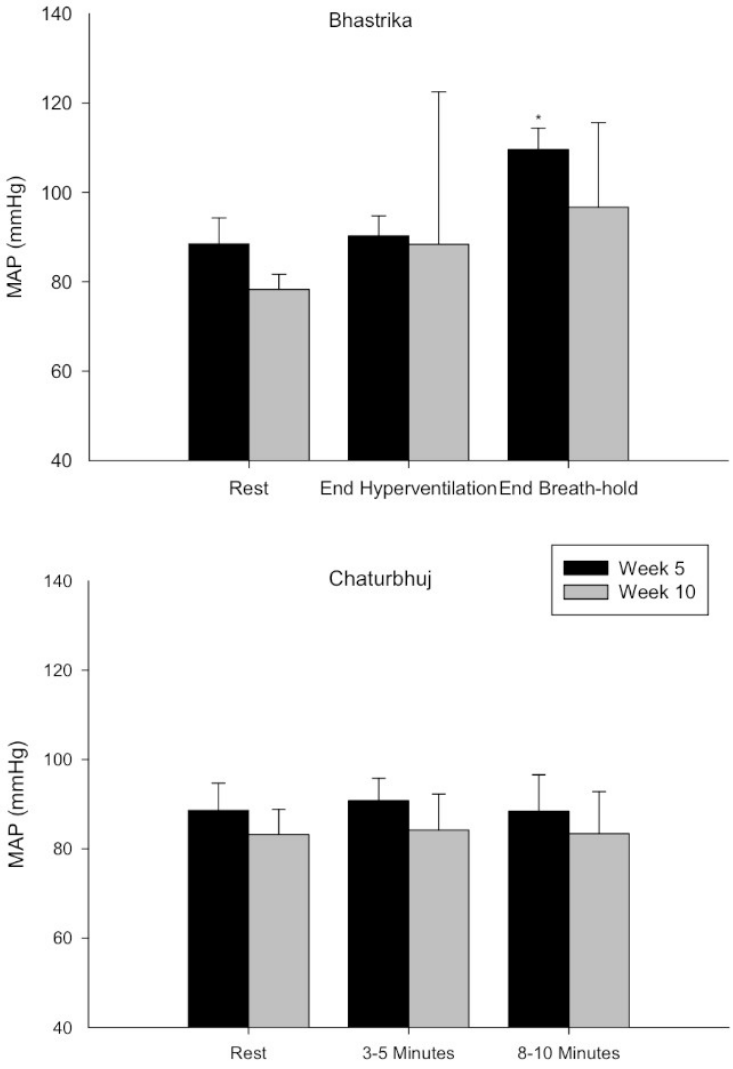

Figure 16. * Different from rest $P < 0.05$. Average MAP (± SD) response to *bhastrika* and *chaturbhuj* in weeks 5 and 10. Notice in *bhastrika* that the MAP at end breath-hold is higher than at rest in week 5. There was no difference in the MAP response between weeks 5 and 10 in either *bhastrika* or *chaturbhuj*.

Chapter 5: Discussion

Study 1

In this study, experienced YB and inexperienced matched controls performed two YBE and two Duffin re-breathe tests. The results support three major findings. First, *bhastrika* produced a mild hypoxic and hypercapnic stimulus similarly in both YB and controls, whereas *chaturbhuj* produced hypoxia and hypercapnia to a greater degree in the YB. Second, contrary to published literature showing a decrease in MAP during a hypoventilation YBE (Pramanik *et al.*, 2009), it was demonstrated that MAP increases in response to *chaturbhuj*, and this increase in MAP was higher in the YB compared with controls. Finally, a decreased hypercapnic ventilatory sensitivity in hyperoxia in the YB was observed. This is consistent with the idea that a central drive to breathe is decreased following long-term practice of YBE.

During apnea, the peripheral chemoreflex is activated and the cardiovascular system works to conserve oxygen for the brain and the heart. In addition to increasing ventilation, the peripheral chemoreflex results in increased MAP due to sympathetic activation (Dujic *et al.*, 2008). In *chaturbhuj*, there was a trend for higher MAP in the YB. *Chaturbhuj* is considered an intermediate difficulty YBE and is progressive in nature. With practice, YB aim to decrease the *f*b used during *chaturbhuj*, resulting in a more challenging YBE. The YB had lower $P_{ET}O_2$ and higher $P_{ET}CO_2$ than the control group because they performed *chaturbhuj* with lower *f*b and consequently, lower V_E. During *chaturbhuj*, the result of a lower *f*b is more time spent per breath in both internal and external apnea. More apnea results in increased $P_{ET}CO_2$ and decreased $P_{ET}O_2$, and more severe hypercapnic hypoxia results in increased sympathetic outflow (Breskovic *et*

al., 2010). Though sympathetic nerve activity was not measured, this is a probable explanation for the increased MAP during *chaturbhuj*.

This study showed no difference in peripheral chemosensitivity between groups. This finding is in contrast with a previous that found decreased peripheral chemosensitivity in YB compared to matched controls (Spicuzza *et al.*, 2000). However, differences between studies may be attributed to methodological assessment of chemosensitivity. In this study two re-breathe conditions ($P_{ET}O_2$ = 45 mmHg; hypoxic, and $P_{ET}O_2$ = 145 mmHg; hyperoxic) were employed to assess the peripheral and central contribution to ventilation, whereas the cited study used a single hypoxic-normocapnic re-breathe (Spicuzza *et al.*, 2000). The hypoxic-normocapnic re-breathe only measures peripheral chemosensitivity to O_2, whereas the hypoxic Duffin re-breathe assesses peripheral chemosensitivity with the contribution of both low O_2 and high CO_2. These are two different responses and explain the difference between the results of this thesis and the cited study. The hyperoxic re-breathe decreases the contribution of the peripheral chemoreceptors, resulting in a centrally based ventilatory response (Duffin & Mahamed, 2003); whereas the hypoxic re-breathe test includes contribution from both chemoreflexes. Hypercapnic ventilatory sensitivity in hyperoxia was lower in the YB, indicating a decreased central chemoresponse. This result is consistent with previous research examining YB (Spicuzza *et al.*, 2000), and other populations that regularly experience hypoxic hypercapnia (breath-hold divers (Dujic *et al.*, 2008); sleep apnea patients (Dempsey *et al.*, 2010)).

In this study, it was hypothesized that YB would have reduced hypercapnic chemosensitivity compared to matched controls. Specifically, reduced hypercapnic

ventilatory sensitivity and increased hypercapnic ventilatory threshold. The data supported a reduced hypercapnic ventilatory sensitivity in YB, however, only in the hyperoxic re-breathe. This was the first study to use the Duffin re-breathe and measure hypercapnic ventilatory threshold in YB. Though there was no difference between groups in hypercapnic ventilatory threshold, the sample size was very low.

The increased MAP response to *chaturbhuj* in the YB compared to controls was not expected. Many studies report that YBE are beneficial for cardiovascular health and reduce blood pressure (Bernardi *et al.*, 2001; Pramanik *et al.*, 2009; Pramanik *et al.*, 2010). However, these studies only include YBE that are variations on the full yogic breath, or in other words: slow, deep breathing. Most intermediate YBE include significant breath-holds and reductions in V_E (Muktibodhananda, 2006). *Bhastrika* and *chaturbhuj* are in this category and according to the data presented, it appears that they significantly increase MAP during their practice.

Study 2

Summary

In this study, 17 subjects new to YBE were randomly assigned to control and experimental groups. Experimental subjects were trained for 2 weeks in beginner YBE and then for 4 weeks in intermediate YBE. This study supported five major findings. First, it was confirmed that *bhastrika* and *chaturbhuj* both produce hypercapnia and mild hypoxia. Second, performance of *bhastrika* results in significantly increased MAP and MCAv. Third, performance of *chaturbhuj* results in cyclic oscillation of MAP, HR, SV, and MCAv with the phases of respiration. Fourth, following the intervention, there was

no difference in normal breath-hold time between groups. Fifth, contrary to the hypothesis, post-intervention, there were no differences between groups in cardiovascular, cerebrovascular, or chemosensitivity measures.

Control of breathing and YBE

The physiological effects of YBE cannot be explained without considering ventilatory control. Originally, it was thought that ventilation was controlled by two separate systems – the peripheral and central chemoreflexes – which responded independently to O_2 and CO_2 respectively. It is now evident that the chemoreflex systems are mutually dependent (Duffin & Mahamed, 2003; Day & Wilson, 2007). As a whole, the human chemoreflex system is made up of three parts: sensors, effectors, and the central controller. The peripheral chemoreceptors are located at the bifurcation of the carotid artery in the carotid body, and in the aortic arch. Their primary job is to measure the amount of O_2 in arterial blood. Animal denervation studies have shown that the hypoxic ventilatory drive originates solely from the carotid chemoreceptors (Izumizaki *et al.*, 2004). Recent work has shown that the peripheral chemoreceptors also contribute approximately one third of the total hypercapnic ventilatory response while the central chemoreceptors contribute the other two thirds (Forster *et al.*, 2008). The peripheral chemoreceptors relay information through the 9th cranial nerve to the respiratory centres in the medulla for integration (West, 2005).

The central chemoreceptors are surrounded by the brain extracellular fluid and are anatomically located just below the ventral surface of the medulla in the brainstem, adjacent to the cerebrospinal fluid (CSF). They are sensitive only to CO_2 through

changes in H^+ concentration. As blood levels of CO_2 rise, CO_2 diffuses through the blood brain barrier and brain extracellular fluid, into the CSF causing increased H^+ concentration and decreased pH in the CSF. The central chemoreceptors respond to decreased pH with increased firing (West, 2005).

There are two systems of "higher control," the brainstem and the cortex. The neurons in the brainstem are responsible for the involuntary, periodic nature of inspiration and expiration. Based on information from the chemoreceptors, they can increase or decrease their firing rate through the phrenic nerve to regulate ventilation. To a point, signals from the cortex can override the brainstem to produce voluntary changes in ventilation such as hyper/hypoventilation and prolonged apnea (West, 2005) (Ferretti, 2001). Voluntary control is where YBE come in.

Bhastrika and *chaturbhuj* are involved breathing exercises that significantly affect cardiorespiratory and cerebrovascular variables. Performance of *Bhastrika* produces hypocapnia and hyperoxia and then hypercapnia and mild hypoxia. When performing *bhastrika* regularly, the goal is to increase breath-hold time. In a breath-hold, there are two breaking-points: the physiological breaking point, and termination of the breath-hold (Lin *et al.*, 1974), which may be more psychological than physiological. The physiological breaking point occurs when the subject begins to make involuntary ventilatory movements and is determined primarily by increasing CO_2 (Hentsch & Ulmer, 1984). From this point, continuation of the breath-hold requires increasing inhibitory input from the cortex (Lin *et al.*, 1974).

Breath-hold time in *bhastrika* was longer than in the normal breath-hold. However, at and breath-hold, $P_{ET}CO_2$ was lower in *bhastrika*. $P_{ET}O_2$ was not different

between holds. Increased breath-hold time in *bhastrika* was expected due to prior hyperventilation and blow-off of CO_2. However, it was not expected that $P_{ET}CO_2$ levels would differ between the two breath-holds. If CO_2 levels were the only variable to influence the breath-hold breaking points, then we would expect to see equal $P_{ET}CO_2$ levels at end breath-hold in both tests. In *bhastrika*, this would have resulted in a further increased breath-hold time. However, there are more variables to consider (Figure 16).

Pulmonary stretch receptors may play a role in regulating breath-hold time and with maximal inspirations, they are sure to be activated. Theoretically, increased activation of pulmonary stretch receptors would result in increased output to central command to end the breath-hold. However, increased lung volume provides more gas for exchange and has a much larger affect on breath-hold time. This is why elite breath-hold divers employ techniques like glossopharyngeal insufflation to further increase lung volume past their inspiratory maximum (Chung *et al.*, 2010). As lung volume increases, so does breath-hold time (Andersson & Schagatay, 1998).

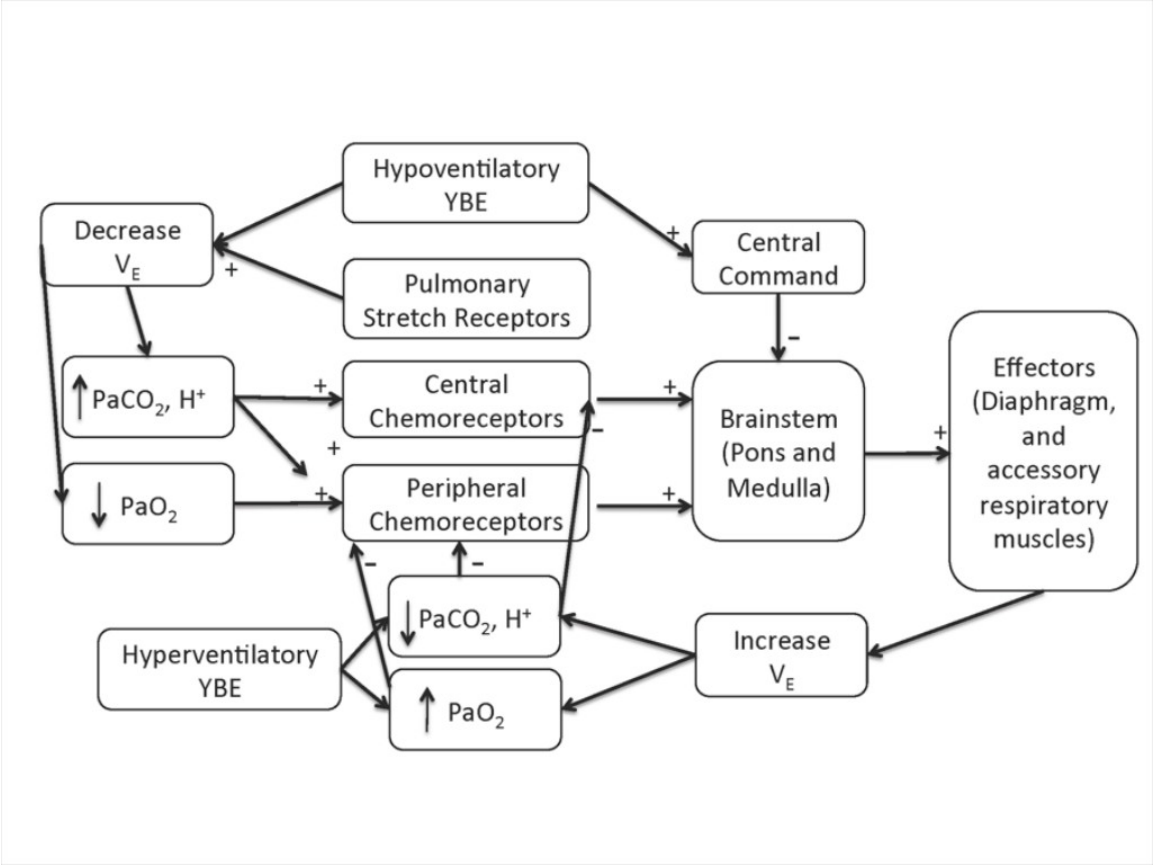

Figure 17. A model of the ventilatory response to hypoxia/hypercapnia and how the practice of YBE may influence their feedback loops. For the purposes of this figure, the YBE considered are those that increase $PaCO_2$ and decrease PaO_2. Good examples are *bhastrika* and *chaturbhuj*. As the rise in $PaCO_2$ and fall in PaO_2 are sensed by the central and peripheral chemoreceptors, the chemoreceptors increase their output to the brainstem. Approximately 2/3 of the hypercapnic drive to breathe comes from the central chemoreceptors, and 1/3 from the peripheral chemoreceptors. 100% of the hypoxic drive to breathe comes from the peripheral chemoreceptors. Normally, this increased drive to breathe results in firing of the phrenic nerve and an increase in ventilation. However, during a breath-hold or a hypoventilation YBE, central command overrides the drive to breathe and the breath-hold or pattern breath is continued, causing a further rise in $PaCO_2$ and fall in PaO_2. This pattern continues until the drive to breathe becomes greater than the inhibitory inputs from central command. At this point, the breath-hold or pattern breath is terminated and ventilation increases.

During the intermediate YBE training phase of the study (weeks 6 – 9), students were instructed to reduce the *f*b used during *Chaturbhuj* each week if it was comfortable.

Mean data shown (Table 3) are from week 5. In week 5, every subject performed *chaturbhuj* at 2.5 bpm. This is most likely why the changes in $P_{ET}O_2$ and $P_{ET}CO_2$ were smaller than those seen in Study 1. In the available week 10 data, levels of $P_{ET}O_2$ and $P_{ET}CO_2$ in subjects who reduced their *f*b are similar to the levels achieved by the experienced YB in Study 1. To help compensate for the large decrease in *f*b during *chaturbhuj*, V_t increased from resting values. However, this increase in V_t was not sufficient to maintain V_E at resting levels and the result was increased $P_{ET}CO_2$.

Cardiovascular control and YBE

Performance of *Chaturbhuj* resulted in the cyclic oscillation of SV, HR, CO (not shown), MCAv, and MAP with the phases of respiration (Figures 4, 7-10). Another study (Shannahoff-Khalsa *et al.*, 2004) examined the hemodynamic effects of a 1 breath per minute YBE where each breath was broken up into three equal parts: 20 seconds of inspiration, 20 second breath-hold, and 20 seconds of expiration. Similarly, the cited study noted oscillations of MAP, HR, and SV in response to inspiration and expiration during the YBE. The oscillation of MAP seen in this study, and the study by Shannahoff-Khalsa *et al.* (2004), can be compared to MAP oscillations seen in sleep apnea patients (Figure 18). However, this is a challenging comparison to make.

YBE are voluntary breathing exercises performed for a short duration while awake. Sleep apnea is involuntary and can occur throughout sleep. When performed by an experienced YB, hypoventilatory YBE result in hypoxic hypercapnia, though not to the same degree as severe OSA (Dempsey *et al.*, 2010). Patients with OSA often get very little sleep and are plagued with chronic sympathetic activation, which can lead to many problems, including cardiovascular disease (Dempsey *et al.*, 2010). To this

author's knowledge, resting sympathetic activation has not been measured in YB. Training in YBE did not affect the response of MCAv to either Duffin re-breathe test. This data supports the theory that reduced cerebrovascular reactivity in OSA patients is not singularly due to hypoxic hypercapnia exposure. Though *chaturbhuj* shares a similar cyclical MAP response with OSA, it seems unlikely that acute exposure to hypoxic hypercapnia through voluntary breathing exercises would cause physiological problems similar to OSA.

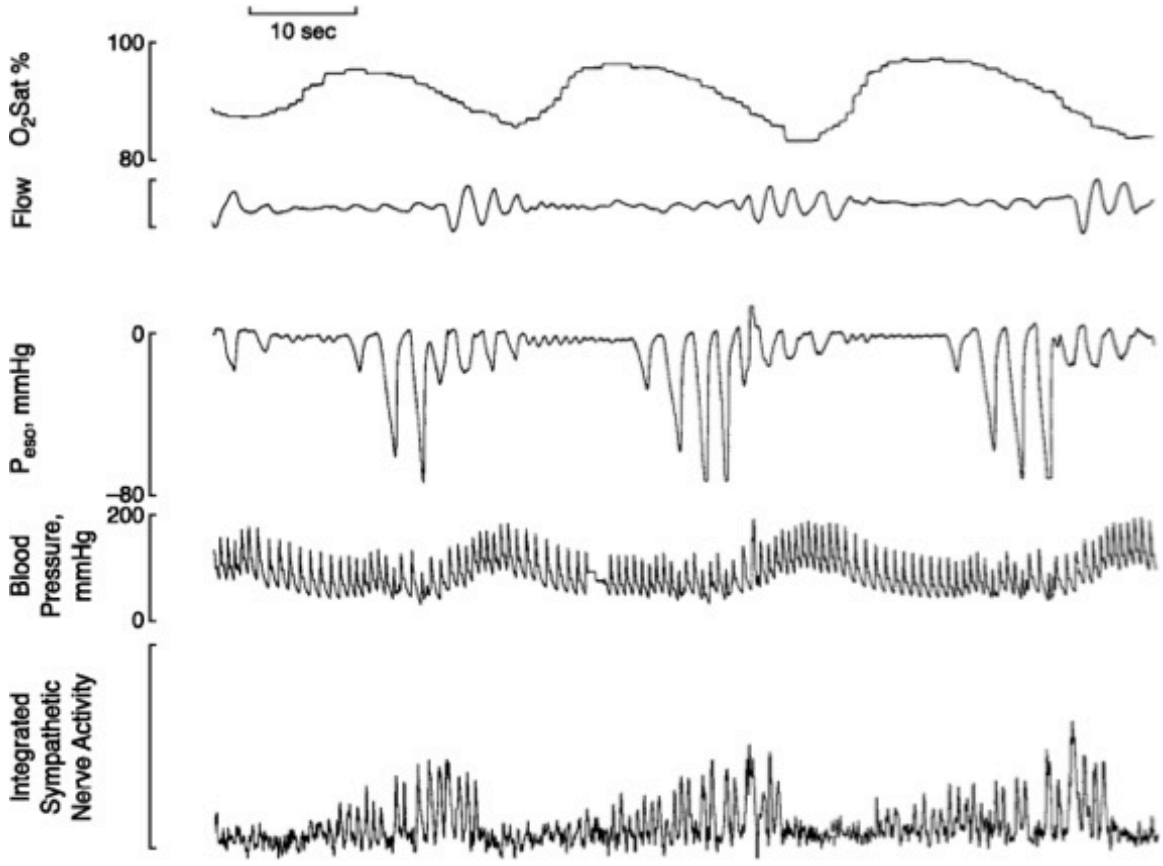

Figure 18. Oscillations of SaO_2, esophageal pressure (P_{eso}), blood pressure, and sympathetic nerve activity in response to sleep apnea in a single subject (Skatrud, 1999).

Chemosensitivity measures

The Duffin re-breathe was used instead of the Read re-breathe in order to quantify both hypercapnic ventilatory threshold and sensitivity. The Duffin re-breathe adds a 5 minute hyperventilation prior to re-breathing from a bag. During the 5 minutes of hyperventilation, the subject is coached to maintain their $P_{ET}CO_2$ between 18 – 25 mmHg. This "blows off" CO_2 in the blood and CO_2 stored in tissues. By blowing off CO_2, after each subject takes their three deep breaths from the bag (which contains a standard gas mixture), every subject starts the re-breathe test at the same level of O_2 and CO_2. From this starting point, as $P_{ET}CO_2$ rises, we can pin-point the level of $P_{ET}CO_2$ from which V_E starts to increase – the hypercapnic ventilatory threshold (Duffin & Mahamed, 2003). This was an important value to collect because hypercapnic ventilatory threshold has been shown to change in response to IH (Koehle *et al.*, 2007), and has never been studied in YB.

The purpose of doing two Duffin re-breathe tests (one hypoxic, and one hyperoxic) was to assess the contribution to the drive to breathe from both and O_2 and CO_2. In the hypoxic test, $P_{ET}O_2$ was kept between 45 – 50 mmHg and resulted in a SaO_2 of approximately 80 %. In this test, the drive to ventilate came from both low oxygen levels and as the re-breathe progressed, rising carbon dioxide levels. In the hyperoxic test, $P_{ET}O_2$ was kept between 145 – 150 mmHg and resulted in a SaO_2 of approximately 98 %. Thus, the drive to breathe in the hyperoxic test was solely from rising carbon dioxide levels.

It is important to note the trend of decreasing baseline ventilation in each subsequent testing session. The Duffin re-breathe is not a comfortable test, and some subjects were anxious both before, and during testing. Perhaps baseline ventilation decreased with subsequent testing sessions because the subjects became more comfortable with the procedure each time they were tested. This points to a slight flaw in the study design. If subjects performed four re-breathes for familiarization instead of just one, perhaps they would have been more relaxed during each testing session.

Other YBE studies have shown decreased hypercapnic ventilatory sensitivity in experienced YB (Stanescu *et al.*, 1981; Spicuzza *et al.*, 2000; Roggla *et al.*, 2001; Miyamura *et al.*, 2002), however, this study showed no change following the intervention. There are four possible main reasons that sensitivity did not change. First, our stimulus may not have been severe enough. 6 weeks of training may not have been long enough to see a change. The amount of time subjects were required to practice at home was less than would be recommended for a serious student. YBE are supposed to be practiced every day and our subjects practiced 5 days per week. If 5 days a week was sufficient, perhaps 20 minutes per day did not create enough hypoxia and hypercapnia to result in altered in chemosensitivity. If subjects were instructed to practice the YBE 7 days a week, and for a longer duration, subject enrollment and compliance would almost certainly have been lower.

Second, though experimental subjects reported practicing the required amount at home, there is no way to know if they did. One subject reported that she practiced the whole time until she was scheduled for her final test, when she explained that in fact, she hadn't practiced at home from week 6 – 9.

Third, because our sample size was small, removing one subject has a large effect on statistical analysis. Two experimental subjects and one control subject ascended to altitude before their final test even though this was an exclusion criterion for participation in the study. Since we have no way of knowing what their sensitivities would have been had they not ascended to altitude, this may have had a significant effect. Exposure to normobaric hypoxia has been shown to increase hypoxic ventilatory sensitivity upon return to sea level (Ainslie *et al.*, 2005) and in this situation, the exposure could have 'washed' any chemosensitivity changes that were due to the intervention.

Fourth, it has been documented that re-breathe tests are quite variable within subjects (Macnutt *et al.*, 2011), and with a small sample size, variability has an especially big impact on statistical significance.

Exposure to lower body negative pressure during hypoxia results in an increased hypoxic ventilatory response (Koehle *et al.*, 2010). This is thought to be because the carotid chemo and baroreceptors are anatomically so close together (Koehle *et al.*, 2010). Because of their location, their afferent inputs may interact as they converge into the nucleus tractus solitaries, resulting in modulation of the signals. A future study may look at the affect of lower body negative pressure on the MAP response to *bhastrika* and *chaturbhuj*, and hypercapnic ventilatory sensitivity in experienced YB.

Limitations

Though 8 out of 9 experimental subjects finished the study, there were some subject related events to note. One control subject had asthma, however, it was under control. Only in patients who have had near-fatal asthma episodes have been shown to have reduced chemosensitivity, therefore her asthma most likely did not affect her

outcome measures (Hida, 1999). A second control subject went to Kenya between weeks 5 and 10 where he was at altitude for three weeks. Altitude has been shown to have an effect on chemosensitivity (Masuda *et al.*, 1981), however his final test was postponed until 4 weeks after he returned to sea level. One experimental subject had exercise-induced asthma, but reported no ill-effects from the re-breathe test. Results did not differ when these subjects were removed from statistical analysis.

Beginners in YBE are normally required to practice the three locks and the four purifications every day for three months before they move onto any YBE with significant breath-holds such as *bhastrika*, *chaturbhuj*, or *sahit kumbhak*. Also, it takes some time before a student becomes comfortable with the intermediate YBE. During the beginning stage, the student is focused on the mechanics of the YBE. With regular practice, the YBE become second nature and the student can simply focus on the breath. However, because the experimental subjects came in every week for an instructional class, this sped up the process of getting them to perform the YBE correctly.

We could not quantify the physiological effects of *sahit kumbhak* due to the experimental setup. The facemask prevented access to the nose, which is required in *sahit kumbhak*. In the future, data could be collected for a modified *sahit kumbhak* where each breath is through both nostrils. This would be worth studying as this YBE employed a inspiration:breath-hold:expiration ratio (1:4:2) that was weighted towards apnea.

Chapter 6: Conclusion

The overall goal of this thesis was to quantify the cardiorespiratory effects of two YBE: *bhastrika* and *chaturbhuj,* and determine the effect of their practice on chemosensitivity. Though we only studied two YBE in the laboratory, from these two studies three things have become clear that can be applied to many YBE. First, hyperventilatory YBE can produce hyperoxic hypocapnia, while hypoventilatory YBE can produce hypoxic hypercapnia. YBE include complex patterns and in *bhastrika,* both of these situations occur: hyperoxic hypocapnia in response to the hyperventilation, and hypoxic hypercapnia in response to the breath-hold. In *chaturbhuj* there is hypoxic hypercapnia due to significantly reduced $V_{t,}$ and V_E.

Second, in response to hypoxic hypercapnia produced by the breath-hold in *bhastrika* and low V_E in *chaturbhuj*, there was an increase in MAP. The increase in MAP was more pronounced in YB (Study 1), and in trained subjects during *bhastrika* (Study 2, week 10) because of more severe hypoxic hypercapnia, and most likely increased sympathetic activation. In week 10, there was no difference in the MAP response to *chaturbhuj*. Though it is impossible to know, this result may have been due to the much smaller data set.

Third, performance of *chaturbhuj* results in cyclical oscillations of HR, MAP, SV, and MCAv. This was a novel and significant finding. To this author's knowledge, these were the first two studies to examine the physiological effects of *chaturbhuj*. However, more analysis is needed to determine how these oscillations affect cardiovascular control in the long term.

In the future, a productive YBE training study should include a longer time commitment from the experimental subjects. If subjects practiced *bhastrika*, *chaturbhuj*, and *sahit kumbhak* for 30 minutes a day, 7 days a week, for two months, that would be a more likely stimulus to reduce chemosensitivity. Additionally, it would be worth examining the respiratory ratio of *sahit kumbhak* in an experienced YB to determine the effect of its practice on end-tidal gases, and to see if there are cardiovascular oscillations similar to those in *chaturbhuj*.

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