SUMMA OP3 – Tech Manuscript

SCOGA (SCI – YOGA)

Effect of Slow Breathing and Yogic-Derived Breathing on Respiration and Cardiovascular Variability in Spinal Cord Injury Patients

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Dedication

…

I dedicate this work to my loving partner, Teodora, whose unwavering support and encouragement have been the foundation of my journey.

To my remarkable mother and father, whose boundless love and guidance have shaped me into the person I am today.

And to my cherished sisters, who have been my constant source of inspiration.
Your presence in my life has been invaluable, and I am grateful for the lessons you have taught me.
As a yoga teacher and pranayama instructor, you have been my favorite disciples, reminding me that even in my role of guiding others, I too need to pause, take a deep breath, and find the stillness within.

In a single breath, more molecules of air will pass through your nose

than all the grains of sand on all the world's beaches—trillions and trillions of them.

These little bits of air come from a few feet or several yards away. As they make their way toward you,

they'll twist and spool like the stars in a van Gogh sky, and they'll keep twisting and spooling and

scrolling as they pass into you, traveling at about five miles per hour.

James Nestor, Breath: The New Science of a Lost Art

ABSTRACT

Spinal Cord Injury (SCI) results in a variable pathophysiologic profile in which deficits vary with level and degree of injury but can markedly impact both autonomic and respiratory control. Therefore, the SCI population may be especially needful of therapeutic slow-breathing interventions to mitigate the impact of altered autonomic control of the heart and blood vessels and to improve the efficiency of respiration. Therefore, we assessed cardiovascular variabilities during 7 minutes of uncontrolled breathing, controlled breathing at 0.25 Hz and at 0.083 Hz, and ujjayi breathing (0.083 Hz with expiratory resistance via throat constriction) in 12 individuals with SCI from T8 to C4. Beat-to-beat heart rate and blood pressure was measured as well as end-tidal CO2 and oxygen saturation. Compared to uncontrolled breathing, all controlled breathing increased coherence between systolic pressure and RR interval variabilities at the respiratory frequency (0.71 Hz vs >0.85 Hz, $p<0.05$). The phase relation was shifted by 0.25 Hz breathing to a markedly positive, feedforward relationship (–4° vs +43°, $p<0.05$), whereas both 0.083 and ujjayi breathing shifted it to a markedly negative, feedback relationship –45° and –44°, $p<0.05$). Moreover, compared to uncontrolled breathing, 0.25-Hz breathing markedly decreases variabilities at the respiratory frequency in both RR interval and systolic pressure; 0.083-Hz breathing has no effect, whereas ujjayi markedly increases both variabilities (all $p<0.05$). While 0.25 Hz and 0.083 Hz breathing had variable effects on end tidal CO_2 and oxygen saturation, only ujjayi breathing decreases end tidal CO_2 and increases oxygen saturation while also resulting in a modest tachycardia (all $p<0.05$). These findings suggest that slow yogic breathing with respiratory resistance (ujjayi) shifts cardiovascular control to a baroreflex-mediated feedback mechanism and increases both mean heart rate and its variability such that there is improved ventilation-perfusion matching, resulting in increased systemic oxygen saturation. understanding the impact of respiratory changes, such as increased work of breathing, and exploring interventions that can improve respiratory function, cardiovascular control, ventilation-perfusion matching, and oxygen saturation is crucial in the SCI population to manage respiratory complications, enhance respiratory function, and optimize overall health and well-being.

Acknowledgments

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1. INTRODUCTION

1.1 — Background

Spinal Cord Injury (SCI) is a devastating condition that affects approximately 17,000 individuals every year in the United States. SCI frequently results in severe impairment of the respiratory system, with the degree of impairment increasing with the level of injury (1). Respiratory insufficiency is the leading cause of mortality and morbidity after SCI, with around two-thirds of patients experiencing complications such as atelectasis, pneumonia, and/or respiratory failure (2). In the acute setting, mechanical ventilation support is necessary when patients are unable to maintain adequate ventilation. However, long-term mechanical ventilation can weaken the respiratory muscles, leading to ventilator-dependent diaphragmatic dysfunction (VIDD) or diaphragmatic atrophy (3). The Veterans of America respiratory care guidelines recommend a range of therapeutic options for SCI patients with respiratory complications, including incentive spirometry, nebulization, intermittent positive pressure, and aerosol therapy as well as referral to a respiratory therapist (4). Despite these recommendations, respiratory therapy regimens remain non-standardized and inadequately characterized in research, leading to heterogeneous treatment approaches around the world. Therefore, effective respiratory health management after SCI is crucial to reduce the risk of premature death and excess disability. To achieve this goal, a clear understanding of the relationship between lung function impairment and the level and completeness of SCI is necessary. One promising approach to understand cardiopulmonary physiology better is the study of breath pacing and breathing exercises, which have been extensively researched in healthy individuals but remains unstudied in the SCI population.

1.2 — Classification and Mechanisms of Spinal Cord Injury

SCI can be classified based on their level and completeness. In complete SCI, the spinal cord is fully severed, resulting in the loss of function below the site of injury. In contrast, incomplete SCI, which frequently occurs due to compression, preserves the brain's ability to send signals below the injury site. Respiratory muscle impairment can start as low as L5[1] but becomes more pronounced at higher thoracic and cervical lesion levels where the diaphragm is innervated. Total lung capacity and vital capacity are often restricted, leading to atelectasis, chronic infections, and obstructive sequelae (5, Figure 1). Although functional residual capacity is also reduced, it is not as severely impacted as total lung capacity. The reduction in functional residual capacity occurs due to a decrease in expiratory reserve volume, which compensates for an increase in residual volume. The loss of expiratory reserve volume can be attributed to the denervation of abdominal musculature and other muscles necessary for forced exhalation. As most SCI patients have some respiratory involvement, careful long-term respiratory management is crucial to minimize the risk of respiratory complications, especially for individuals with a high risk of developing such complications (5).

1.3 — Respiratory Impairment Following Spinal Cord Injury

SCI causes restrictive impairment of the respiratory muscles, resulting in reduced vital capacity (VC), as well as increased respiratory effort and difficulty with coughing (6). The ventilatory pattern can change significantly during the first year following SCI, with respiratory muscle pressure-generating capacity changing over time and correlating with lesion level (7, 8). After SCI injury, flaccid paralysis disables intercostal and abdominal muscles, causing paradoxical breathing movements and reducing VC by 20–60% in

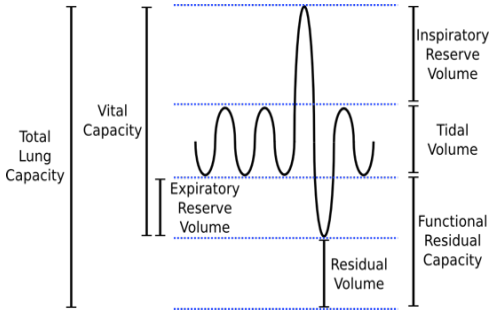


Figure 1: Volumes of the Lung

[1] The 5th Lumbar Vertebra

tetraplegics and 80–90% in paraplegics (9, 10, Figure 1). This paradoxical ventilation occurs when the diaphragm contracts against an unstable rib cage (11). Over time, the rib cage stabilizes, and VC returns to approximately 60% of the pre-injury level (12). However, functional residual capacity is progressively diminished during this period, leading to atelectasis and basal pulmonary fibrosis (12). The residual volume remains elevated compared to values in the normal population (12–14). Unfortunately, the total lung capacity does not appear to recover significantly during this time (12, 13).

1.4 — Respiratory Management for Individuals with Spinal Cord Injury

Studies have shown that, like other skeletal muscles, respiratory muscles in able-bodied individuals can be trained for both strength and endurance (15). However, respiratory weakness is common in individuals with SCI, which increases the risk of respiratory fatigue. Nevertheless, significant improvements in respiratory muscle performance can be achieved with substantial training efforts (16–19). It is worth noting that even marathon runners, who breathe vigorously but not forcefully, do not exhibit enhanced respiratory muscle strength, emphasizing the importance of vigorous and forceful efforts to produce a meaningful effect (20). During the prolonged process of weaning patients with SCI from assisted ventilation, concurrent respiratory muscle training may be beneficial (21). However, the optimal training regimen remains unclear, and it is not known how long individuals will continue the training and whether any improvements will be maintained after training cessation. Targeting both endurance and strength training would seem to be the most desirable approach. It is also noteworthy that although previous studies have shown the benefits of respiratory muscle training, none of the participants chose to continue the training after the study ended, preventing any possibility of long-term follow-up (16-21).

1.5 — Pranayama: Exploring the Mind-Body Benefits of Yoga Breathing Techniques

Interest in mind-body medicine has surged in recent years, with a focus on interventions such as meditation, yoga, tai chi, biofeedback, and cognitive behavioral therapy to promote health. One specific area of interest is the potential therapeutic effects of slow breathing (<6 breaths/min or 0.1Hz) and traditional yogic (pranayama) breathing practices, which date back to ancient India and the origins of yoga in the sixth and fifth centuries BCE. The Sanskrit word pranayama combines *prana* (life energy) and *ayama* (extension or expansion, to draw out) and generally refers to breath control and practicing specific yogic breathing techniques (YBTs) such as nostril manipulation, breath holding/retention, modification in the pace of breath, production of humming sounds, and more (22, 23). Although breath is an integral part of any yoga or fitness practice, pranayama involves breathing exercises that can be practiced alone, as a supporting practice, or integrated as part of a physical practice. While several studies have evaluated healthy cardiopulmonary physiology after various YBTs, few have studied these effects during the practice of YBTs (22–27). Moreover, there are no existing studies that have examined these pranayama techniques in the SCI population.

1.6 — The Importance and Application of Heart Rate Variability

Heart Rate Variability (HRV) is a measure of the variation in times between heartbeats, controlled by the autonomic nervous system (ANS). Autonomic tone – or the balance between the sympathetic and the parasympathetic nervous system – regulates heart rate, blood pressure, breathing, and digestion among other key body functions. It responds to meet the stresses one encounters in a day and with elevated transient stressors this balance can be disrupted. Studies have shown that slow breathing, a significant component of yogic breathing exercises, can impact blood pressure regulation and cardiac autonomic tone (28–31). Patterns of breathing can significantly affect cardiovascular variabilities, including Heart Rate Variability (HRV) and Blood Pressure Variability (BPV). In fact, these variabilities have been used as indicators of autonomic circulatory control in various studies (32). However, pathophysiological processes

can disrupt the relationship between breathing rate and cardiovascular variability, particularly in those with SCI (33). Individuals with SCI above T6[2] experience persistent hypotension and bradycardia daily and SCI has repeatedly been linked to an increased risk for cardiovascular disease (CVD) that may be related to increased BPV and decreased HRV (34–36). Decreased HRV is associated with cardiac diseases and is prognostic for those with known CVD (37, 38). Moreover, HRV decreases with age, is lower in those with a sedentary lifestyle, and is inversely related to inflammatory markers in both healthy individuals and those with CVD (39). Specific to SCI, HRV decreases within the first 24 months after injury, suggesting that this decline is mainly due to a direct impact of the autonomic injury itself rather than the lifestyle effects of SCI (40). Individuals with SCI may benefit from therapeutic slow-breathing interventions to mitigate the impact of altered autonomic control on the heart and blood vessels and improve respiratory efficiency.

Research has shown that slow breathing techniques can lead to a resonance effect on heart rate and blood pressure due to the low-frequency respiratory sinus arrhythmia (RSA) that occurs during slow breathing (41, 42). Studies examining the effects of traditional slow breathing techniques on heart rate variability have found that they generally result in greater HRV and synchronous BPV (43–46). Data from studies comparing three separate meditation techniques found that all practices create prominent low-frequency oscillations coupled with a slow respiratory rate (46). Other studies have found that yogic-derived slow breathing at 0.10 Hz leads to decreased blood pressure during the slow breathing period in healthy individuals, as well as those with hypertension and congestive heart failure (28, 29, 47–49). These findings suggest a relationship between slow breathing and hemodynamic changes, however, there is still a need for research on the association between slow breathing and cardiopulmonary changes in individuals with SCI.

1.7 — Study Rationale

Respiratory insufficiency is the primary cause of morbidity and mortality in all individuals with SCI, and therefore, improving respiratory function in all SCI patients is a priority for research. However, little research has focused on the interrelationships between the respiratory and cardiovascular systems in SCI patients. The loss of autonomic control in SCI predisposes these individuals to cardiovascular dysregulation, which increases their cardiovascular risk and accelerates aging. This suggests that the effects of slow breathing on cardiovascular variabilities, such as heart rate and blood pressure, may differ in individuals with SCI. While the relationship between slow breathing and cardiovascular variability has been studied in healthy individuals, breathing exercises, as a low-cost and non-invasive intervention, have the potential to improve the respiratory and cardiovascular health of individuals with SCI. Since most SCI patients experience some loss of respiratory muscle function and altered sympathetic and parasympathetic balance, research may show that these individuals can benefit from the effects of YBTs. However, the lack of research into the effects of YBTs on individuals with SCI indicates a need to examine whether slower breathing patterns are possible in this population and their effect on cardiovascular control and downstream health effects. Therefore, this physiological study aims to compare the effects of spontaneous breathing and traditional yogic slow-breathing practices on cardiovascular variabilities in individuals with SCI.

[2] The 6th Thoracic Vertebra

2. MATERIALS AND METHODS

2.1 — Participants

The study included medically stable male and female participants between the ages of 18–60, with a body-mass index (BMI) between 18.5–35 kg/m^2, a history of traumatic SCI, and who used a wheelchair. Exclusion criteria consisted of non-English speakers, a history of renal, neurological, or coronary artery disease, cancer related to SCI, uncontrolled or type II diabetes, significant arrhythmia, smoking, or current use of cardioactive medications. The study protocol was approved by the Institutional Review Board of the Mass General Brigham Human Research Protection Program (HRPP) and registered on clinicaltrials.gov with the ID# NCT05480618.

15 participants with traumatic SCI and with no prior experience in yogic–breathing were recruited to the study were recruited from the Exercise for Persons with Disabilities (ExPD) program at Spaulding Rehabilitation Hospital in Cambridge, Massachusetts, USA. Though all subjects completed their two-week breathing training period, three subjects failed to complete the final lab visit — two due to respiratory illness and one showed signs of autonomic dysreflexia during data acquisition. The remaining 12 individuals had an average age of 33 (±5.9) years (Table 1). All participants provided written informed consent, and confidentiality was maintained through the use of signed certificate of confidentiality forms.

Table 1: Characteristics of the Study Cohort

	Participants (N=12)
Sex	
Male	9 (75%)
Female	3 (25%)
Age (years)	
Mean (SD)	33 (5.9)
Median (CV%)	32 (17.8)
[Min, Max]	[23, 45]
Race	
White	8 (66.7%)
Black or African-American	4 (33.3%)
Ethnicity	
Not Hispanic or Latino	11 (91.7%)
Hispanic or Latino	1 (8.3%)
Level of Injury	
Cervical	6 (50%)
Thoracic	6 (50%)
ASIA Classification	
A	8 (66.7%)
B	3 (25%)
C	1 (8.3%)
Body Weight (kg)	
Mean (SD)	86.1 (19.1)
Median (CV%)	82.3 (22.2)
[Min, Max]	[56.7, 120.2]
Height (cm)	
Mean (SD)	181.2 (10.29)
Median (CV%)	184.2 (5.7)
[Min, Max]	[157.5, 198.1]
BMI (kg/m²)	
Mean (SD)	26.1 (5.02)
Median (CV%)	24.0 (19.2)
[Min, Max]	[19.7, 35.0]

2.2 — Study Design

This randomized controlled trial was a within-subject physiological study investigating blood pressure and heart rate variability during spontaneous breathing, 0.025-Hz paced breathing, and four different YBTs paced at 0.083 Hz. The intervention was unblinded and conducted once. Prior to the intervention, participants received two weeks of familiarization and training during which they were provided in-person coaching from a certified yoga/pranayama instructor (MM). Coaching sessions were held on Day 1 and around Day 7, during which participants practiced YBTs. Participants were asked

to practice the YBTs at home for two weeks (Figure 2). After the two-week practice period, participants came to the Cardiovascular Lab to perform the YBTs while their outcome variables were measured.

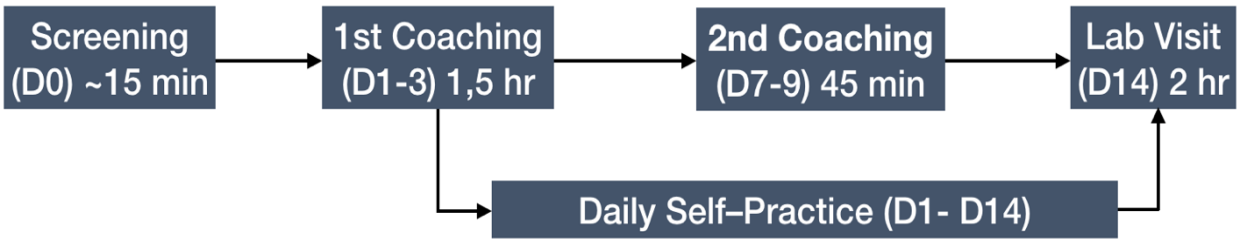


Figure 2: Study Schema for Two-Week Training Period (Coaching & Daily Self-Practice) and Acquisition of Cardiopulmonary variabilities in Cardiovascular Laboratory (D14) during each YBT.

2.3 — Experimental Protocols & Assessments

Baseline and Pulmonary Function Testing

All subjects were tested by the same researcher (MM[3]) and were instructed to loosen their belts and wear a nose clip. On both day 1 and day 14, participants performed standard spirometry for SCI, as described in reference (50), to obtain baseline and post-practice measurements.

For the measurement of functional expired volume in 1 through 6 s (FEV1-6) and functional vital capacity (FVC), a spirometer (ParvoMedics TrueOne 2400, Sandy, Utah) was used. Participants were instructed to inhale completely and exhale maximally with sustained effort of at least 6 s or longer, depending on the subject's ability to continue. The volume-time curve was recorded on a kymograph, and the flow-volume loop was electronically displayed for review. Acceptable efforts required a minimum exhalation time of 3s, a rapid start, and a well-defined early peak inflow that was smooth and continuous. Typically, three acceptable efforts were obtained in six to eight attempts.

A handheld respiratory pressure meter (MicroRPM, Vyaire Medical, Mettawa, IL, USA)recorded the maximal inspiratory pressure (MIP) and maximal expiratory pressure (MEP). Participants were instructed to inhale and exhale using maximal force to achieve the greatest pressure, and a sustained effort was not necessary. Participants were encouraged to surpass their previous effort, and typically, three acceptable efforts were obtained in four to six attempts.

Final Lab Visit: Cardiovascular Testing

All participants were instructed to avoid vigorous exercise for 24 hours prior to each lab visit to minimize autonomic and neuroendocrine effects. They were also asked to refrain from consuming caffeine and alcohol for the previous 24 hours and eat no large meals during the two-hour period before their lab visit. Throughout the protocol, participants were positioned in a semi-supine position with the trunk at a 30° angle and were instrumented for analysis with various devices, including a Dash 5000 portable patient monitor with 5-lead EKG, brachial cuff and finger pulse oximeter (GE Healthcare, Chicago, IL, USA), limb doppler (Doppler BoxX, DWL, Singen, Germany), Finometer finger photoplethysmograph (Finapres Medical Systems B.V., Netherlands), Respitrace transducer bands around the chest and belly (Respitrace, ADInstruments, Sydney, Australia), Model 17515A Gold Edition CO_2 monitor with a nasal cannula (Vacumed, Ventura, CA, USA), and a microphone. To ensure the accuracy of beat-by-beat blood pressure measurements, brachial arterial blood pressure was recorded from the right arm using an automated brachial cuff every two minutes during the data collection period. At the final lab visit, audio tracks were

[3] MM: Marc Mazur

used with minimal additional coaching from the research staff. During instrumentation and calibration, the subjects underwent a period of acclimation with an unpaced breathing pattern for about five to ten minutes. Each breathing pattern (Figure 3) was recorded for approximately seven minutes to provide sufficient breath cycles for reliable data analysis at this breathing frequency. All participants performed six different types of breathing patterns in a random order, which was generated using an online randomizer[4]. Upon completion, subjects were asked to submit their two-week breathing practice logs.

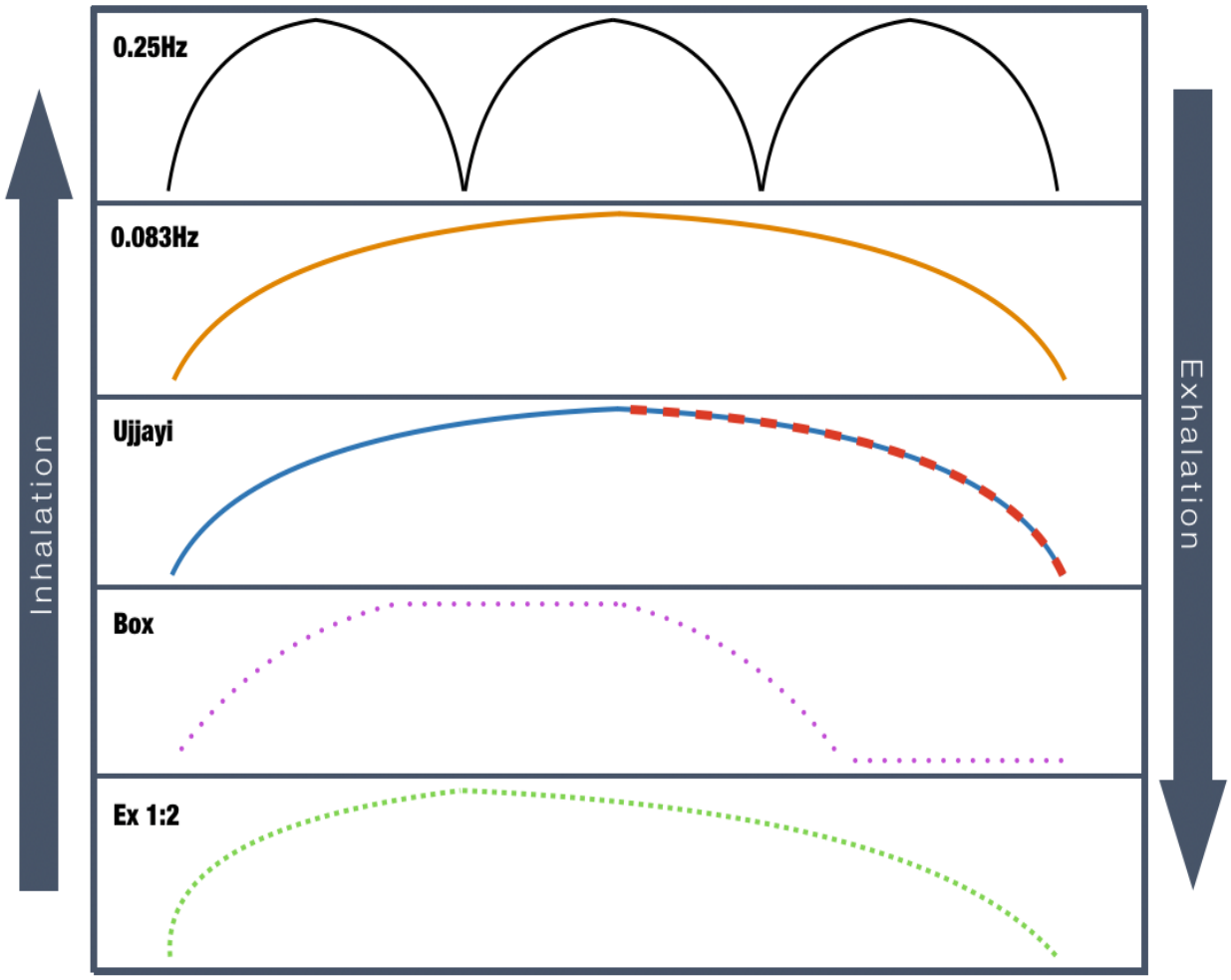


Figure 3: Schematic of different Breathing Patterns and Frequencies – 0.25Hz breathing (normal frequency at 15 breaths per minute), paced 0.083 Hz breathing (Resonant or Coherent breathing at 5 breaths per minute), Ujjayi or Victorious breathing with resistance on the exhalation, Box Breathing with 3 s segments with inspiratory and expiratory breath holding, Extended exhalation with a 1:2 Duty Cycle (4 s inhale, 6 s exhale), Variable or Spontaneous breathing (not pictured). All YBTs were paced at 0.083Hz and all breathing patterns including spontaneous breathing were practiced through the nose.

2.4 — Yogic-Breathing 'Pranayama' Intervention

In this study, the breathing patterns were manipulated with variations in breath frequency (ranging from 0.25 Hz to 0.083 Hz), duty cycle (inspiratory-expiratory ratio, I:E), and the presence or absence of Ujjayi, a yogic throat constriction technique, as well as the incorporation of inspiratory and expiratory breath holding known as Kumbhaka or 'Box Breathing' (Table 2 & Figure 3).

[4]Randomization performed at https://www.random.org/sequences/

SCOGA Breathing Patterns and Techniques					
#	Common Name	Pranayama Name	f (Hz)	f (Breaths/min)	Duty Cycle (I:E)
1	Unpaced Breathing	—	Variable	Variable	~2:3
2	Fast Paced Breathing	—	0.25	15	1:1
3	Slow Paced Breathing	Resonant Breathing	0.083	5	1:1
4	Victorious Breathing	Ujjayi Pranayama	0.083	5	1:1'
5	Box Breathing	Sama Vritti Pranayama	0.083	5	1:1:1:1
6	Slow Exhale Breathing	Rechaka Pranayama	0.083	5	1:2

Table 2: SCOGA Breath Patterns Details – Traditional Name, Duty Cycle, Frequency (Hz), and Breaths per Minute.

The participants were instructed to breathe exclusively through both nostrils for all breathing patterns, and their breathing saturation was actively monitored for signs of hyperventilation. Verbal instructions were given to participants to breathe less deeply if hyperventilation was observed. For the 0.25-Hz breathing pattern, participants were paced to inhale for two seconds and exhale for two seconds. The 0.083-Hz breathing pattern involved a slower pace, with participants instructed to inhale for six seconds and exhale for six seconds. For Ujjayi breathing, participants were paced at the same rate as during slow breathing but instructed to add resistance (yogic throat constriction) to the exhalation. During Box Breathing, participants were instructed to inhale for three seconds, hold their breath at the top of inhalation for three seconds, exhale for three seconds, and hold their breath at the bottom of exhalation for three seconds. Lastly, for Long Exhale Breathing, participants were instructed to inhale for four seconds and exhale for eight seconds.

2.5 — Data Collection and Analysis

The acquired physiological signals were digitized and recorded at a sampling rate of 1000 Hz[5], using Power Lab software by ADInstruments. R-R intervals were detected from ECG recordings through a peak detection algorithm in Powerlab, and then visually inspected for artifacts and errors. Similarly, beat-to-beat systolic blood pressure (SBP), SaO_2, and $EtCO_2$ were determined with a built-in peak detection algorithm and verified visually. Data was exported as Matlab files and processed using custom Matlab code (Version 9.13; Mathworks, Natick, MA) adapted from existing code used in the lab and modified by MM (see Section 7.2). Power spectral densities of R-R intervals and SBP were computed using fast Fourier transforms based on Welch's periodogram algorithm on 5-Hz resampled time series. The mean respiratory, R-R interval, and systolic pressure powers were log-transformed to ensure normality. Cardiopulmonary interactions were assessed by conducting a cross-spectral analysis between the power of R-R interval and systolic blood pressure at the designated breathing frequencies.

2.6 — Statistical Analysis

We used a two-tailed paired t-test to compare the mean R-R interval, systolic blood pressure, SaO_2, and $EtCO_2$ among different breathing techniques and unpaced breathing. Additionally, pre- and post-study PFTs, R-R interval powers, systolic pressure powers, coherence, phase, and gain were subjected to two-tailed paired t-tests for comparison. The results are presented as means with standard deviation, and statistical significance was considered at a significance level of $p < 0.05$.

[5] Except for the microphone, which had a higher sampling rate of 20,000 Hz.

3. RESULTS

3.1 — Pulmonary Function Testing

We averaged all MIP and MEP values for each subject before and after the familiarization period and calculated the absolute change (Δ) was for each subject (as shown in Figure 3, Left & Table 3, Appendix). The analysis of the group data reveals a moderate, yet statistically significant increase in inspiratory muscle strength, with a mean absolute change of 8.6 cmH2O (Figure 4 (right) & Table 4, Appendix $p < 0.05$). However, there are no significant changes observed in MEP.

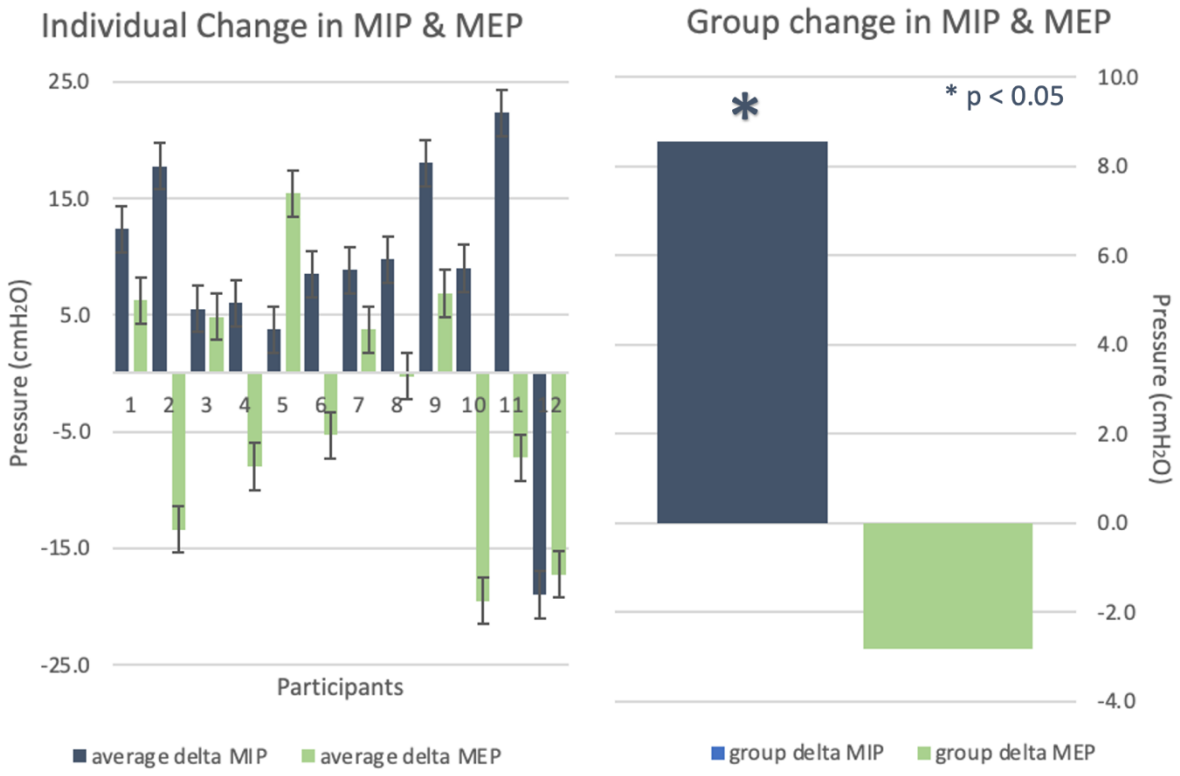


Figure 4: Left – column graph for each SCOGA participant with absolute change (Δ) for pre- and post-coaching MIP and MEP values. Right – column graphs for the whole group with group absolute change (group Δ). Significance ($P < 0.05$) for comparison against unpaced breathing is indicated by ''.*

3.2 — Time-Series Analysis: Average Cardiopulmonary Variables

We compared cardiopulmonary values during all YBTs and 0.25-Hz breathing to unpaced breathing (Figure 5 & Table 5, Appendix). Among the techniques assessed, Ujjayi breathing exhibits a statistically significant increase in heart rate by 5 beats per minute, and RR interval variability of 78.9 ms vs. 69.8 ms ($p < 0.05$). Additionally, when compared to unpaced, Ujjayi breathing results in a significant decrease of $EtCO_2$ and an increase in SaO_2 (32.5 mmHg vs 35.2 mmHg & 97.7 % vs 96.1 %; $p < 0.05$). Similar to Ujjayi, 0.25Hz breathing shows a significant decrease in $EtCO_2$ to 31.4 mmHg (Figure 5 (middle), Table 6, Appendix). Furthermore, 0.083-Hz breathing is the only other breathing pattern to show a significant increase in SaO_2 to about 97.0 % (Figure 5 (right), Table 6, Appendix). No significant changes occur in systolic or diastolic blood pressure or RR interval (Table 6, Appendix).

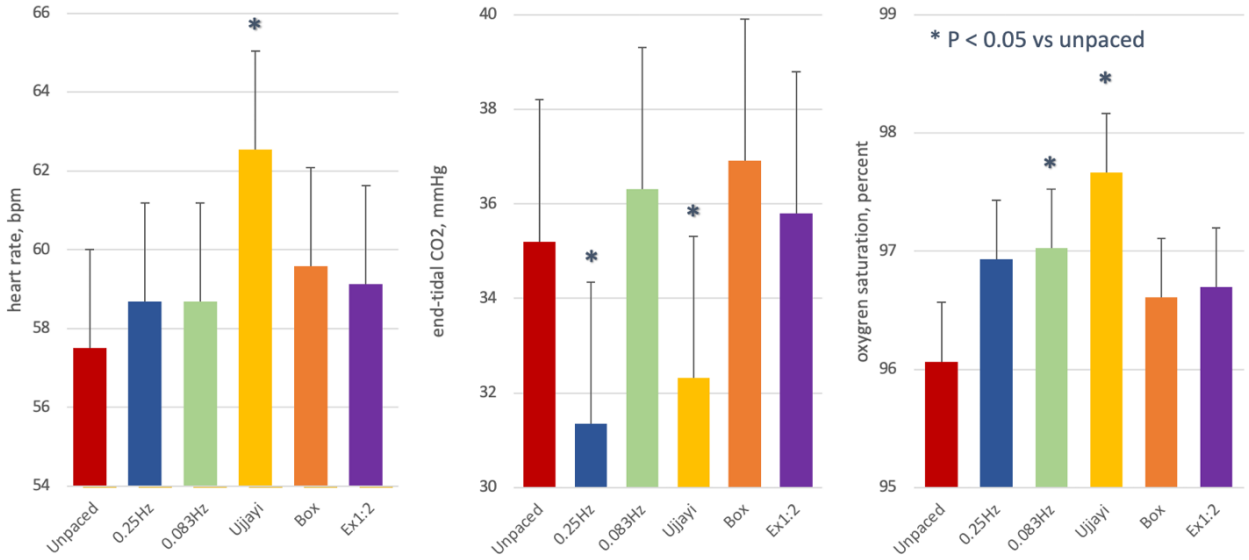


Figure 5: Column graphs for mean HR (left), mean $EtCO_2$ (middle), and SaO_2 (right) during all YBTs. $p < 0.05$ for comparison against unpaced breathing is indicated by ''.*

3.3 — Frequency-Based Analysis: Power Spectral Density

Compared to uncontrolled breathing, 0.25-Hz breathing significantly reduces the power of variabilities at the respiratory frequency in both RR interval and systolic pressure (1.3 mmHg/Hz and 608.6 ms/Hz vs 3.1 mmHg/Hz & 1845.4 ms/Hz, Figure 6 & Table 7, Appendix; $p < 0.05$), while Ujjayi breathing notably increased both measures (13.9 mmHg/Hz & 4832.5 ms/Hz, Figure 6 & Table 7, Appendix; $p < 0.05$).

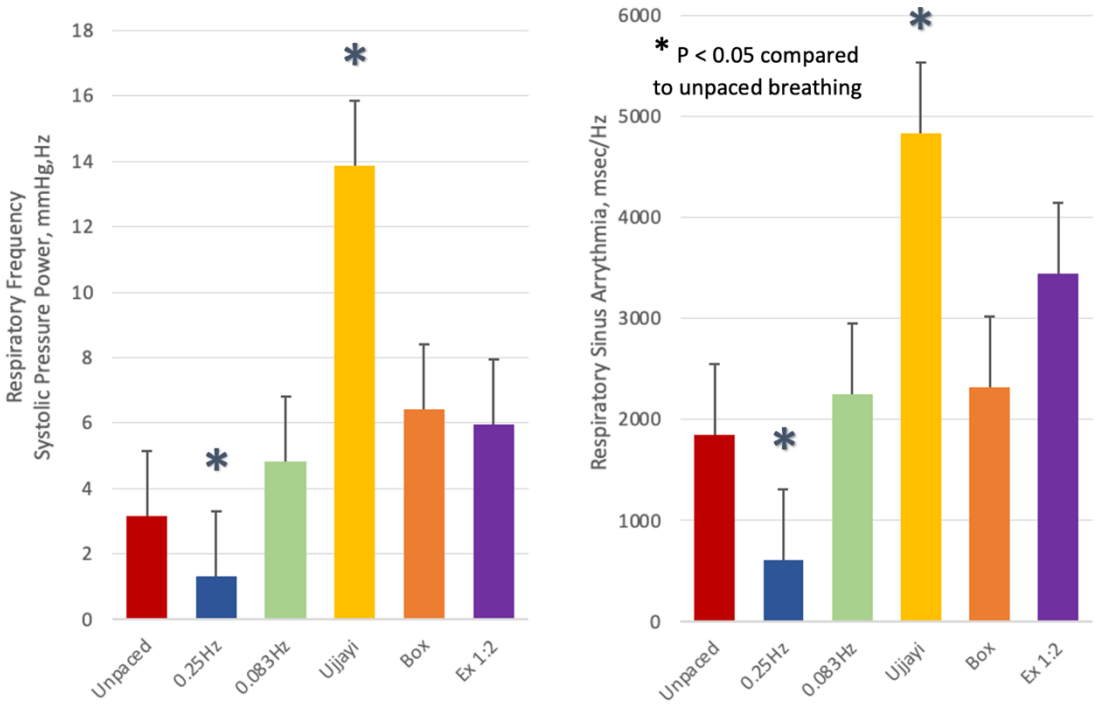


Figure 6: Column graphs for the power of mean SBP (left) and the power of mean RSA (right) at the desired respiratory frequency during all YBTs. RSA is the component of HRV that can be attributed to respiration. $P < 0.05$ for comparison against unpaced breathing is indicated by ''.*

3.4 – Frequency-Based Analysis: Cross-Spectral Density

Figure 7 and Table 8 (Section 7.1) reveal significant findings regarding the effects of different breathing techniques on the coherence and phase relation between systolic pressure and RR interval variabilities. Compared to unpaced breathing, all breathing techniques – 0.25 Hz, 0.083 Hz, Ujjayi, Box, & 1:2 breathing – exhibit increased coherence between systolic pressure and RR interval variabilities at the respiratory frequency (0.71 Hz vs. 0.87 Hz, 0.88 Hz, 0.85 Hz, 0.90 Hz, and 0.88 Hz, respectively; all $p<0.05$). Notably, 0.25-Hz breathing induces a marked positive, feedforward phase relationship, while 0.083-Hz, Ujjayi, Box, and 1:2 breathing induced a marked negative, feedback phase relationship (+34° vs. –36°, –34°, –40°, and –27° degrees, respectively; all $p<0.05$).

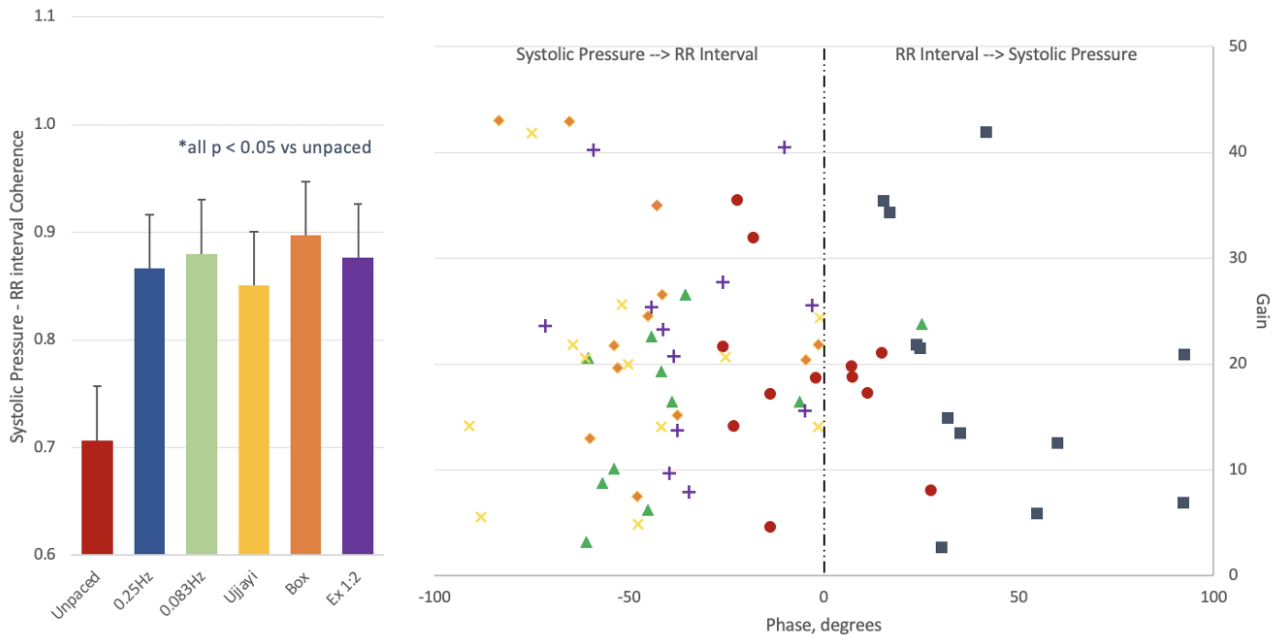


Figure 7: Left – Column graph for cross-spectrum between powers of mean SBP and mean RSA. Right – Phase vs Gain scatter plot for all SCOGA breathing techniques, negative vs. positive phase indicates the directionality of the relationship between SBP and RRi. P < 0.05 for comparison against unpaced breathing is indicated by ''.*

4. DISCUSSION

4.1 — Hypothesis Testing

The primary objective of this study is to assess the ability of individuals with SCI to perform slow yogic breathing patterns and examine the relationship between respiratory control and the level of injury. We hypothesized that a two-week familiarization program with two coaching sessions would result in adequate respiratory control (maintenance of frequency and volume over time) in patients with traumatic SCI, regardless of their injury level.

This investigation yields compelling results, demonstrating that all participants are able to successfully perform slow yogic breathing techniques after the two-week self-practice period. It is worth noting that one participant experienced autonomic dysreflexia during the protocol, but this was likely attributable to their position rather than the breathing technique. These findings strongly support the hypothesis that individuals with SCI can achieve sufficient respiratory control through proper coaching and consistent practice of slow yogic breathing, regardless of their injury level.

Pulmonary function testing conducted before and after the training period reveals a modest yet significant increase in maximal inspiratory pressures, which is often considered an indicator of muscular strength. However, it is important to mention that the yogic breathing exercises are not designed to enhance strength but rather to promote respiratory control. While changes in the strength of respiratory muscles are possible, they require more intense effort and prolonged training (as discussed in Section 1.4). This finding suggests that the daily practice and focused attention on breathing may influence neuroplasticity or the neuromuscular connection of the inspiratory muscles. No significant changes occur in expiratory pressures. In fact, some patients even displayed a decrease in maximal expiratory pressures over the course of the study. This may be attributed to the expiratory muscles being innervated at a lower vertebral level, making them more susceptible to impairment or damage in individuals with SCI (Figure 8). While it is possible that patients experience an increase in maximal inspiratory pressure due to repeated exposure to pulmonary function testing, the decreases in maximal expiratory pressure for some subjects makes this scenario less likely.

We observed an increased level of difficulty or effort in performing yogic breathing techniques among individuals with cervical injuries (C3-C7). This observation indicates that the specific physical limitations imposed by such injuries, including impaired function of the inspiratory and expiratory muscles associated with higher-level injuries (Figure 8), contributes to challenges in achieving precise respiratory control. Previous studies exploring the intricate relationship between SCI and long-term respiratory motor control align with the findings of this study (16-21, 51-53).

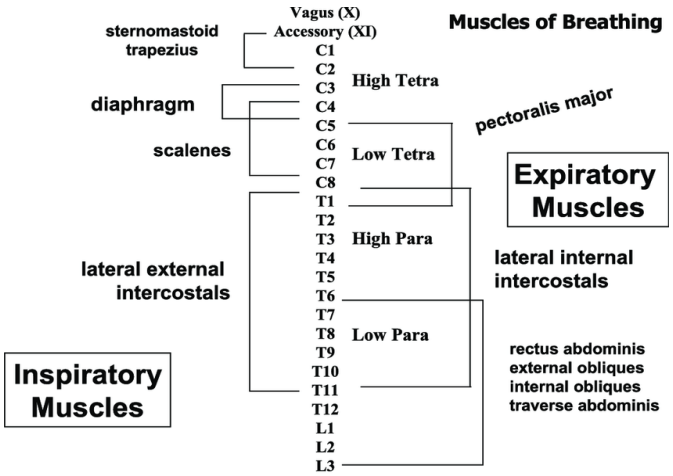


Figure 8: Inspiratory and Expiratory muscles and SCI. (From Schilero GJ, Spungen AM, Bauman WA, et al. Pulmonary function and spinal cord injury. Respir Physiol Neurobiol 2009;166(3):130;

These results highlight the importance of considering injury characteristics when designing tailored respiratory rehabilitation programs. The findings not only support our initial hypothesis that this population is able to perform these techniques, but also underscore the significance of consistent and

frequent engagement in conscious breathing exercises to optimize respiratory function in this population. Moreover, the influence of injury level on respiratory control warrants attention in the development of personalized respiratory rehabilitation strategies.

As a secondary objective, this study evaluates the hemodynamic changes associated with slow yogic breathing in individuals with SCI. We that during slow yogic breathing, as the respiratory rate decreases, heart rate will increase, and blood pressure decreases compared to spontaneous breathing.

Our investigation reveals significant alterations in heart rate, heart rate variability, $EtCO_2$, and SaO_2 during the practice of Ujjayi pranayama compared to spontaneous breathing. Specifically, we observe a notable increase in heart rate and oxygen saturation, along with a decrease in EtCO2 levels during the use of the Ujjayi breathing technique. The significant increase in heart rate and oxygen saturation, along with the decrease in EtCO2 levels observed during Ujjayi pranayama, indicate that this particular breathing technique induces a heightened autonomic response. The Ujjayi technique involves the constriction of throat muscles, creating resistance and increasing intrathoracic pressure. While the exact mechanisms behind these findings are still unknown, these findings suggest that Ujjayi pranayama, more so than other yogic breathing techniques, activates the parasympathetic nervous system, leading to increased heart rate variability and improved ventilation-perfusion matching. This aligns with previous research on the benefits of slow yogic breathing in activating the parasympathetic system and improving ventilation in healthy patients (29, 55-58).

Contrary to the second hypothesis, we do not observe significant changes in blood pressure during slow yogic breathing compared to spontaneous breathing. It is important to note that although the overall mean blood pressure does not show significant differences, individual variations occur within the participant group. These variations may be attributed to the complex interplay between respiratory and cardiovascular systems and the potential influence of individual physiological responses and the ability to buffer blood pressure changes. Although this study does not detect significant changes in blood pressure during slow yogic breathing, the observed alterations in heart rate and heart rate variability during Ujjayi pranayama provide valuable insights into the hemodynamic effects of this breathing technique in individuals with SCI. The significant increase in heart rate and heart rate variability suggests that slow yogic breathing elicits physiological responses associated with enhanced autonomic modulation, contributing to our understanding of the potential cardiovascular benefits of this practice in the context of SCI.

The third objective of this study was to investigate the changes in the relationship between respiratory frequency, heart rate, and blood pressure variabilities induced by slow yogic breathing. We hypothesize that slow yogic breathing would result in a more synchronized pattern of fluctuations between heart rate and blood pressure.

Through spectral analysis of the heart rate and blood pressure data, we observe a significant increase in coherence and phase synchronization between these variables during slow yogic breathing. The observed synchronization of heart rate and blood pressure fluctuations during slow yogic breathing suggests a potential mechanism by which this practice may help restore cardiovascular homeostasis and improve overall cardiovascular function in individuals with SCI. This finding supports our hypothesis that slow yogic breathing leads to a more synchronized pattern of heart rate fluctuations and systemic blood pressure fluctuations in individuals with SCI. This holds significant implications for cardiovascular health in individuals with SCI, as disrupted autonomic regulation is often observed in this population.

Furthermore, based on the findings of increased coherence and phase synchronization between heart rate and blood pressure during slow yogic breathing, it is plausible to speculate that this practice may enhance the functioning of the baroreflex-mediated feedback mechanism. The baroreflex is a highly intricate, but vital, physiological mechanism involving the interplay between the cardiovascular and autonomic nervous systems. It encompasses a complex feedback loop that regulates blood pressure by sensing changes in arterial pressure and initiating appropriate adjustments in heart rate and vascular tone. Understanding the intricacies of the baroreflex requires comprehensive knowledge of the neural pathways, receptor systems, and physiological responses involved, making it a challenging system to fully comprehend and study (Figure 9). The observed synchronization of heart rate and blood pressure fluctuations suggests that slow yogic breathing with respiratory resistance, such as Ujjayi pranayama, activates and optimizes the baroreflex response in individuals with SCI.

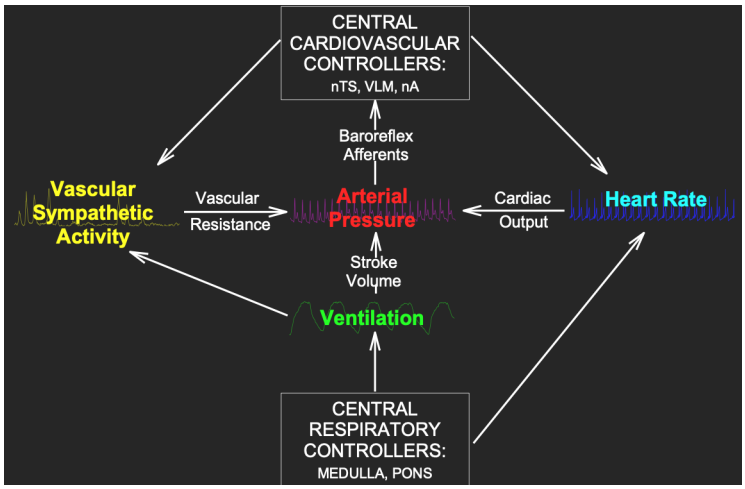


Figure 9: Schematic containing the cardiovascular and respiratory controllers of Arterial Pressure, Heart Rat, and Vascular Sympathetic Activity, including the Baroreflex afferents. (From dr. J Andrew Taylor with permission).

Ujjayi pranayama generates resistance by inducing a slight constriction of the throat muscles causing an increase in intrathoracic pressure, which in turn leads to enhanced baroreceptor activation. This increased activation of the baroreceptors, coupled with the slower respiratory rate and the associated autonomic response, could result in a more robust baroreflex-mediated control of heart rate and blood pressure. This speculation is supported by previous studies demonstrating the potential of slow yogic breathing to influence baroreflex function and improve cardiovascular regulation in various populations (29, 58-60).

It is possible that the observed shifts in cardiovascular control towards a baroreflex-mediated feedback mechanism during slow yogic breathing with respiratory resistance contribute to the increased mean heart rate and its variability, as well as the improved ventilation-perfusion matching and systemic oxygen saturation. However, further research is needed to directly investigate the involvement of the baroreflex in the observed cardiovascular effects of slow yogic breathing in individuals with SCI. Such investigations could help elucidate the underlying mechanisms and provide valuable insights for the development of targeted interventions for improving cardiovascular health in this population.

4.2 — Limitations

Sample Size and Study Design

The study had a limited sample size, consisting of only 15 participants, which may restrict the generalizability of the findings to a larger population. Additionally, specific exclusion criteria, such as non-English speakers, certain medical histories, smoking, and use of cardioactive medications potentially limit the representativeness of the sample and the generalizability of the results. Moreover, the absence of a control group hinders the ability to differentiate the effects of the yogic-breathing intervention from natural variations or other confounding factors. The lack of blinding in the intervention may introduce bias, as both the participants and researchers were aware of the specific breathing techniques being practiced. Furthermore, the study's recruitment strategy from a single rehabilitation hospital program

may introduce selection bias, limiting the diversity of the sample and potentially impacting the external validity of the findings. Finally, the study lacked long-term follow-up to assess the sustainability and persistence of the observed changes in pulmonary function and cardiopulmonary variables.

Statistical Analysis

The statistical analysis used multiple t-tests without appropriate correction for multiple comparisons. Conducting multiple t-tests without adjusting for multiple comparisons increases the risk of obtaining false positive results (Type I error) due to chance alone. This can inflate the overall significance level of the study, leading to potentially erroneous conclusions and overestimation of the effectiveness of the breathing techniques. To mitigate this limitation, alternative statistical approaches such as Bonferroni correction, false discovery rate correction, or other suitable methods should be considered to account for multiple comparisons. Implementing these corrections would help maintain the appropriate overall significance level, reduce the likelihood of spurious findings, and enhance the validity and reliability of the study's conclusions.

4.3 — Future Directions for Respiratory Research and SCI

Several avenues for future research emerge from the present study's findings, highlighting areas of focus that can enhance our understanding of the therapeutic potential of YBTs in individuals with SCI. Firstly, qualitative data collection should be integrated into future investigations to delve into the complex nature of the SCI population, considering the interplay of physical, emotional, and socioeconomic factors that influence quality of life. Incorporating qualitative research methods is crucial because they provide in-depth understanding and contextual insights into participants' experiences and perceptions. Qualitative research allows for participant voices to be heard, explores new areas, and complements quantitative data by adding meaning and context to numerical findings. Overall, it enriches the research process by capturing nuanced insights and contributing to a comprehensive understanding of the topic under investigation.

Secondly, while the study examined four specific YBTs, it is crucial to explore the efficacy of other YBTs and modern forms of breathwork in improving respiratory function and cardiovascular health in SCI patients. There are dozens, if not hundreds, of different pranayama techniques documented in various yoga and meditation traditions and many more modern breathing techniques that have been developed since (61). Each technique may have its unique breathing patterns, ratios, and purposes, targeting different aspects of breath control Investigating a broader range of YBTs would provide a more comprehensive understanding of their diverse effects and expand the therapeutic options available.

Furthermore, future studies should incorporate control groups, including healthy individuals and other patient groups, to compare the effects of YBTs in SCI patients to healthy physiology. This comparative approach would shed light on the underlying physiology and the benefits of YBTs within the SCI population compared to other cohorts.

To assess the long-term impact of YBTs, further research should investigate the sustained effects of these techniques on cardiopulmonary variables and their interrelationships. Longitudinal studies would offer valuable insights into the durability of the observed improvements and elucidate the underlying mechanisms that contribute to the therapeutic benefits. Moreover, exploring the optimal training regimens for SCI patients is essential. This research should address both the acute phase following injury and long-term maintenance, considering factors such as frequency, duration, and intensity of YBT sessions. By tailoring training regimens to the specific needs of SCI patients, personalized respiratory rehabilitation strategies can be developed. Lastly, examining the potential synergistic effects of

combining YBTs with exercise regimens or specific medications is an area of interest. Exploring combination therapies could lead to enhanced respiratory function and cardiovascular health in SCI patients. Evaluating the integration of YBTs into comprehensive treatment approaches would pave the way for multifaceted and even more effective interventions. By addressing these research directions, we can advance our knowledge of YBTs' therapeutic potential, optimize their implementation in respiratory therapy regimens for individuals with SCI, and ultimately improve their overall well-being and health outcomes.

5. CONCLUSION

SCI is a devastating condition that significantly impacts numerous individuals each year, leading to severe respiratory impairments and complications. The primary cause of morbidity and mortality in SCI patients is respiratory insufficiency, highlighting the critical need to focus on improving respiratory function in this population. However, the interrelationships between the respiratory and cardiovascular systems in SCI patients have been inadequately researched. This study aimed to fill this research gap by examining the effects of YBTs on cardiovascular variabilities in individuals with SCI. The findings support the effectiveness of slow yogic breathing as a therapeutic intervention for respiratory rehabilitation, blood pressure management, and cardiovascular health in this population. The results demonstrate improvements in respiratory function, as evidenced by increased MIP, following a two-week familiarization with yogic breathing techniques. These findings align with previous research highlighting the positive impact of respiratory muscle training on respiratory performance. The findings of this study provide compelling evidence for the effectiveness of slow yogic breathing as a therapeutic intervention for respiratory rehabilitation, blood pressure management, and cardiovascular health in individuals with SCI. Specifically, the results demonstrated notable improvements in respiratory function, as indicated by increased MIP, following a two-week familiarization period with yogic breathing techniques. These findings align with previous research highlighting the positive impact of respiratory muscle training on respiratory performance. Furthermore, the study elucidates the interplay between the respiratory and cardiovascular systems in individuals with SCI, highlighting the potential benefits of yogic breathing techniques to enhance both domains. The practice of YBTs may help restore autonomic balance, mitigate cardiovascular dysregulation, and improve respiratory efficiency in SCI patients. This study underscores the promising avenue of non-pharmacological interventions, specifically YBTs, to enhance overall well-being and respiratory health in SCI patients. Further research is warranted to investigate the long-term effects and optimal implementation strategies of these techniques in respiratory therapy regimens for individuals with SCI, with the aim of reducing complications, premature mortality, and disability within this vulnerable population.

6. REFERENCES

1. Zimmer MB, Nantwi K, Goshgarian HG. Effect of Spinal Cord Injury on the Respiratory System: Basic Research and Current Clinical Treatment Options. J Spinal Cord Med. 2007;30:319-330. doi:10.1080/10790268.2007.11754564

2. Galeiras Vázquez R, Rascado Sedes P, Mourelo Fariña M, Montoto Marqués A, Ferreiro Velasco ME. Respiratory management in the patient with spinal cord injury. Biomed Res Int. 2013;2013:168757. doi:10.1155/2013/168757

3. Linn WS, Spungen AM, Gong H, Adkins RH, Bauman A, Waters RL. Forced vital capacity in two large outpatient populations with chronic spinal cord injury. Spinal Cord. 2001;39(5):263-268. doi:10.1038/SJ.SC.3101155

4. Paralyzed Veterans of America. Clinical practice guideline for the management of respiratory dysfunction, cardiovascular disease, and related conditions in spinal cord injury. Washington, DC: Paralyzed Veterans of America; 2021. Available from: https://pva.org/wp-content/uploads/2021/09/cpg_resmgmt.pdf. Accessed May 11, 2023.

5. Winslow C, Rozovsky J. Effect of spinal cord injury on the respiratory system. Am J Phys Med Rehabil. 2003;82(10):803-814. doi:10.1097/01.PHM.0000078184.08835.01

6. Berlowitz DJ, Wadsworth B, Ross J. Respiratory problems and management in people with spinal cord injury. Breathe. 2016;12(4):328-340. doi:10.1183/20734735.012616

7. Van Silfhout L, Peters AEJ, Berlowitz DJ, Schembri R, Thijssen D, Graco M. Long-term change in respiratory function following spinal cord injury. Spinal Cord. 2016;54(9):714-719. doi:10.1038/SC.2015.233

8. Mueller G, de Groot S, van der Woude L, Hopman MTE. Time-courses of lung function and respiratory muscle pressure generating capacity after spinal cord injury: a prospective cohort study. J Rehabil Med. 2008;40(4):269-276. doi:10.2340/16501977-0162

9. Ledsome JR, Sharp JM. Pulmonary function in acute cervical cord injury. Am Rev Respir Dis. 1981;124(1):41-44. doi:10.1164/arrd.1981.124.1.41

10. Baydur A, Adkins RH, Milic-Emili J. Lung mechanics in individuals with spinal cord injury: effects of injury level and posture. J Appl Physiol. 2001;90(2):405-411. doi:10.1152/jappl.2001.90.2.405Morgan, M. D. L., Gourlay, A. R., Silver, J. R., Williams, S. J., & Denison, D. M. (1985). Contribution of the rib cage to breathing in tetraplegia. Thorax, 40(8), 613–617. https://doi.org/10.1136/THX.40.8.613

11. Morgan MDL, Gourlay AR, Silver JR, Williams SJ, Denison DM. Contribution of the rib cage to breathing in tetraplegia. Thorax. 1985;40(8):613-617. doi:10.1136/THX.40.8.613

12. Haas F, Axen K, Pineda H, Gandino D, Haas A. Temporal pulmonary function changes in cervical cord injury. Arch Phys Med Rehabil. 1985 Mar;66(3):139-44. doi: 10.5555/URI:PII:000399938590560X. PMID: 3978344.

13. Forner JV. Lung volumes and mechanics of breathing in tetraplegics. Spinal Cord. 1980 Apr;18(4):258-66. doi: 10.1038/SC.1980.45. PMID: 7416345.

14. Loveridge B, Sanii R, Dubo HI. Breathing pattern adjustments during the first year following cervical spinal cord injury. Paraplegia. 1992 Jul;30(7):479-88. doi: 10.1038/SC.1992.102. PMID: 1517147.

15. Leith DE, Bradley M. Ventilatory muscle strength and endurance training. J Appl Physiol. 1976 Oct;41(4):508-16. doi: 10.1152/jappl.1976.41.4.508. PMID: 993238.

16. Gross D, Ladd HW, Riley EJ, Macklem PT, Grassino A. The effect of training on strength and endurance of the diaphragm in quadriplegia.

17. Kogan I, McCool FD, Liberman SL, Garshick E, Shannon K, Frisbee JH, Brown R. Diaphragm hypertrophy during inspiratory muscle training in tetraplegia (abstract). Am J Respir Crit Care Med. 1996;153(4):A25. doi: 10.1164/ajrccm.153.4.8616557.

18. Rutchik A, Weissman AR, Almenoff PL, Spungen AM, Bauman WA, Grimm DR. Resistive inspiratory muscle training in subjects with chronic cervical spinal cord injury. Arch Phys Med Rehabil. 1998 Mar;79(3):293-7. doi: 10.1016/S0003-9993(98)90009-0. PMID: 9523785.

19. Uijl SG, Houtman S, Folgering HT, Hopman MT. Training of the respiratory muscles in individuals with tetraplegia. Spinal Cord. 1999 Aug;37(8):575-9. doi: 10.1038/sj.sc.3100887. PMID: 10478658.

20. Loke J, Mahler DA, Virgulto JA. Respiratory muscle fatigue after marathon running. J Appl Physiol Respir Environ Exerc Physiol. 1982 Apr;52(4):821-4. doi: 10.1152/jappl.1982.52.4.821. PMID: 7076068.

21. Bissett B, Gosselink R, van Haren FMP. Respiratory Muscle Rehabilitation in Patients with Prolonged Mechanical Ventilation: A Targeted Approach. Crit Care. 2020 Mar 24;24(1):103. doi: 10.1186/s13054-020-2783-0. PMID: 32204719; PMCID: PMC7092518.

22. Brown R, DiMarco AF, Hoit JD, Garshick E. Respiratory Dysfunction and Management in Spinal Cord Injury. Respir Care. 2006 Aug;51(8):853. doi: 10.1007/978-981-19-0228-4_23. PMID: 16867136.

23. Saoji AA, Raghavendra BR, Manjunath NK. Immediate effects of yoga breathing with intermittent breath retention on the autonomic and cardiovascular variables amongst healthy volunteers. Indian J Physiol Pharmacol. 2018 Jan-Mar;62(1):41-50. doi: 10.1016/j.jaim.2018.06.009. PMID: 31041462.

24. Saoji AA, Raghavendra BR, Manjunath NK. Effects of yogic breath regulation: A narrative review of scientific evidence. J Ayurveda Integr Med. 2019 Apr-Jun;10(2):50-58. doi: 10.1016/j.jaim.2019.02.002. PMID: 31005339.

25. Raghuraj P, Telles S. Immediate effect of specific nostril manipulating yoga breathing practices on autonomic and respiratory variables. Appl Psychophysiol Biofeedback. 2008 Mar;33(1):65-75. doi: 10.1007/s10484-008-9055-7. PMID: 18299933.

26. Sharma VK, Trakroo M, Subramaniam V, Rajajeyakumar M, Bhavanani AB, Sahai A. Effect of fast and slow pranayama on perceived stress and cardiovascular parameters in young health-care students. Int J Yoga. 2013 Jan;6(1):104-10. doi: 10.4103/0973-6131.105935. PMID: 23440614; PMCID: PMC3573542.

27. Pramanik T, Pudasaini B, Prajapati R. Immediate effect of a slow pace breathing exercise Bhramari pranayama on blood pressure and heart rate. Nepal Med Coll J. 2010 Dec;12(4):154-7. PMID: 21932686.

28. Nivethitha L, Mooventhan A, Manjunath NK. Evaluation of cardiovascular functions during the practice of different types of yogic breathing techniques. Int J Yoga. 2021 Jul-Dec;14(2):158-162. doi: 10.4103/ijoy.ijoy_119_20. PMID: 34295270; PMCID: PMC8300326.

29. Bernardi L, Porta C, Spicuzza L, Bellwon J, Spadacini G, Frey AW, Yeung LY, Sanderson JE, Pedretti R, Tramarin R. Slow breathing increases arterial baroreflex sensitivity in patients with chronic heart failure. Circulation. 2002 Jan 15;105(2):143-5. doi: 10.1161/hc0202.103617. PMID: 11790712.

30. Joseph CN, Porta C, Casucci G, Casiraghi N, Maffeis M, Rossi M, Bernardi L. Slow breathing improves arterial baroreflex sensitivity and decreases blood pressure in essential hypertension. Hypertension. 2005 Sep;46(3):714-8. doi: 10.1161/01.HYP.0000179581.68566.7d. PMID: 16087721.

31. Naughton MT, Rahman MA, Jamal M, Bradley TD. Respiratory correlates of muscle sympathetic nerve activity in heart failure. Clin Sci (Lond). 1998;95(3):277-285. doi:10.1042/cs0950277

32. Goso Y, Asanoi H, Ichise H, Kameyama T, Hirai T, Nozawa T, Takashima S, Umeno K, Inoue H. Respiratory modulation of muscle sympathetic nerve activity in patients with chronic heart failure. Circulation. 2001;104(4):418-423. doi:10.1161/hc2901.092250

33. Parati G, Mancia G, Di Rienzo M, Castiglioni P, Taylor JA, Studinger P. Point: Counterpoint: Cardiovascular variability is/is not an index of autonomic control of circulation. J Appl Physiol (1985). 2006;101(2):676-682. doi:10.1152/japplphysiol.00446.2006

34. Taylor JA. Autonomic consequences of spinal cord injury. Auton Neurosci. 2018;209:1-3. doi:10.1016/j.autneu.2017.09.015

35. Draghici AE, Taylor JA. Baroreflex autonomic control in human spinal cord injury: Physiology, measurement, and potential alterations. Auton Neurosci. 2018;209:37-42. doi:10.1016/j.autneu.2017.08.007

36. Vivodtzev I, Taylor JA. Cardiac, autonomic and cardiometabolic impact of exercise training in spinal cord injury: A qualitative review. J Cardiopulm Rehabil Prev. 2021;41(1):6-13. doi:10.1097/HCR.0000000000000564

37. Buker DB, Oyarce CC, Plaza RS. Effects of Spinal Cord Injury in Heart Rate Variability After Acute and Chronic Exercise: A Systematic Review. Top Spinal Cord Inj Rehabil. 2018;24(2):167-176. doi:10.1310/sci17-00028

38. Szabó BM, van Veldhuisen DJ, Brouwer J, Haaksma J, Lie KI. Relation between severity of disease and impairment of heart rate variability parameters in patients with chronic congestive heart failure secondary to coronary artery disease. Am J Cardiol. 1995;76(10):713-716. doi:10.1016/s0002-9149(99)80204-5

39. Lahiri MK, Kannankeril PJ, Goldberger JJ. Assessment of Autonomic Function in Cardiovascular Disease: Physiological Basis and Prognostic Implications. J Am Coll Cardiol. 2008;51(18):1725-1733. doi:10.1016/j.jacc.2008.01.038

40. Haensel A, Mills PJ, Nelesen RA, Ziegler MG, Dimsdale JE. The relationship between heart rate variability and inflammatory markers in cardiovascular diseases. Psychoneuroendocrinology. 2008;33(10):1305-1312. doi:10.1016/j.psyneuen.2008.08.007

41. Solinsky R, Vivodtzev I, Hamner JW, Taylor JA. The effect of heart rate variability on blood pressure is augmented in spinal cord injury and is unaltered by exercise training. Clin Auton Res. 2021 Apr;31(2):293-301. doi: 10.1007/s10286-020-00677-2. PMID: 32892290.

42. Taylor JA, Myers CW, Halliwill JR, Seidel H, Eckberg DL. Sympathetic restraint of respiratory sinus arrhythmia: implications for vagal-cardiac tone assessment in humans. Am J Physiol Heart Circ Physiol. 2001 Jun;280(6):H2804-14. doi: 10.1152/ajpheart.2001.280.6.H2804. PMID: 11356643.

43. Cooke WH, Cox JF, Diedrich AM, et al. Controlled breathing protocols probe human autonomic cardiovascular rhythms. Am J Physiol. 1998 Feb;274(2 Pt 2):H709-18. doi: 10.1152/ajpheart.1998.274.2.H709. PMID: 9486238.

44. Russo MA, Santarelli DM, O'Rourke D. The physiological effects of slow breathing in the healthy human. Breathe (Sheff). 2017 Dec;13(4):298-309. doi: 10.1183/20734735.009817. PMID: 29225932; PMCID: PMC5720762.

45. Li C, Chang Q, Zhang J, Chai W. Effects of slow breathing rate on heart rate variability and arterial baroreflex sensitivity in essential hypertension. Medicine (Baltimore). 2018 May;97(18):e10639. doi: 10.1097/MD.0000000000010639. PMID: 29718808; PMCID: PMC5958124.

46. Stancak Jr A, Kuna N, Novak P, et al. Observations on respiratory and cardiovascular rhymicities during yogic-high frequency respiration. Physiol Res. 1991;40(4):345-54. PMID: 1837113.

47. Peng CK, Henry IC, Mietus JE, et al. Heart rate dynamics during three forms of meditation. Int J Cardiol. 2004 Feb;95(1):19-27. doi: 10.1016/j.ijcard.2003.02.006. PMID: 15066718.

48. Schein MH, Gavish B, Herz M, et al. Treating hypertension with a device that slows and regularizes breathing: a randomized, double-blind controlled study. J Hum Hypertens. 2001 Apr;15(4):271-8. doi: 10.1038/sj.jhh.1001163. PMID: 11349820.

49. Grossman E, Grossman A, Schein MH, et al. Breathing control lowers blood pressure. J Hum Hypertens. 2001 Apr;15(4):263-9. doi: 10.1038/sj.jhh.1001161. PMID: 11349819.

50. Elliott WJ, Izzo JL Jr, White WB, et al. Graded blood pressure reduction in hypertensive outpatients associated with use of a device to assist with slow breathing. J Clin Hypertens (Greenwich). 2004 Oct;6(10):553-9. doi: 10.1111/j.152

51. Kelley A, Garshick E, Gross ER, Lieberman SL, Tun CG, et al. (2003) Spirometry testing standards in spinal cord injury. Chest. 123: 725-730. doi: 10.1378/chest.123.3.725.

52. Holmes GM, Wynnie PM, Carlton SM. Respiratory motor control disrupted by spinal cord injury: mechanisms, evaluation, and restoration. Respir Physiol Neurobiol. 2011 Jun 30;179(1):47-56. doi: 10.1007/s12975-011-0114-0. PMID: 22408690; PMCID: PMC3297359;

53. Schilero GJ, Spungen AM, Bauman WA, Radulovic M, Lesser M. Pulmonary function and spinal cord injury. Respir Physiol Neurobiol. 2009 May 15;166(3):129-41. doi: 10.1016/j.resp.2009.04.002. Epub 2009 Apr 9. PMID: 19442929.

54. Schilero GJ, Bauman WA, Radulovic M. Traumatic Spinal Cord Injury: Pulmonary Physiologic Principles and Management. Clin Chest Med. 2018 Jun;39(2):411-425. doi: 10.1016/j.ccm.2018.02.002. PMID: 29779599.

55. Jerath R, Beveridge C, Barnes V. Self-regulation of breathing as an adjunctive treatment of insomnia. Front Psychiatry. 2019;10:935. doi: 10.3389/fpsyt.2019.00935

56. Bernardi L, Gabutti A, Porta C, Spicuzza L. Slow breathing reduces chemoreflex response to hypoxia and hypercapnia, and increases baroreflex sensitivity. J Hypertens. 2017;35(6):1305-1312. doi: 10.1097/HJH.0000000000001302

57. Tyagi A, Cohen M. Yoga and heart rate variability: A comprehensive review of the literature. Int J Yoga. 2016;9(2):97-113. doi: 10.4103/0973-6131.183712

58. Pal R, Gupta N. Yoga and heart rate variability: A comprehensive review of literature. Int J Clin Exp Physiol. 2015;2(3):147-152. doi: 10.4103/2348-8093.157912

59. Pal GK, Velkumary S, Madanmohan. Effect of short-term practice of breathing exercises on autonomic functions in normal human volunteers. Indian J Med Res. 2004 Feb;120(2):115-121. PMID: 15347854.

60. Mishra SK, Singh P, Bunch SJ, Zhang R. The effect of slow breathing on cardiovascular and autonomic parameters in patients with chronic atrial fibrillation. J Atr Fibrillation. 2018 Jun 30;11(3):2005. doi: 10.4022/jafib.2005. PMID: 31384319; PMCID: PMC6667383.

61. Balban MY, Neri E, Kogon MM, Weed L, Nouriani B, Jo B, Holl G, Zeitzer JM, Spiegel D, Huberman AD. Brief structured respiration practices enhance mood and reduce physiological arousal. Cell Rep Med. 2023 Jan 17;4(1):100895. doi: 10.1016/j.xcrm.2022.100895. Epub 2023 Jan 10. PMID: 36630953; PMCID: PMC9873947.

7. APPENDICES

7.1 — Data Tables

Table 3: Average maximal inspiratory and expiratory mouth pressures (MIP and MEP) before and after coaching and self-practice, including standard deviations and absolute change.

Maximal Inspiratory and Expiratory Mouth Pressures

Subjects (N = 12)	BEFORE COACHING				AFTER COACHING				Δ AVG MIP (cmH2O)	Δ AVG MEP (cmH2O)
	AVG MIP (cmH2O)	SD (cmH2O)	AVG MEP (cmH2O)	SD (cmH2O)	AVG MIP (cmH2O)	SD (cmH2O)	AVG MEP (cmH2O)	SD (cmH2O)		
1	86.0	3.0	47.0	15.1	98.3	3.1	53.3	5.8	12.3	6.3
2	95.8	8.5	103.7	5.5	113.5	16.0	90.3	15.4	17.8	-13.4
3	93.3	13.1	84.7	7.5	98.8	13.2	89.5	7.3	5.5	4.8
4	121.0	8.0	71.0	6.6	127.0	6.8	63.0	7.0	6.0	-8.0
5	141.3	11.4	73.3	10.9	145.0	3.5	88.7	1.5	3.8	15.4
6	105.3	7.4	75.3	2.1	113.8	6.4	70.0	3.2	8.5	-5.3
7	68.5	4.1	30.3	1.0	77.3	1.5	34.0	3.5	8.8	3.8
8	103.3	10.3	62.0	7.0	113.0	1.0	61.8	11.2	9.8	-0.3
9	102.8	5.5	46.5	6.2	120.8	5.4	53.3	2.9	18.0	6.8
10	56.0	5.0	56.0	4.2	65.0	11.3	36.5	3.0	9.0	-19.5
11	146.7	5.8	100.5	10.1	169.0	4.6	93.3	10.7	22.3	-7.3
12	64.8	7.9	56.8	6.4	45.8	5.4	39.5	3.4	-19.0	-17.3

Table 4: Change in overall maximal inspiratory and expiratory pressures calculated by group absolute change.

Overall Change In MIP & MEP

Test	MIP (cmH2O)	SD (cmH2O)	MEP (cmH2O)	SD (cmH2O)
Group Δ	8.6	10.3	-2.8	10.7
T-Test	0.01*		0.4	

Table 5: Group average mean Systolic Blood Pressure (SBP), mean Diastolic Blood Pressure (DBP), mean Heart Rate, and mean Heart Rate Variability, mean $EtCO_2$, and SaO_2 and Standard Deviations during SCOGA YBTs.

Cardiopulmonary Averages & Standard Deviations

Breathing Pattern (N = 12)	mean SBP (mmHg)	SD SBP (mmHg)	mean DBP (mmHg)	SD DBP (mmHg)	mean RRi (mSec)	SD RRi (mSec)	mean HR (bpm)	SD HR (bpm)	mean $EtCO_2$ (mmHg)	SD $EtCO_2$ (mmHg)	mean SaO_2 (%)	SD SaO_2 (%)
Unpaced	113.1	5.4	62.5	2.8	1074.0	69.8	57.5	3.9	35.2	3.7	96.1	1.6
0.25Hz	114.9	5.1	64.3	2.7	1070.9	62.4	57.4	3.2	31.4	5.1	96.9	1.6
0.083Hz	112.0	4.3	63.2	2.6	1024.9	76.9	59.9	4.5	36.3	4.0	97.0	1.1
0.083Hz Ujjayi	111.0	5.6	63.0	3.3	1001.6	78.9	61.4	4.9	32.3	5.5	97.7	1.5
0.083Hz Box	115.9	4.7	65.8	2.7	1031.4	75.7	60.0	4.5	36.9	4.9	96.6	1.4
0.083Hz 1:2	115.5	4.9	65.1	2.8	1056.3	65.5	58.2	3.5	35.8	4.4	96.7	2.1

*Table 6 Statistical Comparison of YBTs versus unpaced breathing for cardiopulmonary variables. Significance was set at α=0.05 (indicated by *).*

Cardiopulmonary Averages & Standard Deviations Statistics

Breathing Pattern Comparisons	mean SBP	SD SBP	mean DBP	SD DBP	mean Rri	SD RRi	mean HR	SD HR	mean $EtCO_2$	mean SaO_2
0.25Hz vs Unpaced	0.83	0.70	0.61	0.92	0.90	0.14	0.84	0.08	**0.001***	0.17
0.083Hz vs Unpaced	0.58	0.42	0.28	0.25	0.23	0.98	0.35	0.95	0.20	**0.01***
0.083Hz Ujjayi vs Unpaced	0.68	0.82	0.54	0.36	**0.01***	0.11	**0.01***	**0.02***	**0.03***	**0.01***
0.083Hz Box vs Unpaced	0.89	0.76	0.63	0.97	0.12	0.77	0.13	0.63	0.11	0.38
0.083Hz 1:2 vs Unpaced	0.44	0.45	0.29	0.77	0.06	0.42	0.07	0.46	0.48	0.26

Table 7: Systolic Pressure and RR Interval Variabilities – Powers and Cross Spectral Density during SCOGA YBTs

Power Spectral Density & Cross-Spectrum					
Breathing Pattern (N = 12)	Power mean SBP (mmHg/Hz)	Power mean RRi (mSec/Hz)	Coherence	Phase (degrees)	Gain
Unpaced	3.15	1845.4	0.71	-4.23	19.06
0.25Hz	1.31	608.6	0.87	43.22	19.36
0.083Hz	4.81	2253.7	0.88	-44.70	24.27
Ujjayi	13.86	4832.5	0.85	-43.51	18.03
Box	6.40	2317.7	0.90	-49.84	18.96
Ex 1:2	5.96	3444.2	0.88	-34.20	22.83

*Table 8: Statistical Comparison of YBTs versus unpaced breathing for Systolic Blood Pressure and RR interval, Coherence, Phase, and Gain. Significance was set at α=0.05 (indicated by *).*

Power Spectrum & Cross-Spectrum Statistics					
Breathing Pattern Comparisons	Power mean SBP	Power mean RRi	Coherence	Phase	Gain
0.25Hz vs Unpaced	0.04*	0.01*	0.01*	0.000002*	0.92
0.083Hz vs Unpaced	0.39	0.50	0.004*	0.00001*	0.07
0.083Hz Ujjayi vs Unpaced	0.03*	0.01*	0.008*	0.0005*	0.72
0.083Hz Box vs Unpaced	0.10	0.56	0.0007*	0.00004*	0.96
0.083Hz 1:2 vs Unpaced	0.10	0.12	0.006*	0.0005*	0.18

7.2 — MATLAB Code (written by MM)

```
maxsubjects = 15;
numpatterns = 5;

data_exist = logical([
    1 %001
    1 %002
    1 %003
    0 %004
    1 %005
    1 %006
    1 %007
    1 %008
    1 %009
    1 %010
    0 %011
    1 %012
    1 %013
    0 %014
    1 %015
    ]);

Column_Names = {'mSBP','stdSBP','mDBP','stdDBP','mRRi','stdRRi','mHR','stdHR',...
    'P_RespC','P_RespB','P_SBP','P_DBP','P_RRi',...
    'SBP2RRi-Coh','SBP2RRi-Phase','SBP2RRi-Gain'...
    'chest2belly-Coh','chest2belly-Phase','chest2belly-Gain'...
    'chest2SBP-Coh','chest2SBP-Phase','chest2SBP-Gain'...
    'belly2SBP-Coh','belly2SBP-Phase','belly2SBP-Gain'};

forest_green_RGB = [34 139 34]/255;

data_array = NaN(maxsubjects, length(Column_Names), numpatterns);


for k = 1:maxsubjects
    for n = 1:numpatterns

        if data_exist(k)
```

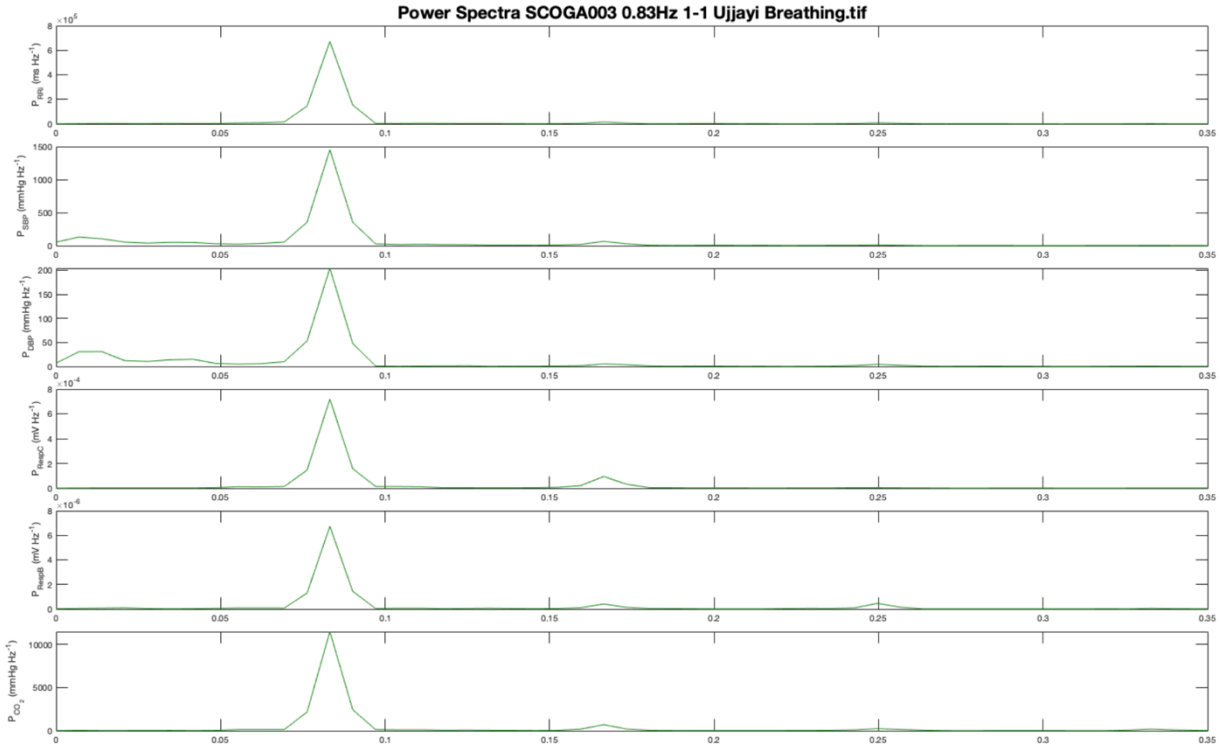
```
scogaID         =    k; %%% name chooses practitioner
scogaptrn       =    n; %%% condition chooses breathing variant

if scogaptrn == 1
    breathpattern = '0.83Hz 1-1 Breathing'; %slow
elseif scogaptrn == 2
    breathpattern = '0.83Hz 1-1 Ujjayi Breathing'; %ujjayi
elseif scogaptrn == 3
    breathpattern = '0.83Hz 1-1-1-1 Box Breathing'; %box
elseif scogaptrn == 4
    breathpattern = '0.83Hz 1-2 Breathing'; %slow12
elseif scogaptrn == 5
    breathpattern = '0.25Hz 1-1 Breathing'; %fast
elseif scogaptrn == 6
    breathpattern = 'Unpaced Breathing'; %unpaced
end


% calls function filenamegrab and loads concatenated filename
fnamefull = filenamegrabSCOGAarray(scogaID,scogaptrn);
load(fnamefull)

% Channel Settings: the order channels are imported from PowerLab
CO2_Chan    = 1;
ECG_Chan    = 2;
BP_Chan     = 3;
RespB_Chan  = 4;
RespC_Chan  = 5;
BBF_Chan    = 6;
Mphone_Chan = 7;    %% microphone sampled @ 20KHz
Sa02_Chan   = 8;

SBP_Chan    = 9;
DBP_Chan    = 10;
HR_Chan     = 11;

% LabChart can store channels at different samplerates
SR = 20000; % base sample rate at which lower sample rates are indexed

CO2     =  data(datastart(CO2_Chan):dataend(CO2_Chan));
ECG     =  data(datastart(ECG_Chan):dataend(ECG_Chan));
BP      =  data(datastart(BP_Chan):dataend(BP_Chan));
RespB   =  data(datastart(RespB_Chan):dataend(RespB_Chan));
RespC   =  data(datastart(RespC_Chan):dataend(RespC_Chan));
BBF     =  data(datastart(BBF_Chan):dataend(BBF_Chan));
Mphone  =  data(datastart(Mphone_Chan):dataend(Mphone_Chan));
SaO2    =  data(datastart(Sa02_Chan):dataend(Sa02_Chan));

HR      =  data(datastart(HR_Chan):dataend(HR_Chan));

% timeseries t (original), t @ 100Hz, & t @ 1000Hz
t           =   1/SR:1/SR:(length(CO2))/SR; % old code still necessary for original plots
t100Hz      =   1/samplerate(BP_Chan):1/samplerate(BP_Chan):(length(BP))/samplerate(BP_Chan);
t1K         =   1/samplerate(ECG_Chan):1/samplerate(ECG_Chan):length(ECG)/samplerate(ECG_Chan);
R_ECG       =   tickrate/samplerate(ECG_Chan);
iRRi        =   com((com(:,1)==ECG_Chan) & (com(:,4)==2), 3);
iRRi_1k     =   round(iRRi/R_ECG); % RR interval to beatquency
tRRi        =   t1K(iRRi_1k);
RRi         =   diff(tRRi)*1000;
tRRi(end)   =   [];  % diff is tRRi - 1 so drop last value

% SBP beat-by-beat
R_BP        =   tickrate/samplerate(BP_Chan);
iSBP        =   com((com(:,1)==BP_Chan) & (com(:,4)==2), 3);
iSBP_1k     =   round(iSBP/R_BP);
tSBP        =   t100Hz(iSBP_1k);
SBP         =   BP(iSBP_1k);

% DBP beat-by-beat
DBP = zeros(1,length(SBP));
iDBP_1k = zeros(length(iSBP_1k),1);
[DBP(1),iDBP_1k(1)] = min(BP(1:BP(iSBP_1k)));
for g = 1:length(iSBP_1k)-1
    [DBP(g+1),iDBP_1k(g+1)] = min(BP(iSBP_1k(g):iSBP_1k(g+1)));
    iDBP_1k(g+1) = sum(iDBP_1k(g+1) + iSBP_1k(g));
end
tDBP = t100Hz(iDBP_1k);

% code for interpolation
SRdec       =   5; % target samplerate
tstart      =   max([tRRi(1) tSBP(1)]);
tend        =   min ([tRRi(end) tSBP(end)]);
tInterp     =   tstart:1/SRdec:tend;
RRi_interp  =   interp1(tRRi,RRi,tInterp,'linear');
SBP_interp  =   interp1(tSBP,SBP,tInterp,'linear');
DBP_interp  =   interp1(tDBP,DBP,tInterp,'linear');
DBP_interp(1) = [];
```

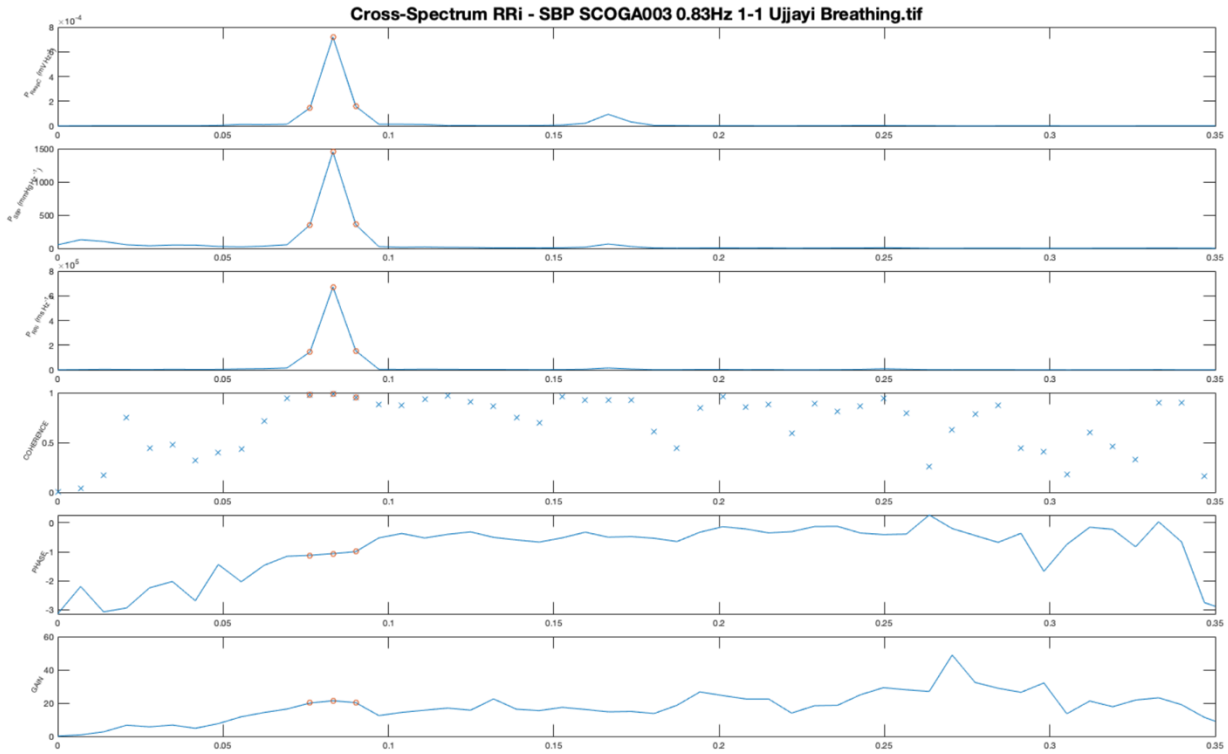
```
CO2_dec = resample(CO2 - mean(CO2),1, samplerate(CO2_Chan)/SRdec) + mean(CO2);
RespC_dec = resample(RespC - mean(RespC),1, samplerate(RespC_Chan)/SRdec) + mean(RespC);
RespB_dec = resample(RespB - mean(RespB),1, samplerate(RespB_Chan)/SRdec) + mean(RespB);

%detrend interpolated and decimated data
RRi_interp_dt   = detrend(RRi_interp,'linear');
SBP_interp_dt   = detrend(SBP_interp,'linear');
DBP_interp_dt   = detrend(DBP_interp,'linear');
RespC_dec_dt    = detrend(RespC_dec,'linear');
RespB_dec_dt    = detrend(RespB_dec,'linear');
CO2_dec_dt      = detrend(CO2_dec,'linear');

%Standardize time-series
t_dec  = 1/SRdec:1/SRdec:length(RespC_dec)/SRdec;
ikeep = (t_dec>=tstart)&(t_dec<=tend);
CO2_dec_dt = CO2_dec_dt(ikeep);
RespB_dec_dt = RespB_dec_dt(ikeep);
RespC_dec_dt = RespC_dec_dt(ikeep);

% All channels to same length
L = min([length(RRi_interp_dt) length(SBP_interp_dt) length(DBP_interp_dt) length(RespC_dec_dt) length(RespB_dec_dt)]);
RRi_interp_dt   = RRi_interp_dt(1:L);
SBP_interp_dt   = SBP_interp_dt(1:L);
DBP_interp_dt   = DBP_interp_dt(1:L);
RespC_dec_dt    = RespC_dec_dt(1:L);
RespB_dec_dt    = RespB_dec_dt(1:L);
CO2_dec_dt      = CO2_dec_dt(1:L);

% spectral analysis parameters (standard across studies)
nwin         = floor(length(RRi_interp_dt)/3);
overlap      = floor(nwin/2);
window       = hamming(nwin);

% Welch's Modified Periodogram method for power spectral density
[P_RRi, F]     = pwelch(RRi_interp_dt, window, overlap, nwin, SRdec);
P_SBP          = pwelch(SBP_interp_dt, window, overlap, nwin, SRdec);
P_DBP          = pwelch(DBP_interp_dt, window, overlap, nwin, SRdec);
P_RespC        = pwelch(RespC_dec_dt, window, overlap, nwin, SRdec);
P_RespB        = pwelch(RespB_dec_dt, window, overlap, nwin, SRdec);
P_CO2          = pwelch(CO2_dec_dt, window, overlap, nwin, SRdec);
```

```
figure(2)
set(gcf,'units','normalized','outerposition',[0 0 1 1],'Menubar','none');
clf

h1 = subplot(6,1,1);
plot(F,P_RRi,'color',forest_green_RGB)
fig2_title=['Power Spectra SCOGA00' num2str(scogaID) ' ' num2str(breathpattern) '.tif'];
title(['\fontsize{20}' fig2_title]);
ylabel('P_{RRi} (ms Hz^{-1})')
xlim([0 0.35])

h2 = subplot(6,1,2);
plot(F,P_SBP,'color',forest_green_RGB)
ylabel('P_{SBP} (mmHg Hz^{-1})')
xlim([0 0.35])

h3 = subplot(6,1,3);
plot(F,P_DBP,'color',forest_green_RGB)
ylabel('P_{DBP} (mmHg Hz^{-1})')
xlim([0 0.35])

h4 = subplot(6,1,4);
plot(F,P_RespC,'color',forest_green_RGB)
ylabel('P_{RespC} (mV Hz^{-1})')
xlim([0 0.35])

h5 = subplot(6,1,5);
plot(F,P_RespB,'color',forest_green_RGB)
ylabel('P_{RespB} (mV Hz^{-1})')
xlim([0 0.35])

h6 = subplot(6,1,6);
plot(F,P_CO2,'color',forest_green_RGB)
ylabel('P_{CO_{2}} (mmHg Hz^{-1})')
xlim([0 0.35])

set(h1,'pos',[0.05    0.83    0.92    0.13])
set(h2,'pos',[0.05    0.67    0.92    0.13])
set(h3,'pos',[0.05    0.51    0.92    0.13])
set(h4,'pos',[0.05    0.35    0.92    0.13])
set(h5,'pos',[0.05    0.19    0.92    0.13])
set(h6,'pos',[0.05    0.03    0.92    0.13])
saveas(gca,fig2_title,'tiff');

% Fourier transform SBP-RRi, chest2SBP, belly2SBP, chest2belly
TF_SBP2RRi = tfci(SBP_interp_dt,RRi_interp_dt,nwin,window,50,SRdec);
TF_DBP2RRi = tfci(DBP_interp_dt,RRi_interp_dt,nwin,window,50,SRdec);
TF_chest2SBP = tfci(RespC_dec_dt,SBP_interp_dt,nwin,window,50,SRdec);
TF_belly2SBP = tfci(RespB_dec_dt,SBP_interp_dt,nwin,window,50,SRdec);
TF_chest2belly = tfci(RespC_dec_dt,RespB_dec_dt,nwin,window,50,SRdec);

[~, lim83] = min(abs(F - 0.083));
[~, lim25] = min(abs(F - 0.25));
```

```
figure(3)
set(gcf,'units','normalized','outerposition',[0 0 1 1],'Menubar','none');
clf

h1 = subplot(6,1,1);
plot(F,P_RespC, F(RFI),P_RespC(RFI),'o')
fig3_title = ['Cross-Spectrum RRi ~ SBP SCOGA00' num2str(scogaID)...
    ' ' num2str(breathpattern) '.tif'];
title(['\fontsize{20}' fig3_title]);
ylabel('P_{RespC} (mV Hz^{-1})','FontSize',8)
xlim([0 0.35])
set(get(gca,'ylabel'),'Rotation',60)

h2 = subplot(6,1,2);
plot(F,P_SBP, F(RFI),P_SBP(RFI),'o')
ylabel('P_{SBP} (mmHg Hz^{-1})','FontSize',8)
xlim([0 0.35])
set(get(gca,'ylabel'),'Rotation',60)

h3 = subplot(6,1,3);
plot(F,P_RRi, F(RFI),P_RRi(RFI),'o')
ylabel('P_{RRi} (ms Hz^{-1})','FontSize',8)
xlim([0 0.35])
set(get(gca,'ylabel'),'Rotation',60)

h4 = subplot(6,1,4);
plot(TF_SBP2RRi.freq,TF_SBP2RRi.coh, 'x', F(RFI),TF_SBP2RRi.coh(RFI),'o')
ylabel('COHERENCE','FontSize',8)
xlim([0 0.35]) %% in the future chop data below 0.02Hz
set(get(gca,'ylabel'),'Rotation',60)

h5 = subplot(6,1,5);
plot(TF_SBP2RRi.freq,TF_SBP2RRi.phase, F(RFI),TF_SBP2RRi.phase(RFI),'o')
ylabel('PHASE','FontSize',8)
xlim([0 0.35])
set(get(gca,'ylabel'),'Rotation',60)

h6 = subplot(6,1,6);
plot(TF_SBP2RRi.freq,TF_SBP2RRi.gain, F(RFI),TF_SBP2RRi.gain(RFI),'o')
ylabel('GAIN','FontSize',8)
xlim([0 0.35])
set(get(gca,'ylabel'),'Rotation',60)

set(h1,'pos',[0.05    0.83    0.92    0.13])
set(h2,'pos',[0.05    0.67    0.92    0.13])
set(h3,'pos',[0.05    0.51    0.92    0.13])
set(h4,'pos',[0.05    0.35    0.92    0.13])
set(h5,'pos',[0.05    0.19    0.92    0.13])
set(h6,'pos',[0.05    0.03    0.92    0.13])
saveas(gca,fig3_title,'tiff');
```

7.3 — Example of MATLAB Power Spectrum Analysis & Cross-Spectrum




Cross-Spectrum RRi - SBP SCOGA003 0.83Hz 1-1 Ujjayi Breathing.tif
COHERENCE
PHASE
GAIN