

## Chapter 6

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# Results

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## **6.0 RESULTS**

The results of the two studies conducted during this thesis work are described below. (i) The changes in the autonomic and cardiovascular variables before and immediately following YBH and control sessions. (ii) Performance in the stop-signal task at baseline and following YBH and control sessions.

### **6.1 AUTONOMIC AND CARDIOVASCULAR VARIABLES**

#### **6.1.1 RECAPITULATION**

The autonomic and cardiovascular variables were recorded in thirty nine participants before and immediately following the YBH and control sessions. The following data were extracted from the 16-channel polygraph.

The variables assessed were the frequency and time domain analysis of heart rate variability (HRV), heart rate, respiratory rate and the cardiovascular measures assessed using the Finapres non-invasive blood pressure monitoring system. Heart rate in beats per minute was calculated by counting the R waves of the QRS complex in the ECG. Frequency domain analysis of HRV revealed the following specific frequency bands were studied viz., Low-frequency (LF) band (0.04–0.15 Hz) and high-frequency (HF) band (0.15–0.5 Hz). The LF/HF ratio was also calculated. The following components of time domain HRV were analyzed: (i) the standard deviation of NN intervals (SDNN), (ii) the square root of the mean of the sum of the squares of differences between adjacent NN intervals (RMSSD), (iii) the proportion derived by dividing NN50 by the total number of NN intervals (pNN50). The respiratory rate in cycles per minute (cpm) was calculated by counting the total breath cycles.

Brachial artery systolic (SBP) and diastolic pressures (DBP), mean arterial pressure (MAP) were expressed in mmHg. The computed measurements of Stroke volume (SV) was measured in ml, cardiac output (CO) in liter/min. the Total Peripheral resistance (TPR) was expressed in IU and Baroreflex Sensitivity (BRS) in ms/mmHg.

Repeated measures ANOVAs were carried out for each variable. This was followed by *post hoc* analyses with Bonferroni adjustment for multiple comparisons between the mean values of post values with that of the respective pre states. The group mean values  $\pm$  S.D. are given in **Table 8**. The actual data of cardiovascular and autonomic variables of individual participants is given in **Appendix 3**.

**Table 8: Changes in the Autonomic and Cardiovascular Variables before and following the Experimental and Control Sessions. (n= 39)**

	Experimental Session		Control Session	
	Pre	Post	Pre	Post
Heart rate (beats/min)	78.54±10.54	74.92±9.25***	76.67±9.58	73.94±9.29**
SDNN (ms)	64.47±26.27	74.76±29.20**	65.67±28.02	70.84±28.57
RMSSD (ms)	45.98±22.90	52.39±24.96***	49.23±23.22	54.81±23.93*
pNN50 (% units)	21.26±16.65	24.99±16.49**	25.20±17.78	30.67±17.75**
LFnU	61.72±17.55	67.51±15.77*	57.38±18.96	55.56±20.55
HFnU	38.53±17.16	32.65±15.27*	42.48±18.57	43.71±19.65
LF:HF	2.58±2.72	3.10±2.70	2.07±1.96	2.11±2.59
Respiratory Rate (cycles/min)	15.45±3.21	14.78±3.46	16.13±3.09	15.38±2.98
Systolic BP (mmHg)	102.10±12.21	101.04±11.39	104.44±10.91	106.83±11.86***
Diastolic BP (mmHg)	59.33±8.76	58.49±8.30	60.88±8.46	60.61±8.23
Mean Arterial pressure (mmHg)	77.88±10.12	76.35±9.54*	79.52±9.10	79.59±8.99
Stroke Volume (ml)	68.35±15.83	66.20±15.75*	70.36±12.04	72.22±11.41***
Cardiac output (l/min)	5.27±1.20	4.88±1.05***	5.32±0.85	5.26±0.81**
Total Peripheral Resistance	0.99±0.37	1.04±0.39***	1.07±0.70	0.99±0.36
Baroreflex Sensitivity (ms/mmHg)	14.56±5.55	15.81±5.71**	16.24±8.83	16.50± 7.75

Repeated Measures Analyses of Variance with post hoc Bonferoni adjustment,

\*=p<0.05, \*\*=p<0.01, \*\*\*=p<0.001

SDNN: standard deviation of NN intervals; RMSSD: root of the mean of the sum of the squares of differences between adjacent NN intervals; pNN50: proportion derived by dividing NN50 by the total number of NN intervals.

### 6.1.2 HEART RATE VARIABILITY (HRV)

The repeated measures ANOVA which consisted of the two within-subjects factors, i.e., (i) Sessions (YBH and Control) and (ii) States (Pre and Post) was performed for the frequency and time domain variables of the HRV.

There was a significant difference between Sessions for (i) LF normalised unit (LFnu)  $F(1, 38) = 13.81, p < 0.01$  (ii) HF normalised unit (HFnu)  $F(1, 38) = 13.61, p < 0.01$  (iii) LF/HF Ratio  $F(1, 38) = 6.17, p < 0.05$  (iv) pNN50  $F(1, 38) = 5.17, p < 0.05$ . Significant differences were noted between states in (i) HR  $F(1, 38) = 37.96, p < 0.001$  (ii) RMSSD  $F(1, 38) = 15.79, p < 0.001$  (iii) pNN50  $F(1, 38) = 16.77, p < 0.001$  (iv) SDNN  $F(1, 38) = 13.04, p < 0.01$ . A significant interaction was observed between Sessions and States in (i) HFnu  $F(1, 38) = 4.12, p < 0.05$ .

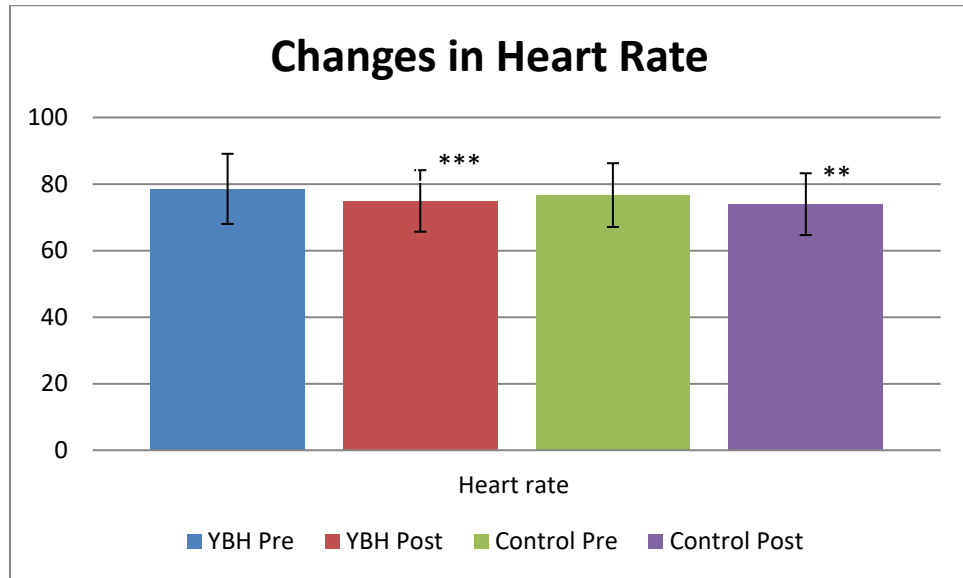
*Post hoc* analyses with Bonferroni adjustment showed significant reduction in HR ( $p < 0.001$ ), whereas increase was noted in LFnu ( $P < 0.05$ ), SDNN ( $P < 0.001$ ), RMSSD ( $P < 0.01$ ), pNN50 ( $P < 0.01$ ) following the YBH session. Following the control session, reduction was noted in HR ( $P < 0.01$ ), and increase in RMSSD ( $P < 0.05$ ) and pNN50 ( $P < 0.01$ ). These changes have been indicated in Table 9 and Figures 10-13.

**Table 9: Summary of the Repeated Measures Analysis of Variance (RM-ANOVA) showing statistically significant results.**

Variables	Factor	F Value	Df	Level of Significance
LFnu	Sessions	13.81	1, 38	0.01
HFnu	Sessions	13.61	1, 38	0.01
LF/HF Ratio	Sessions	6.17	1, 38	0.05
pNN50	Sessions	5.17	1, 38	0.05
Systolic BP	Sessions	81.20	1, 38	0.001
Diastolic BP	Sessions	213.62	1, 38	0.001
Mean Arterial Pressure	Sessions	18.84	1, 38	0.001
Cardiac Output	Sessions	612.38	1, 38	0.001
Stroke Volume	Sessions	6.81	1, 38	0.05
Total Peripheral Resistance	Sessions	581.17	1, 38	0.001
Heart Rate	States	37.96	1, 38	0.001
RMSSD	States	15.79	1, 38	0.001
pNN50	States	16.77	1, 38	0.001
SDNN	States	13.04	1, 38	0.01
Systolic BP	States	81.33	1, 38	0.001
Diastolic BP	States	732.29	1, 38	0.001
Mean Arterial Pressure	States	343.79	1, 38	0.001
Cardiac Output	States	13.83	1, 38	0.01
Stroke Volume	States	22.67	1, 38	0.001
Total Peripheral Resistance	States	580.45	1, 38	0.001
HFnu	Sessions x states	4.12	1, 38	0.05
Systolic BP	Sessions x states	81.31	1, 38	0.001
Diastolic BP	Sessions x states	1030.13	1, 38	0.001
Mean Arterial Pressure	Sessions x states	292.73	1, 38	0.001
Cardiac Output	Sessions x states	14.07	1, 38	0.01
Total Peripheral Resistance	Sessions x states	617.01	1, 38	0.001

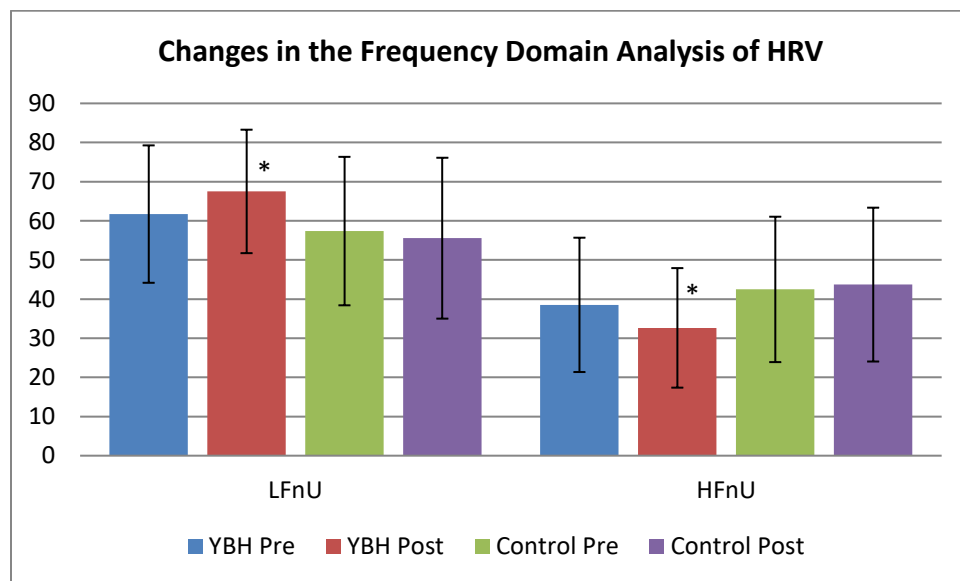
**Figure 10: Heart Rate recorded before and following the YBH and Control Sessions**

Values are group Mean  $\pm$  SD



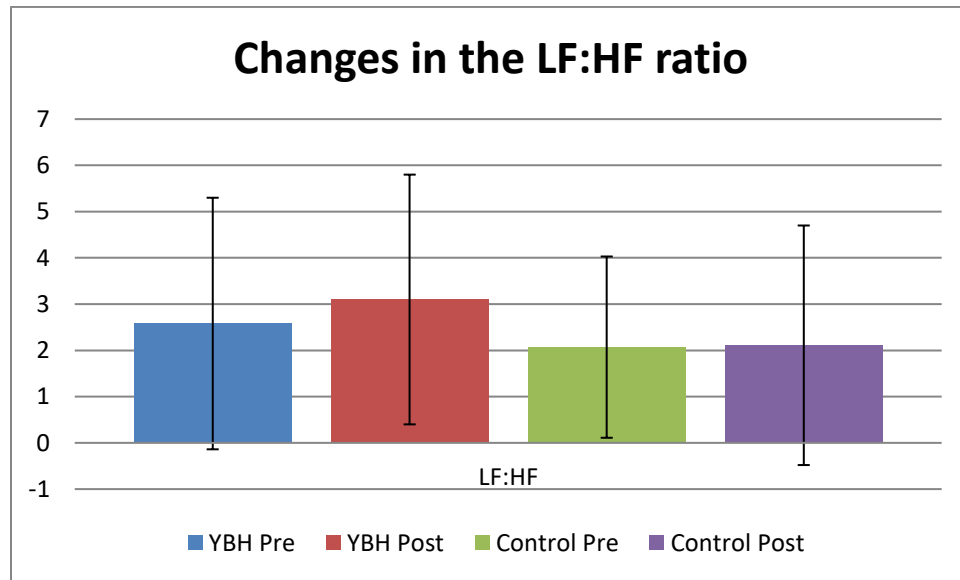
**Figure 11: Changes in the Lfnu & HFnu of HRV; before and following the YBH and Control Sessions**

Values are group Mean  $\pm$  SD



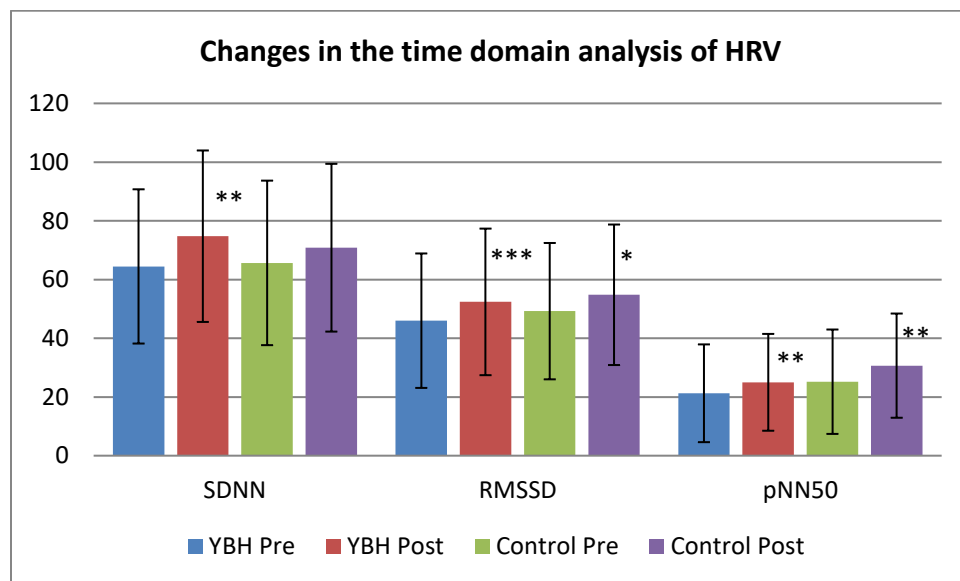
**Figure 12: Graphical representation of the changes in the LF:HF ratio**

Values are mean  $\pm$  SD



**Figure 13: Changes in the time domain analysis of HRV; before and following the YBH and Control Sessions**

Values are group Mean  $\pm$  SD



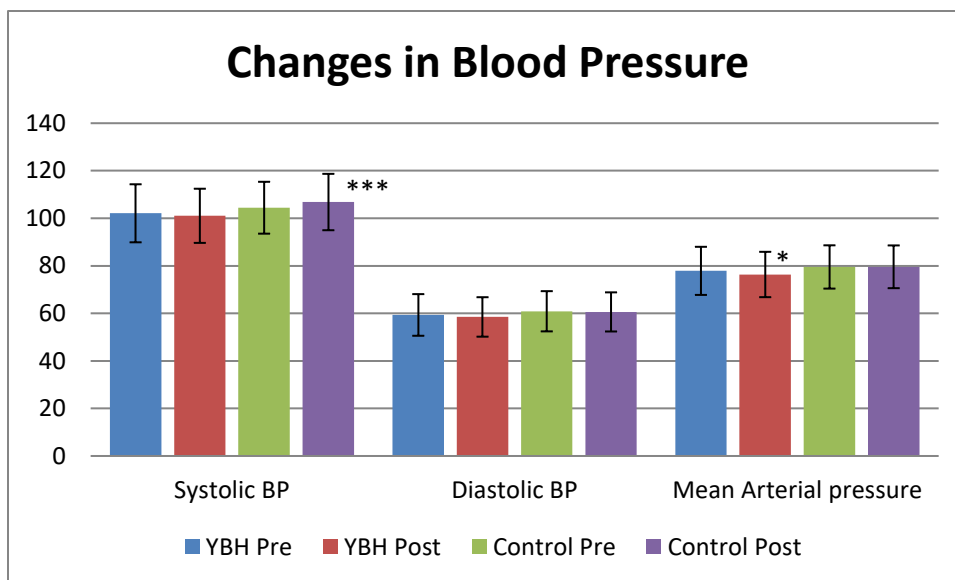
### 6.1.3 CARDIOVASCULAR CHANGES

The repeated measures ANOVA which consisted of the two within-subjects factors, i.e., (i) Sessions (YBH and Control) and (ii) States (Pre and Post) was performed for cardiovascular variables assessed.

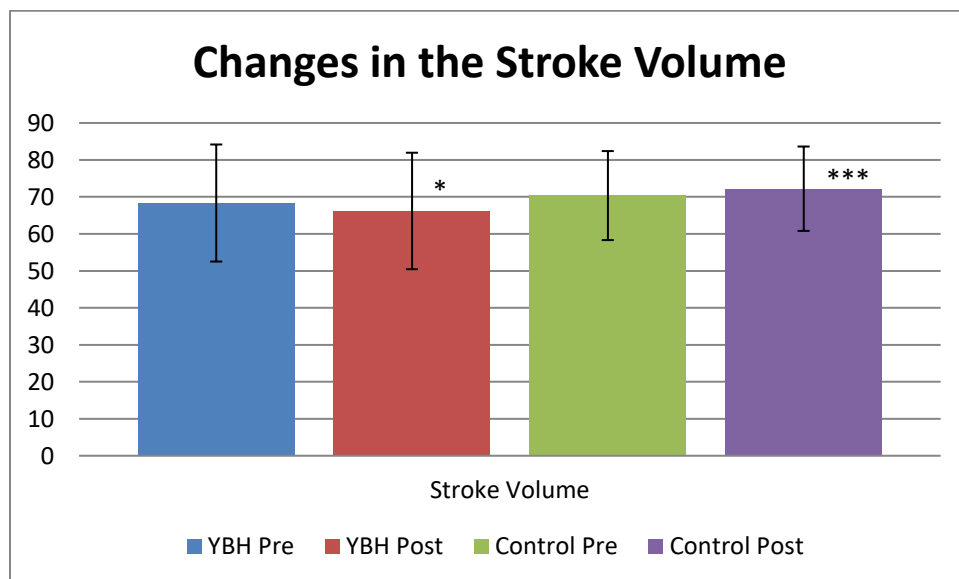
There was a significant difference between Sessions for (i) SBP  $F(1, 38) = 81.205, p < 0.001$ , (ii) DBP  $F(1, 38) = 213.62, p < 0.001$ . (iii) MAP  $F(1, 38) = 18.84, p < 0.001$  (iv) CO  $F(1, 38) = 612.38, p < 0.001$ . (v) SV  $F(1, 38) = 6.81, p < 0.05$ . (vi) TPR  $F(1, 38) = 581.17, p < 0.001$ . Significant difference between the states was observed in (i) SBP  $F(1, 38) = 81.33, p < 0.001$  (ii) DBP  $F(1, 38) = 732.29, p < 0.001$  (iii) MAP  $F(1, 38) = 343.79, p < 0.001$  (iv) CO  $F(1, 38) = 13.83, p < 0.01$  (v) SV  $F(1, 38) = 22.67, p < 0.001$  (vi) TPR  $F(1, 38) = 580.45, p < 0.001$ . Significant interaction between the sessions and states was found in (i) SBP  $F(1, 38) = 81.31, p < 0.001$  (ii) DBP  $F(1, 38) = 1030.126, p < 0.001$ . (iii) MAP  $F(1, 38) = 292.73, p < 0.001$  (iv) CO  $F(1, 38) = 14.07, p < 0.01$ . (v) TPR  $F(1, 38) = 617.01, p < 0.001$ .

*Post hoc* analyses with Bonferroni adjustment showed significant reduction in MAP ( $P < 0.05$ ), SV ( $P < 0.05$ ), CO ( $P < 0.001$ ), whereas there was an increase in TPR ( $P < 0.001$ ) and BRS ( $P < 0.01$ ) following the YBH session. Following the control session, there was reduction in CO ( $P < 0.01$ ) and TPR ( $P < 0.001$ ), whereas increase was noted in SBP ( $P < 0.001$ ) and SV ( $P < 0.001$ ). These changes have been indicated in **Tables 8 and 9 and figures 14-18**.

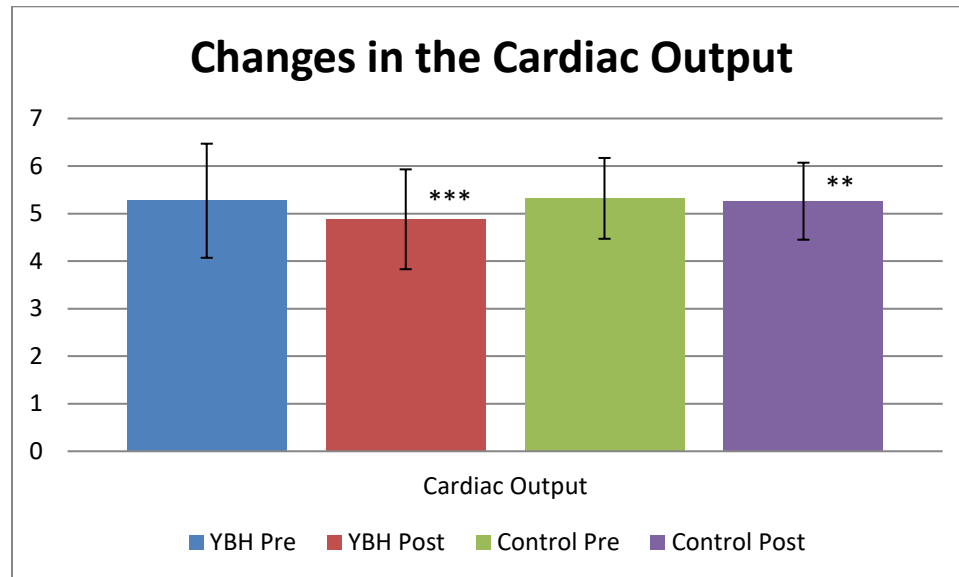
**Figure 14: Changes in Blood Pressure before and following the YBH and Control Sessions (Values are group Mean  $\pm$  SD)**



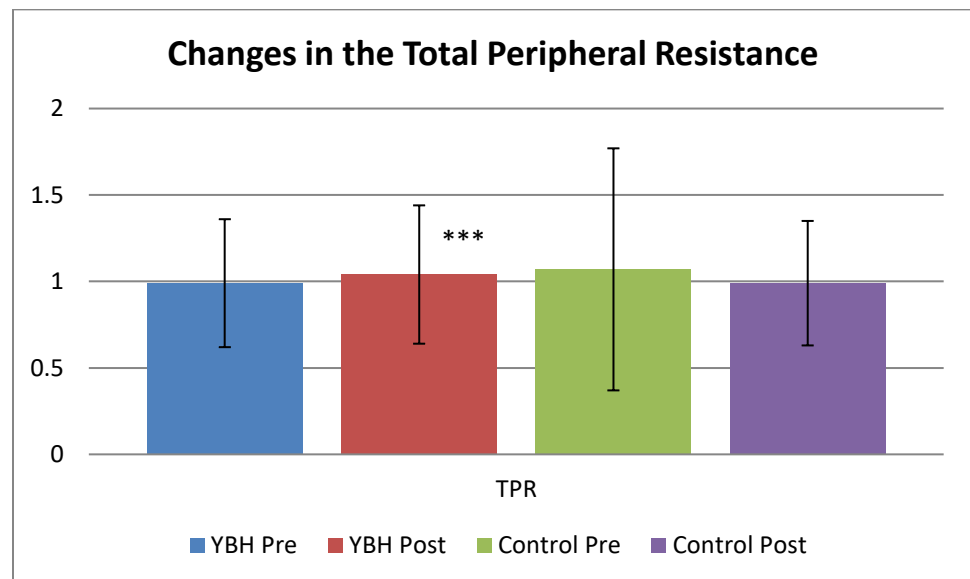
**Figure 15: Changes in Stroke Volume before and following the YBH and Control Sessions (Values are group Mean  $\pm$  SD)**



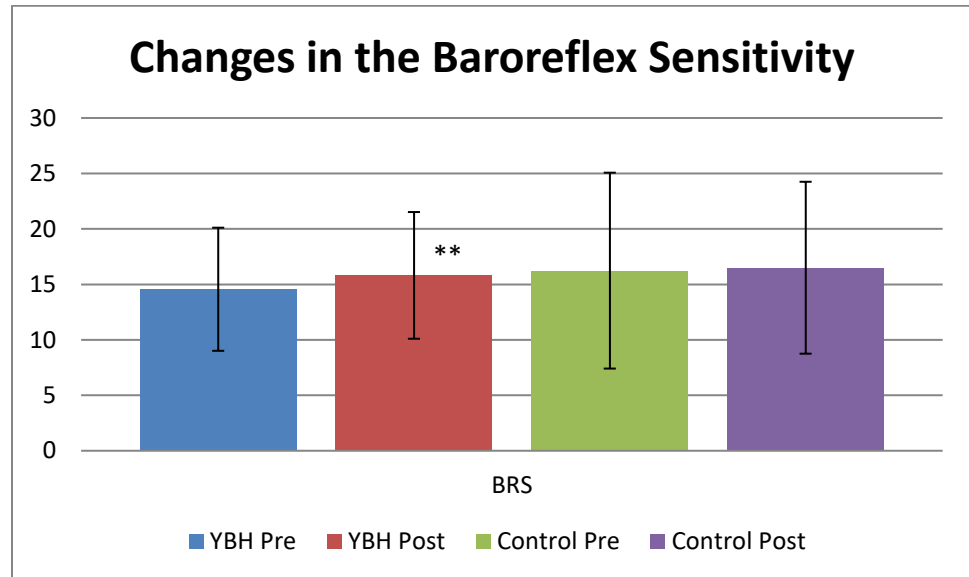
**Figure 16: Cardiac Output before and following the YBH and Control Sessions (Values are group Mean  $\pm$  SD)**



**Figure 17: Total Peripheral Resistance before and following the YBH and Control Sessions (Values are group Mean  $\pm$  SD)**



**Figure 18: Baroreflex sensitivity before and following the YBH and Control Sessions (Values are group Mean  $\pm$  SD)**



## 6.2 PERFORMANCE IN STOP-SIGNAL TASK

### 6.2.1 RECAPITULATION

Thirty Six participants were assessed for performance in the stop signal task (SST) at baseline and immediately following the YBH and control sessions. The Stop-Signal Reaction time (SSRT), mean RT to go stimuli (go RT) and the probability of responding on stop-signal trials ( $p$  [r/s]) were assessed.

Repeated measures ANOVAs were carried out for each variable. This was followed by *post hoc* analyses with Bonferroni adjustment for multiple comparisons between the mean values following YBH and control sessions with that of the baseline. The group mean values  $\pm$  S.D. are given in Table 10.

There was a significant difference between Sessions for SSRT  $F(2, 34) = 4.74$ ,  $p = 0.015$ , however there were significant interactions were found in  $p$  (r/s)  $F(2, 34) = 0.64$ ,  $p = 0.53$ ; and Go RT  $F(2, 34) = 1.076$ ,  $p=0.35$ . The Significant changes in the SSRT are represented graphically in Figure 19. The actual data of cardiovascular and autonomic variables of individual participants is given in Appendix 4.

**Table 10: The outcomes of the Stop Signal Task at baseline and following the experimental and control sessions.**

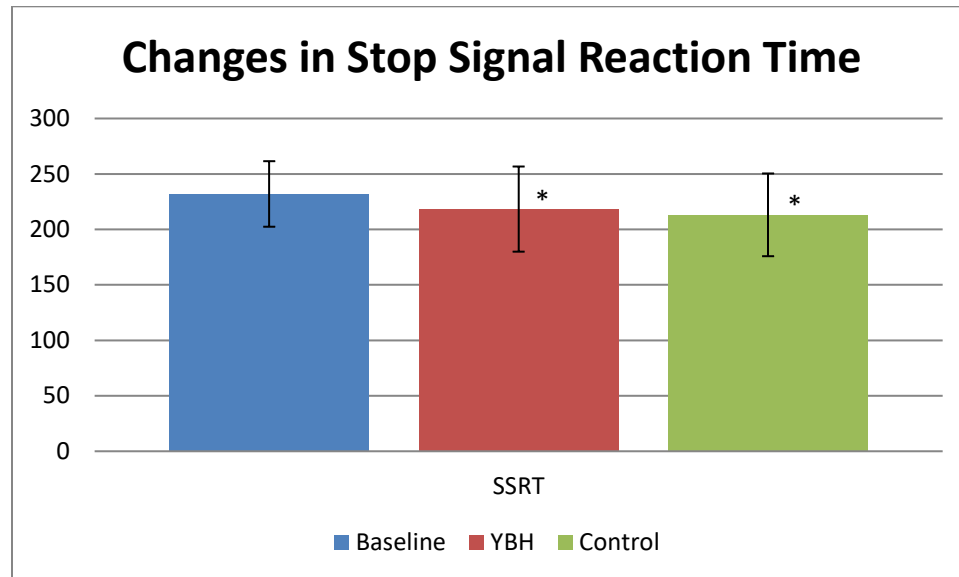
	Baseline	Yoga breathing with intermittent breath hold	Yoga breathing with awareness
SSRT (ms)	231.98 ± 29.54	218.33 ± 38.38*	213.15 ± 37.29*
p (r/s)	47.69 ± 4.12	48.79 ± 4.71	48.69 ± 4.71
Go RT (ms)	565.05 ± 138.03	591.74 ± 174.45	604.08 ± 166.50

\* =  $p < 0.05$ ; Repeated Measures ANOVA (RM-ANOVA) with Bonferroni adjustment

**Table 11: Results of the RM-ANOVA done on the variables from the Stop-Signal Task.**

Variable	Factor	F	df	p value
SSRT	Session	4.74	2, 34	0.015
p (r/s)	Session	0.64	2, 34	0.53
Go RT	Session	1.076	2, 34	0.35

**Figure 18: Graphical Representation of the changes in the Stop Signal Reaction Time (Values are group Mean  $\pm$  SD)**



## Chapter 7

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# Discussion

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## **7.0 DISCUSSION**

The most important results detailed in the previous section are discussed under the two main categories of variables (i) autonomic and cardiovascular variables and (ii) performance in the Stop-Signal Task following the practice of Yogic breathing with intermittent breath holding (YBH) and normal breathing with breath awareness (control) sessions.

### **7.1 AUTONOMIC AND CARDIOVASCULAR VARIABLES**

The current study investigated the effect of yoga breathing with intermittent breath holding on frequency and time domain variables of heart rate variability (HRV) and cardiovascular functions in healthy yoga practitioners. To the best of our knowledge, this is the first attempt to scientifically explore the effects of isolated yoga breathing with breath retention among healthy volunteers. The earlier studies where yogic breath retention was used, it was in combination with other yoga breathing techniques (Telles & Desiraju, 1991; Turankar et al., 2013; Villien, Yu, Barthélémy, & Jammes, 2005).

HRV is the physiological phenomenon of variation in the time interval between heartbeats. It is measured by the variation in the beat-to-beat intervals (Task Force of The European Society of Cardiology and The North American Electrophysiology, 1996). HRV is widely utilized to interpret the cardiac autonomic regulation following various yoga practices (Tyagi & Cohen, 2016). HRV is the pattern of several overlapping oscillatory frequency components. Three components of the frequency domain analyses of HRV have been identified

viz., the high frequency (0.15–0.4 Hz), low frequency (0.05–0.15 Hz), and very low frequency (0.005–0.05 Hz). In general, LF component is correlated with the activity of sympathetic and parasympathetic nervous system whereas HF component with parasympathetic activity. The physiological interpretation of VLF component is unclear (Task Force of The European Society of Cardiology and The North American Electrophysiology, 1996). It is also observed that, high-amplitude peaks in the LF range during rhythmical slow breathing may reflect resonance characteristics of the cardiovascular system where respiratory sinus arrhythmia interacts with the baroreflex (Berntson et al., 1997). Breathing at such resonant frequency may increase HRV and be reflected in large increases in the LF band and simultaneous decreases in the HF band. The findings of the spectral analysis of HRV of the current study indicate an increase in LF and a corresponding reduction in HF, with enhanced baroreflex sensitivity. These changes, thus, may be attributed to breathing at a very slow rate of 0.04 Hz. The findings are similar to earlier yoga studies demonstrating an increase in LF with slow yoga breathing (Peng et al., 1999; Bhagat, Kharya, Jaryal, & Deepak, 2017). Such increase in LF band of HRV may be misinterpreted as a false sympathetic arousal (Bernardi et al., 2000). Breathing at such slow rate imitating the resonant frequency is found to influence the heart rate and blood pressure oscillations and thus enhance the overall HRV (Lehrer, Vaschillo, & Vaschillo, 2000) and subsequent reduction in heart rate and blood pressure (Wang et al., 2010). Yet, the blood pressure changes were nonsignificant in the

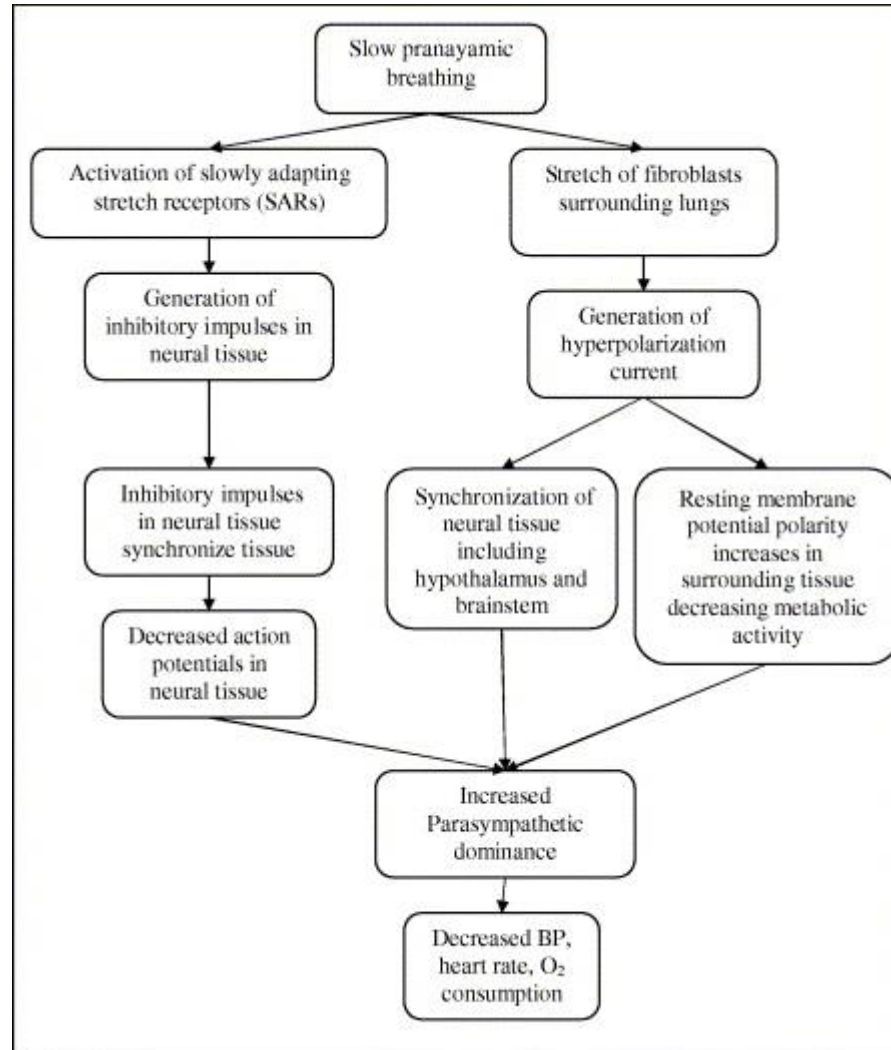
current study. No changes in the frequency components of HRV were observed following the control session.

Among the time-domain variables, SDNN is an indicator of overall heart rate variability, whereas RMSSD and pNN50 are associated with vagal tone (Task Force of The European Society of Cardiology and The North American Electrophysiology, 1996). The changes in the time domain components of the HRV were similar following both experimental and control sessions with an increase in SDNN, RMSSD and pNN50, thus indicating an enhanced HRV. Yet, the magnitude of change was higher following the experimental session. However, the reason for the change following the experimental session remain unclear, whether it was due to intermittent breath retention or slow breathing alone. Since, the participants were long-term yoga practitioners, and performing breath awareness during the control session, they might have entered a meditative state and thus modulating the HRV. Such enhanced HRV is common among long-term yoga practitioners (Tyagi & Cohen, 2016). Similar changes in time-domain variables of HRV were observed in a previous study in participants practicing breath awareness (Telles, Sharma, & Balkrishna, 2014).

We also found a significant increase in the Baroreflex Sensitivity following the 20 min experimental session. Our findings are consistent with earlier studies elucidating the influence of yogic breathing techniques on Baroreflex Sensitivity in healthy (Mason et al., 2013) as well as Clinical population with essential hypertension (Joseph et al., 2005) and chronic heart failure (Bernardi et al., 2002). Such gain in the baroreflex sensitivity may also be

due to slow breathing in the experimental session at the resonant frequency of about 0.04 Hz. Also, the earlier studies attribute the gain in Baroreflex Sensitivity to increased vagal tone, indicated by a gain in RMSSD and pNN50 as well as reduced heart rate. Arterial baroreceptor activity and respiratory sinus arrhythmia are interrelated (Piepoli et al., 1997) and therefore the increase in Baroreflex Sensitivity could be attributed to enhanced HRV following the experimental session. Jerath et. al. propose the action of inhibitory signals and hyperpolarizing current within neural and non-neural tissue activation of slowly adapting stretch receptors, responsible for modulation of the activity of the cardiorespiratory centers (Jerath, Edry, Barnes, & Jerath, 2006).

**Figure 20: Possible mechanism of action for autonomic modulation due to slow breathing. (Coutesy: Jerath et al. 2006)**



Reduced heart rate variability and baroreflex sensitivity is found to be a risk factor for cardiovascular diseases (Rovere, Bigger, Marcus, Mortara, & Schwartz, 1998; Thayer, Yamamoto, & Brosschot, 2010), diabetes mellitus (Silva, Barbosa, Vanderlei, Christofaro, & Vanderlei, 2016; Frattola et al., 1997) and various metabolic syndromes (Stuckey, Tulppo, Kiviniemi, & Petrella, 2014). The enhanced heart rate variability and baroreflex sensitivity observed following

the yoga breathing assessed in the present study may indicate its role in preventing such disorders. Future studies may incorporate clinical population to assess the effect of yoga breathing with intermittent breath retention on the cardiac autonomic regulation.

We also found an increase in the total peripheral resistance and LFnu indicative of a possible sympathetic shift in the autonomic activity. These changes may be due to the very nature of the intervention, which includes focused attention on the verbal cues and constant synchronization of the breathing with it. The nature of intervention needed constant attention, which may be responsible for the selective sympathetic arousal. An earlier study of yoga breathing with breath-retention for short duration indicated an increase in oxygen consumption, which might be considered similar to the results of the present study (Telles & Desiraju, 1991). Also, intermittent hypoxia created in the experimental session would contribute to enhanced sympathetic tone (Spicuzza et al., 2005). Breath holding is known to activate both sympathetic and parasympathetic branches of cardiac autonomic regulation (Chen, Chen, Yun, Huang, & Li, 2014). Despite the sympathetic arousal, the gain in baroreflex sensitivity may be attribute to inhibition of chemoreflex mechanisms due to slow breathing (Bernardi, Gabutti, Porta, & Spicuzza, 2001). Also, long term yoga practitioners demonstrate a generalized reduction in chemoreceptor sensitivity (Spicuzza, Gabutti, Porta, Montano, & Bernardi, 2000). Slow breathing possibly leads to a generalized attenuation in the excitatory pathways for respiratory and cardiovascular systems. Both respiratory and cardiovascular systems share similar control mechanisms,

thus alterations in breathing may be responsible for the cardiovascular changes (Somers, Mark, & Abboud, 1991).

The reduction in cardiac output and stroke volume may be a result of body's compensatory mechanism due to intermittent breath retention along with very slow breath. Also breathing at resonant frequency has been shown to enhance the gaseous exchange and oxygen saturation (Lehrer, Woolfolk, & Sime, 2007), thereby reducing the overall circulatory load. The changes observed in the current study following the intervention are similar to those found following voluntary breath retention in swimmers and divers, which include bradycardia, reduction in stroke volume, cardiac output, and peripheral vasoconstriction (Costalat et al., 2013). However, in the current study, the practice of breath-retention was intermittent and short term instead of the maximal breath retention as practiced by the divers.

Although we could demonstrate differential changes in the autonomic and cardiovascular activity following yoga breathing with intermittent breath retention, the examination of the exact underlying mechanisms was beyond the scope of the study. Assessments during the practice of yoga breathing with intermittent retention may bring clarity on the underlying mechanisms. Lung volume and the partial pressure of CO<sub>2</sub> (PaCO<sub>2</sub>) are known to influence the HRV spectrum in conscious subjects (Pöyhönen, Syväoja, Hartikainen, Ruokonen, & Takala, 2004). We could not control the lung volume as well as assess the PaCO<sub>2</sub> in the current study. Thus, future studies may be planned to assess the effects of lung volume and PaCO<sub>2</sub> in yoga breathing techniques. Future studies may also

include neuroimaging techniques focusing on the neural centers for the vagus nerve to understand the underlying mechanisms.

## 7.2 PERFORMANCE IN STOP SIGNAL TASK

The study was designed to ascertain if the practice of yoga breathing with intermittent breath holding (YBH) affects the Response Inhibition indicated through the performance in the Stop Signal Task (SST). The findings indicate a significant reduction in Stop Signal Reaction Time (SSRT) following both YBH and control sessions, which was in contrast to our initial hypothesis that YBH and control sessions may have differential effects on response inhibition. We also could not elicit significant changes in the  $p$  ( $r/s$ ) and the Go RT in both interventions, when compared to the baseline.

There was a significant reduction in SSRT, suggesting that the practice of YBH and control sessions resulted in enhanced response inhibition (Verbruggen & Logan, 2008). Further, although statistically insignificant, the subject slowed down the go responses following both the conditions, which may be due to proactive response strategy to achieve a balance between competing goals, suggesting a flexible cognitive control (Verbruggen & Logan, 2009). Our results are concurrent to an earlier study on yoga breathing using SST paradigm (Rajesh, Ilavarasu, & Srinivasan 2014). The enhanced SSRT and slowing down of Go RT represent better inhibitory control. A study performed on yoga in prison population showed enhanced response inhibition in a Go-No Go task (Bilderbeck, Farias, Brazil, Jakobowitz, & Wikholm, 2013). Studies have shown similar effects through the administration of medications for ADHD in healthy volunteers (Chamberlain et al., 2009; Nandam et al., 2011).

The observed results may be attributed to relaxation and the autonomic balance attained through the practice, which is indicated in earlier studies on yoga breathings (Mason et al., 2013; Turankar et al., 2013). There was enhanced response inhibition following both YBH and control sessions. A BOLD fMRI study performed to understand neural correlates of the voluntary breath holding demonstrated activity at the bilateral network of cortical and subcortical structures including the insula, basal ganglia, frontal cortex, parietal cortex and thalamus, and pons (McKay, Adams, Frackowiak, & Corfield, 2008). Response inhibition in SST paradigm is mediated through roles of the prefrontal-caudate and striato-thalamic activities (Li, Yan, Sinha, & Lee, 2008). Also, vagus nerve stimulation was found to enhance response inhibition in patients with epilepsy (Schevernels et al., 2016). Therefore, we speculate that yoga breathing with intermittent breath holding may enhance the response inhibition through activation of cortical and sub-cortical brain areas as well as enhanced vagal tone due to slow breathing.

Breath awareness is the basis for several meditation techniques including *Vipasana*, Mindfulness and *Sudarshan Kriya Yoga*. It is understood from the existing literature that being aware of breath could help in to enhance the physiological and cognitive functions to optimal levels through promotion of relaxation and enhanced self-awareness (Carter & III, 2016). Thus, the results following the control session could be attributed to focused attention of the volunteers on the breathing and the relaxation attained through it. Further studies incorporating neuroimaging techniques could reveal the exact mechanisms involved with neuro-cognitive modulation through yoga breathing techniques.

Assessing the long-term effects of yoga breathing on response inhibition was beyond the scope of the current study. Since our study included population of healthy young adults, the results may not be generalized to clinical populations at this stage. Further studies may be taken up to understand how different yoga breathing practices may alter response inhibition. A major limitation of the current study was the inability to ascertain the exact mechanism of action for the observations. It would be interesting to add neuroimaging techniques to further studies, to understand the underlying mechanisms of action. Altered response inhibition is observed in patients with attention deficit hyperactivity disorders (Senderecka, Grabowska, Szewczyk, Gerc, & Chmylak, 2012), schizophrenia (Kiehl, Smith, Hare, & Liddle, 2000), epilepsy (McDonald et al., 2005), obsessive compulsive disorders (McLaughlin et al., 2016), as well as stressful situations (Qi, Gao, & Liu, 2017). It would also be interesting to observe if yoga breathing could be influence the response inhibition in such population.