Stress in the workplace: The contrasting effects of 10 minutes of listening to Chopin vs. HRV biofeedback on autonomic reactivity and cognitive performance.

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2. DECLARATION

I, Sita Smit (Student number: SMTSIT001), hereby declare that the work on which this dissertation/thesis is based is my original work (except where acknowledgements indicate otherwise) and that neither the whole work nor any part of it has been, is being, or is to be submitted for another degree in this or any other university.

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4. Acknowledgements

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5. Abstract

Work related stress has been established as an independent contributing risk factor for the development of cardiovascular disease (CVD) and other life style disease. The aim of this study was therefore to examine the different effects of 10 minutes of biofeedback (BIO) or classical music (CON) intervention on autonomic reactivity and cognitive performance during an induced cognitive stress test and also on the subjective levels of anxiety and relaxation. Both interventions have been shown to help in stress reduction. Forty six healthy male and female volunteers (aged 24 – 59 years) were randomised into a BIO and CON groups. The subjects were required to perform a modified Stroop task both before and after their respective interventions. The BIO group used a biofeedback device, which they had been previously exposed to in the familiarisation trial and the CON groups listened to Fredrick Chopin's Piano Concert No.1 in E minor, op 11 Romanza Larghetto. Significant differences were found between groups in heart rate (HR), respiratory frequency (RF) and various aspects of heart rate variability (HRV), a marker of autonomic reactivity. During the intervention significant differences between groups were apparent in entropy (BIO < CON, $p= 0.000$). The BIO group had significantly lower high frequency power values in the second rest period ($p<0.05$), compared to the CON group. Changes in entropy, standard deviation of normal-to-normal heart beat values; low frequency power and the square root of the mean squared differences of successive normal-to-normal heart beat intervals were evident across time periods. In conclusion, both interventions evoked changes in autonomic reactivity, the biofeedback device engendered more positive subjective responses after cognitive stress compared to the music intervention. The biofeedback device facilitated a carryover effect, whereby low frequency HRV (a marker for vagal withdrawal) was maintained throughout the post rest period and into the post Stroop Task. The classical music intervention - known for its positive effects in enhancing well-being and in decreasing anxiety, reducing stress and causing a relaxation response – on the other hand facilitated an enhanced HRV (a marker for increased vagal modulation) that was carried over into the post rest period. Although there were no cognitive performance differences between groups, only the BIO group experienced a decrease in anxiety and an increase in feelings of relaxation after the post Stroop Task vs. baseline. In contrast the relaxing effects of the music intervention were eliminated by the post Stroop Task. This suggests that preparing for optimal performance is a balance between excess relaxation and over arousal.

Keywords: Stress; Work-related stress; Reaction Time; Biofeedback; Respiratory Rate; Classical Music; Autonomic reactivity.

6. Introduction

Stress is perceived as an inability to cope with related emotional and psychophysiological responses; it can be seen as a reaction to the environment which threatens homeostatic conditions of the body[1, 2].Work stress has been shown to affect individuals both directly and indirectly[3-6]. Directly, stress can induce relative cardiac sympathetic nervous system (SNS) predominance and substantial decrease in parasympathetic nervous system (PNS) [7, 8]. Additionally stress has been identified as an independent risk factor for cardiovascular disease (CVD)[1, 3, 5, 9-12]. Job strain is one of the most common causes of stress and has also been shown to contribute to the development of CVD [1, 3, 5, 9-13].

Work related stress is a growing problem, it is negatively associated with turnover and performance; and it is related to increased work related accidents and substance abuse[14]. Work-related stress contributes a substantial amount to the corporate health costs of the company, which is detrimental to profitability of the company and may be the means to get stakeholders to buy in to stress management interventions[14].

Autonomic reactivity (which heart rate variability (HRV) measures) is a non-invasive method which is associated with the pathophysiological mechanism of CVD[9, 15]. HRV is used to measure the modulation of the heart rate through the autonomic nervous system (ANS) [3, 5, 9, 10]. A decrease in HRV indicates that the ANS is not regulating the heart rate as it should and there is either over activity of the SNS or under activity of the PNS[9]. Cardiovascular recovery from stress was found to be associated with increased vagal modulation despite continual sympathetic activation[15], which supports the association between HRV and risk for CVD[15] and highlights the need for stress management interventions which focus on increasing vagal modulation.

In normal conditions the SNS responds in times of stress: increasing heart rate (HR), contractility of the heart and blood pressure (BP) whereas the PNS initiate the reverse responses, having a dampening influence over the cardiovascular system (CVS) [16]. Optimal cardiac function requires a balance between these two systems[16]. Research has shown decreased HRV in stressed individuals, compared to their healthy counterparts[4, 17]. It also suggested that work stress associated with altered autonomic profiles can be improved

through stress management programs implemented at the worksite[4]. Altering perceptions, increasing emotional adaptability and adjusting psychophysiological state through the use of relaxation techniques are forms of stress management techniques[1].

Biofeedback devices provide a helpful stress management technique which promotes relaxation as well as training healthier coping strategies[1]. Biofeedback forms part of group of methods which utilize "self-regulation therapies", this implies the control of aspects of one ANS[16, 18-20]. Biofeedback devices utilize technology capable in detecting small changes in physiological variables[16, 19, 20].Biofeedback devices have potential for therapeutic use for CVD as deregulation of the ANS is seen in a large number of diseases and in stress[16]. Biofeedback devices provide real time readings of physiological markers which aid in regulating health[20]. The device only provides the feedback, the subjects need to learn the correct means in controlling physiological variables[20]. Biofeedback has been shown to increase HRV[21], the feedback records the R-R interval from blood pulse sensors and increasing variability is achieved through breathing at a resonant frequency[19], normally about 6 breathes per minute in adults. Convincing results have been shown causing short term changes in autonomic variables (HR and BP) when individuals are provided with feedback[19].

Classical music was chosen as the contiguous intervention for its known positive effects in enhancing well-being, decreasing anxiety, reducing stress and causing a relaxation response; the mechanism appears to be a direct physiologic effect through the ANS by decreasing BP, HR and respiratory rate [22-24]. Classical music has also been shown to decrease SNS activation[30]. Relaxing music (self-chosen) has been shown to significantly reduce anxiety and may have an effect on the cognitive component in the stress response when compared to the other variations[25]..

Both biofeedback and classical music have been successful in reducing stress; this paper evaluates the physiological differences between the two modalities in stressed males and females at their place of work. Autonomic reactivity, HR, BP, breathing rate and cognitive performance will be evaluated. Both modalities have an effect on the ANS; biofeedback teaches the control of physiological reactions that form part of the stress response[16], it has

been shown to affect the ANS through exercising slow breathing[26]. Whereas music, especially classical, is known to enhance well-being, decrease anxiety and reduce stress through a direct physiological effect on the ANS[22-25, 27].

7. Literature Review

7.1 Heart Rate Variability

Introduction to Heat Rate Variability

Heart rate variability (HRV) is a reliable indicator of the many physiological factors modulating the normal rhythm of the heart[28-30]. HRV represents the beat-to-beat changes in heart rate and is considered to mirror the ability of the heart to detect, adapt and respond to stimuli[28, 31, 32]. The analyses of HRV requires accurate timing of R waves, which can be done with an electrocardiogram (ECG)[33]. The power spectrum density analysis of HRV is the most widely utilized, it measures heart rate changes in frequency domains[34]. HRV fluctuates and it is divided accordingly into different frequencies, these fluctuations are measured between 0.01 and 0.5 hertz (Hz) with heart rate spectral analysis measures[35]. High frequency (HF) and low frequency (LF) are used mostly[29, 32]. The HF component is mainly modulated by the parasympathetic neurons, whereas the LF component has mixed modulation from both PNS and SNS neural activity[32]. The LF/HF ratio is typically a measure of sympathovagal balance[34]. HRV is useful as it has shown good reproducibility[36], it is a non-invasive measure which gives insight into autonomic activity[36], it able to successfully work on a multi-ethnic sample[37] and with recent technological advances HRV analysis has become a popular tool in clinical measures as well[29].

In resting conditions the vagal tone prevails, and variations in heart rate variability (HRV) are largely dependent on vagal modulation[38]. The vagal and SNS constantly interact, afferent stimulation of the vagal leads to reflex excitation of efferent vagal activity and inhibition of sympathetic efferent activity[38]. The inverse effect is mediated by afferent sympathetic stimulation; efferent vagal activity is restrained by cardiac afferent sympathetic activity[38]. This highlights that both LF and HF can increase under different conditions. Increases in LF have been observed during 90° tilt, standing, mental stress and moderate exercise in healthy subjects[38]. Conversely, increases in HF have be shown under controlled respiration and cold stimulation of the face[38]. HRV only measures the fluctuations in autonomic inputs and not the mean level of autonomic inputs, thus both decreased vagal modulation and increased sympathetic input can lead to diminished HRV[38].

HRV has been shown to provide a way to observe the interaction between the SNS and PNS[28, 29]. Therefore the analyses of HRV may give insight in assessing cardiac health and status of the ANS as it is responsible for regulating cardiac activity[28, 35]. HRV has been shown to predict poor prognosis in a number of cardiac diseases when there is an increased sympathetic predominance[29, 32, 34, 39, 40]and it gives valuable input in assessing stress levels[34, 41, 42]. Standardized measures of HRV are considered valid and reliable makers of vagal modulation of the cardiac Sino arterial (SA) node [41, 42]. The ANS has many associated factors which affect HRV including posture, respiration frequency, age, gender, physical or mental load, pain, numerous disease conditions, and different drugs[41]. Multiple inputs influence the variability including efferent cardiac-vagal input, sympathetic and endocrine influence. It has been repeatedly shown that stress (acute and chronic), depression, hypertension, chronic fatigue syndrome and anxiety diminish HRV[34, 42]. Bottom-up interventions have been shown to increase HRV both during and following therapies[42]. This indicates that there is room for external and individual control of HRV using certain techniques.

The ability of the heart to respond and the functioning of the nervous control can be shown by the extent of variability[28]. HRV, baroreflex and chemoreflex sensitivity are modulated by respiration; abnormal respiration modulation of the HR is often an early sign of autonomic dysfunction in a number of diseases[43]. The action of the ANS in controlling the heart rate is the balancing between the SNS and PNS[28]. Increased vagal activity is associated with increased HRV whereas increased sympathetic drive tends to decrease HRV[28, 42]. Due to the continuous changes in sympathetic and PNS balance there are continuous fluctuations around the mean HR[33]. These frequent adjustments are controlled by cardiovascular (CV) mechanism[33]. These continuous fluctuations cause periodic fluctuations, the main periodic fluctuations are respiratory sinus arrhythmia (RSA), baroreflex-related and thermoregulation-related HRV[33].

<u>Factors that affect Heart Rate Variability:</u>

<u>Breathing and its effect on Heart Rate Variability</u>

The vagaly mediated HR can be externally altered since the vagus is synchronized with the breathing rate which is known as RSA[21]. It has been shown that there is a connection between breathing rate and HRV[33]. Inspiration causes an inhibition of vagal tone while

expiration excites the vagus; breathing thus causes HR fluctuations which equal the breathing rate(BR)[33]. Previous research has shown that slow or deep breathing can acutely increase baroreflex gain in healthy individuals[21]. RSA is optimal when BR is closest to the frequency of the intrinsic baroreflex-related HR fluctuation (the 0.1Hz Meyer wave), which is therefore maximal (in adults) at about six breaths per minute [33]. The standard deviation of the normal-to-normal interval (SDNN) is calculated over short periods, usually 5 minutes. SDNN is therefore a measure of changes in heart rate due to cycles of 5 minutes, it reflects all the cyclic components responsible for variability in the period of recording and therefore it represents total variability. SDNN reflects both LF and HF variability and it is maximum when breathing at 0.1Hz, which is resonant frequency breathing, and is achieved during therapeutic/biofeedback breathing at ±6 breaths per. This highlights that there is both PNS and SNS involved in resonant frequency breathing at ~0.1 Hz since only frequencies above 0.15Hz is considered to be solely due to the PNS.

In quiet respiration there is an association between the changes in vagal and vasomotor activity[44], but there is larger susceptibility to autonomic inputs (excitation on inhibition) in expiration versus inspiration[44]. This was determined when testing twenty apparently healthy volunteers who either performed retrospective (n=12, ages 21-39 years) or prospective (n=8, ages 18-32 years) measurements of HRV in different breathing conditions[44]. The retrospective group were rested and practiced spontaneous breathing whereas the prospective group practiced different types of breathing (spontaneous and 12 breaths per minute to an auditory tone) [44]. It was concluded that inspiration suppressed vasomotor firing and responsiveness; while expiration facilitated vasomotor responsiveness[44].

It has been shown in research over the past 150 years that HRV and its relationship with deep breathing is a highly sensitive measure of cardio vagal function[45-47]. Pranayamic breathing, a type of breathing practiced in yoga, has been shown to decrease oxygen consumption, HR, blood pressure (BP), increase in PNS activity, increased alertness and cause reinvigoration [48]. It is the manipulation or control of breath that ultimately causes a shift in autonomic balance towards PNS control[48]. Pranayamic breathing is a very slow breathing technique and may provide insight to ANS regulation mechanism[49]. The effect of

pranayamic breathing on HRV was investigated on a yoga practitioner before, during, and after exercises[49]. Significant changes were observed in the inter-beat interval ($p<0.001$), LF/HF ratio in favour of HF therefore PNS respiration, and decrease of breathing frequency after the exercise versus the state before the breathing exercise[49].

The effects of different breathing patterns was examined[50]. Thirty-four healthy individuals (mean age 23) were examined while performing different breathing patterns, namely, spontaneous breathing, paced breathing (10 breaths per minute) and breath holding[50]. The results indicated that paced breathing significantly diminished the nonlinearity of HRV[50].

Measurements of respiration, BR interval and BP of twelve healthy participants (average age 29) were taken when either remaining silent, completing mental arithmetic, completing aloud arithmetic, talking freely or reading aloud[39]. Different breathing styles were also compared, spontaneous versus controlled. When comparing spontaneous breathing with silent reading there was an increased BR ($p=0.05$), a decreases BR interval and HRV and increased BP[39]. When reading aloud, free talking and mental arithmetic aloud a change in the LF: HF ratio was observed favouring LF, other changes included minor changes in respiration and BR interval[39]. This showed that while doing simple mental or verbal activities HRV may markedly change due to changes in respiration. The biofeedback device in our study uses breathing as a mean to alter physiological responses of HR and HRV.

<u>Meditation and its effect on Heart Rate Variability</u>

Meditation that is focused on inward attention appears to alter the sympathovagal balances to PNS predominance and induce regular fluctuations in HR which may support health benefits where there is SNS dominance due to stress or disease[32]. A study examining inward-attention meditation and normal rest (mean age 27 experimental group; 26 control group), and its effect on HRV found that there was a decrease in LF: HF ratio which suggests the benefit of sympathovagal balance lies towards PNS activity[32]. All participants with a background in regular meditation will thus be excluded from our study.

Smoking and its effect on Heart Rate Variability

HVR can be affected by smoking, this is shown by a study done on healthy men and women (n=39; mean age =20.33 +/- 3.47yrs). The results of the study showed that habitual cigarette smoking caused changes in the SA node, as baroreflex and HRV were reduced in the group who smoked. There was a noticeable increase in indicators of SNS modulation ($p<0.05$), which was accompanied by reduced vagal drive[51].

Blood Pressure and its association with Heart Rate Variability

In a study measuring BP and HRV over a 24 hour period (mean values of the 48 half hours) in participants who were normotensive or had essential hypertension, it was found that in both groups there was a marked reduction of both HRV and BP during sleep. There was also a positive relationship between mean BP and HR throughout the full 24 hour period in both groups. This further emphasizes the primary role of the central nervous system (CNS) in the overall CV modulation, with no substantial difference between conditions of normal and chronically elevated BP[27].

Circadian rhythm and its association with Heart Rate Variability

The neural control in humans effects continuous, but expected changes throughout the day and night[40]. In a study where dynamic 24 hour monitoring of ECG in hospitalized participants (n=18) and non-hospitalized participants were done, it was found that there was a certain circadian pattern of sympathovagal balance with fluctuated predictably throughout the day and night[40]. The most significant observation was the sudden increase in the LF:HF ratio during the early morning accompanied by a reduction in vagal tone[40].

Heart Rate Variability and Stress

In a recent study 53 students completed a laboratory stress protocol in order to examine the relationships between depressed mood and PNS control of the heart[7]. HRV was measured at rest and during the stress tests and depression was measured with a questionnaire. They concluded that depressed mood is related to the magnitude of decrease in PNS cardiac control during stressors in healthy men and women[7].

In a recent study evaluating the effect clinical training has on students (n=12) HRV and ECGs were measured. The study found significantly lower HF readings at awakening compared to before the clinical training (p 0.01)[8]. The LF/HF ratio was also significantly increased during compared with before clinical training (p 0.01). The findings concluded that the levels of stress induced by clinical training induced relative cardiac SNS predominance in the students[8].

Others factors effecting Heart Rate Variability

The effect of age and ethnicity on HRV was examined in rest and in response to stress in youths and young adults (n= 399) of different ethnicity (European American and African American) both male and female. HRV was measured at rest and during a challenge(video game)[52]. The results showed that African Americans had significantly higher resting root mean square of successive differences (RMSSD) of normal R-R intervals and high-frequency (HF) power than European Americans ($P< 0.01$) [52]. Females displayed larger decrease of RMSSD and HF during the challenge compared to males ($P< 0.05$) [52]. There was no significant sex difference of resting HRV or ethnic difference of HRV response to stress[52]. The relative influence of age, resting HR and sedentary lifestyle on HRV was examined in a young and older population[53]. A five minute HRV reading was measured and fitness levels were measured. HR was similar for both groups, however older sedentary participants showed reduced HRV[53]. There was no difference between sedentary and active subjects in the younger group[53]. The study concluded that HRV decreases with increasing HR and with age regardless of lifestyle[53].The findings did not support the idea that changes in HRV are related to regular physical activity[53].

Many other factors can also influence HRV such as certain drugs[33], alcohol[54] and many different diseases[28]. Therefore we will attempt to control for all the controllable factors.

7.2 Cognitive Performance

Introduction to Cognitive Performance

In our study working memory and reaction time will be used as the cognitive performance measures. Working memory is the ability to utilize attentional mechanisms to select relevant information when faced with distracting information, which occurs when inhibition

of the distracting information fails[55]. Working memory capacity is an important factor that influences tasks which have a high demand for selective attention, such as a Stroop task[56, 57]. This view of working-memory limitations predicts a relation between individual differences in capacity and performance on tasks that either encourage or demand controlled attention[58].

Cognitive Performance and its association with Heart Rate Variability

HRV has been shown to be correlated with improved executive functioning in cognitive tasks, namely improved response time and increased correct responses[59, 60]. A study which investigated the effect of vagal tone on executive and non-executive tasks using a working memory and a sustained attention task, found that higher HRV was associated with better performance in the cognitive tasks[54]. Healthy male participants completed the study the results showed more correct response, less error and increased reaction time correlated with a high HRV[54]. Thus correlates implies that HRV facilitating an increased ability to respond to stimuli during a cognitive task.

Cognitive Performance and its association with Heart Rate Variability and Physical Fitness

The relationship between physical fitness, HVR and cognitive performance was tested in 37 male participants[55]. The participants completed cognitive test before and after completing an eight week physical fitness intervention program they were randomised to either a non-training group or a training group[55]. Measurements of physical fitness (maximum oxygen consumption), resting HRV and cognitive function were recorded[55]. There were no significant differences between the two groups at the pre-test[55]. The untrained group had a decrease in maximum oxygen consumption and a decreased HRV compared to the trained group. The trained group had increase reaction time and positive responses in the post cognitive tests[55]. Physical activity has an effect on the PNS activity which may improve an individuals' ability to respond correctly to cognitive tasks. When comparing aerobic athletes with matched controls it was shown that the athletes had increased power in all frequency bands of their cardiac spectograms[28, 61]. This suggests that athletes will have increased ability to adapt to stimuli when compared with controls.

7.3 Stress

Stress is perceived as an inability to cope with related emotional and psychophysiological responses. Management of stress needs to incorporate the environment, altering triggers where necessary[1]. Altering perceptions, emotional responses which are more adaptive and adjusting psychophysiological state through the use of relaxation techniques are all forms of stress management[1]. Biofeedback devices provide a helpful stress management technique which promotes relaxation as well as training healthier coping strategies. Stress can be seen as reactions to an environment that threatens homeostatic conditions[2]. Several types of stress have been suggested: flight or fight response (activate SNS), depressed response (feel defeated or helpless) or the endocrine counterpart where hypothalamic-pituitary-adrenal (HPA) activation occurs[2, 5].

Work can affect stress directly, by affecting the ANS and BP or indirectly, by affecting lifestyle[3-5].An independent contributing risk factor for the development of cardiovascular disease (CVD) is stress[1, 3, 5, 9-12]. Job strain is one of the most common causes of stress and has also been shown to contribute to the development of CVD [1, 3, 5, 9-12]. A reduction in HRV is an independent predictor of myocardial infarction (MI), stable coronary artery disease and congestive heart failure[9]. HRV measures homeostatic regulation of the ANS in response to changes, and has been previously shown to quantify risk for cardiac events[34].
One research study directly compared laboratory HRV in predicting autonomic regulation of "real-world" emotional stress[34]. The results confirmed the efficacy of laboratory acquired HRV in predicting autonomic response to acute emotional stress[34]. A similar study evaluated the effect of job stress, evaluating the relationships between stress and the indicators of the ANS in employees of a manufacturing industry[62]. Job stress was assessed in 140 participants and was found to have no significant effect on HRV or catecholamine's[62]. The main finding indicated that LF HRV was significantly higher in the high-strain group of subjects with a short duration of employment[62]. The study also concluded that HRV could be used as a physiological indicator for job stress[62].

There is a relationship between stress and depression; and depression is among the top five leading causes of disability and disease burden throughout the world[63] . Stressful events across ones lifespan cause the onset and course of depression, however not all people who

encounter a stressful life experience succumb to its depressogenic effect[63]. The breathing technique used in yoga is a unique method for balancing the autonomic nervous system and influencing psychological and stress-related disorders, it has been shown to help individuals with depression[64]. Alternative therapies have been evaluated the help with depression, these techniques are primarily aimed at decreasing physical and mental tension. Such treatments may include elements of meditation, yoga, and other mind-body therapies[65]. Mixed results have been shown but the overall opinion is that the current research is promising, but further research needs to be completed [65].

Recent research examined CV recovery and the relationship between vagal activity and reflexes to risk for CVD[15]. CV recovery from stress was found to associate with increased vagal modulation despite continual SNS activation[15]. Decreased vagal rebound during recovery was associated with risk factors for CVD. This supports the association between HRV and risk for CVD[15]. Hamer and Steptoe examined the association between physical fitness, cardiac PNS control and inflammatory cytokine responses to mental stress[66]. A cohort of 207 men and women which measured HRV before during and after the stress test, and testing submaximal fitness[66]. The groups were divided according to the results of their fitness test low fit, medium fit and high fit. They concluded that physical fitness was associated with decreased inflammation response to acute stress an effect thought to be mediated through PNS pathways[66]. This is one of the mechanisms proposed that physical fitness protects against CVD[66].

Stress related changes in HRV during sleep were characterized in 59 healthy men and women[67]. Acute psychophysiological stress was associated with decreased levels of PNS modulation during non-rapid eye movement (NREM) and rapid eye movement sleep and increased levels of sympathovagal balance during NREM sleep[67]. An increase in PNS modulation was noted across successive NREM cycles in the control group; these increases were less in the stress group and remained essentially unchanged across successive NREM periods. Sympathovagal balance was higher during NREM sleep and was associated with poorer sleep maintenance and lower with the stress group[67]. The study concluded that changes in HRV associated with acute stress could be a causing factor of disturbed sleep. Stress-related changes in HRV during sleep may also be important in association with

chronic stressors, which are associated with significant morbidity and increased risk for mortality[67].

A recent study implemented a simple worksite stress management program (over a one year period) in order to reduce stress, revert stress related ANS deregulation and lower arterial BP[4]. The findings showed altered HRV in the stressed participants (n=91) compared to their healthy co-workers (n=79)(p=0.001) [4].The stress management program reverted these differences as well as slightly lowering systolic BP[4]. The study showed how work stress is associated with altered autonomic profiles and suggests stress management programs can be implemented at the worksite[4].

In a systematic review it was concluded that adverse psychosocial working conditions are a risk factor for ischaemic heart disease amongst men[3]. The review examined HRV and work based stress in various studies with different time periods, work stress was associated with decreased HRV, but the evidence for sympatho-adrenal and HPA biomarkers were less established [3]. The ability for appropriate physiological and psychological responses depends on recognition of the specific situations.

In recent research in chess players it was found that emotional changes correlated with changes in HRV[68]. Loss of optimism or intensified hopelessness correlated with a decrease in HRV suggesting a decrease in vagal tone[68]. Nervousness, uncertainty, indecisiveness and fright showed similar findings[68]. The emotional aspect is largely overlooked, but is important as it could be an independent risk factor. In a recent study Fourie et al showed that self-esteem increased HRV, while guilt decreased HRV[69].

A research study investigated the changes in HRV and HR during episodes in which stress may be cognitively represented, but not necessarily present[70]. A group of 73 teachers were measured reporting all worry episodes and stressful events. The results showed that during times of worry there was increased HR (neutral time 2 beats/min and time of worry 2.75 beats/min, respectively) and HRV (1.07ms and 1.05, respectively). This showed that episode of worry have independent cardiac effects in addition to the effect of stressful events [70].

Students were examined during "real-life" stress (examinations) in order to compare the magnitude of autonomic and vascular responses[6]. The results showed that the markers of autonomic regulation of the Sino atrial node correlated significantly with cortisol levels[6]. The results support the concept that "real-life" stress increases BP and alters CV homeostasis[6]. These changes implicate the link between psychological stress and increased CV risk of hypertension[6]. A similar study had comparable findings, a group of 42 students were tested during a real life stressors (examination) HRV was significantly decreased in the stress situation[17]. This study concluded that HRV could be used as a stress analyser in real life events[17].

The two main regions of the stress system include ANS and the HPA, theses two neural stress systems co-ordinate the responses of many physiological systems to a stressor including both the immune and the CV system[71]. The neuro-immune interaction were evaluated in order to elucidate the underlying actions and interactions of these system and their role in disease risk in stress related disorders[71]. Vagal tone was measured in 109 white collar workers; the study found that the detrimental work stress effects are partially mediated by increase HR, BP and lower vagal tone[71].

A study analysed the effect of work environment (lighting, views, and cubicle size) on stress response[72]. The findings highlighted that certain aspect contribute changes in HRV, circadian variation and cortisol levels[72]. This implicates important social, economic and public health implications for work environment risk factor on health[72].

7.4 Biofeedback devices

Biofeedback forms part of group of methods which utilize "self-regulation therapies", this implies the control of aspects of one ANS[16, 18-20]. The ANS is also known as the "involuntary" or "visceral" which implies that processes governed by this system are largely unconscious[16, 19]. However through training and conscious thought certain functions of the ANS can be adjusted voluntarily[16, 18, 19]. Biofeedback devices assist in controlling certain aspect of the ANS. Biofeedback devices utilize technology capable in detecting small changes in physiological variables[16, 19, 20].

Biofeedback devices have potential for therapeutic use for CVD as deregulation of the ANS is seen in a large number of diseases. It has been demonstrated that the use of biofeedback techniques can regulate input of the ANS to the heart[16]. Biofeedback devices have been used in patients with hypertension, as well as in patients with CVD and it has recently been incorporated as an aid in reducing feelings of stress (stress management) [16]. In normal conditions the SNS responds in times of stress: increasing HR, contractility and BP whereas the PNS initiate the reverse responses, having a calming influence over the CVS [16]. Optimal cardiac function requires a balance between these two systems[16]. Biofeedback is a useful aid to increase awareness of physiological responses as well as increase control over certain physiological responses.

Biofeedback has been shown to increase HRV, the feedback records the R-R interval from blood pulse sensors and increasing variability is achieved through breathing at a resonant frequency. Convincing results have been shown causing short term changes in autonomic variables (HR and BP) when individuals are provided with feedback[19]. A recent study focused on arterial baroreflex and pulmonary function utilized HRV biofeedback as a method to increase vagal baroreflex gain and improving pulmonary function among 54 healthy adults[21]. The research found that there was acute increases in LF and total spectrum HRV[21]. Vagal baroreflex gain correlated with slow breathing during biofeedback sessions[21]. The biofeedback group also experienced less adverse relaxation side effects[21].The study concluded that HRV biofeedback had strong influences on both baroreflex gain and pulmonary function[21].

A recent study utilized biofeedback training in order to increase awareness of HR and respiration rate[26]. HRV biofeedback uses the same relaxation components found in many relaxation or mindfulness techniques, isolating slow breathing. This has an effect on the ANS altering it through slow breathing[26]. This was compared to functional magnetic resonance imaging (fMRI) which indicated increased activation in the amygdala and the anterior cingulate cortex following HRV biofeedback training, although not significant due to the small sample size(n=2) it opens area for research which looks promising[26].

Biofeedback and Stress Management

Biofeedback can serve as an additional tool to managing stress; it is an effective adjunct to stress management as it teaches the control of physiological reactions that form part of the stress response[16]. It has been shown that mental stress is a significant risk factor for CVD and biofeedback has shown significant impact on both disease and symptoms, given that most CVDs involve inappropriate regulation of the ANS instruction in the use of biofeedback to control activation of the SNS and PNS s is likely to be useful in cardiac patients[16].

Biofeedback-assisted stress management (BFSM) incorporates the use of physiological markers in addressing stress management[20]. The main goal of BFSM is to give individuals tools to control both mental and physiological responses to stress. Over activated SNS is commonly found alongside stress and affects the ANS negatively, rectifying balance is key in the management of stress[20]. BFSM involves learning to regulate physiological feedback[20]. Biofeedback provide real time readings of physiological markers which aid in regulating health[20]. The device only provide feedback and the subjects need to learn the correct means in controlling physiological aspects. [20] Various relaxation techniques, such as slow and rhythmic breathing, guided imagery, progressive muscle relaxation, mindfulness, assertiveness, and how to change negative thought patterns are included with BFSM[20]. Using BFSM to complement stress management techniques is helpful as BFSM does not only teach how to control physiological variables but rather how to utilize the physiology response when faced with stress[20].

Bidirectional interactions between the brain and peripheral tissue have become increasingly evident[42]. Bidirectional communication of both the autonomic and neuroendocrine pathways with the CNS and the periphery, facilitate the expression of affective, autonomic, hormonal, and immune responses. Therapies should be directed toward addressing these functional links, especially in diseases which are multifactorial[42].

7.5 Breathing and Stress

The effect of voluntary slow breathing on cardiac PNS response to a threat was examined[73]. 30 students randomized into three breathing groups (slow, fast and spontaneous breathing) [73]. The HF HRV showed significant increases both the fast and spontaneous breathing

whereas it was unchanged in the slow breathing group. There was no difference in respiration rate during exposure to threat in all the groups. The study concluded that slowed breathing decreases the cardiac PNS withdrawal in response to a threat [73].

Decreasing respiration rate is a common feature in relaxation techniques as well as stress management programs[73]. It is thought to decrease autonomic response to a threat[73]. The SNS activates in response to threat and can go into overdrive imitating the fight or flight response[73]. Breathing rate has been shown to decrease the responsiveness of the SNS as slow breathing has been shown to decrease PNS withdrawal retuning balance to the ANS[73] .

In a recent study by Prinsloo et al participants in the experimental group who completed 10 minutes of deep slow breathing had an increased HRV when comparing to the control group who completed 10 minutes of concentrative meditation without deep breathing[74]. This study also showed that the experimental groups' reaction times and working memory capacity was significantly increased during their Stroop cognitive test. They also felt less anxious, less sleepy and significantly more relaxed than the control subjects[74]. It was concluded that deep breathing without meditation had more significant effects in increasing cognitive performance compared to meditation without deep breathing. This highlights the importance of deep breathing in demanding situations. Breathing and relaxation techniques utilize awareness of breathing rate, rhythm and volume of breath in order to minimise physiological responses to stress by increasing PNS response[75]. Previous research in breathing training found a correlation between slow breathing and an acute increase in HRV [76].

Research into breathing training with and without a biofeedback component has shown positive results on individuals with hypertention[75, 77]. Individuals (age 25-75) diagnosed with hypertension were put in the control or experimental group. The controls listen to classical music and the experimental group listened to BIM (Breathe with Interactive music) which guided the patients towards slow regular breathing[78]. The study outcome showed that controlled breathing with the BIM had more significant temporary reductions in BP when compared to the controls. Relaxation breathing training has been shown to positively affect the physiological and psychological health in children (age 6-14) with moderate to severe asthma [79], but further studies on breathing training in the general population are needed.

Mind-body research has been extensively tested on risk factors for stroke, especially hypertension and mild reductions have been achieved with both meditation and breathing training. The main focus in breathing training is to lower the BR and modify respiratory patterns[77, 78, 80]. Maintaining 6 breaths per minute, both inspiration as well as expiration performed over 5 s each is the optimal rate during breathing training. Even a single session in breathing training has yielded temporary positive results in BP[75,77].
Sustained decreased BP can be achieved if regular breathing training practise is maintained. In a similar study a group of 100 patients (ages 28-72) with essential hypertension (on anti-hypertensive medication or not) were randomly selected into either the slow breathing or mental relaxation group[77]. They practiced mental relaxation or slow breathing for 10 minutes with a 15 minute quiet period both before and after. Slow breathing had significantly larger fall in HR ($p< 0.05$), BR ($p< 0.001$), systolic BP ($p< 0.05$) and diastolic BP ($p< 0.01$).

This effect has been shown repeatedly, a study which examined the effects of diaphragmatic breathing (DB) on BP and HR was investigated in data from a clinically healthy young normotensive subjects (n=42). The participants followed 3 weeks of ambulatory monitoring as a reference standard, both BP and HR was measured with a manual monitor at 1–min intervals for 5 min before and after DB (three deep diaphragmatic breaths). The participants performed DB for about 2 weeks at about 2–h intervals while awake[81]. A decrease in BP was found when comparing individuals to their own reference data[81], however only the acute effect was measured.

A study which found similar results tested thirteen subjects (age 25 – 56 years) who performed the deep breathing test: 6 breathes per min, five second inhalation and five second exhalation[82]. The effect of the deep breathing test on finger BP was measured. It was concluded that deep breathing may cause significant reductions in BP.

7.6 Classical Music

Music, especially classical, is known to enhance well-being, decrease anxiety and reduce stress; this appears to be done by exerting a direct physiologic effect through the ANS by

decreasing BP, HR and BR [22-25, 27]. It has been proven that music effectively reduces anxiety and improves mood of medical and surgical patients, both children and adults[22]. It has also been shown that music improves quality of life of patients, as well as enhancing a sense of comfort and relaxation[22]. For our study classical music was chosen for the control intervention due to its known effect in causing a relaxation response.

Classical music has been found to statistically significant decrease in mean arterial BP, HR, and BR (P <0 .0001) in patients (music group n=115, control group n=88) undergoing surgery[24]. A similar study found that 30 minutes of music (a combination of classical music, religious music and natural sounds) was successful in reducing anxiety in patients (music group n=32, control group n=32) on a ventilation machine[23].

Studies on healthy individuals showed similar results. A group of healthy students (n=56) showed significant reductions in anxiety, anger and SNS nervous activation when comparing classical to sitting in silence or listening to heavy metal music. Another group of sixty healthy students (age 18-49 years) were assigned to either listen to music or sit in silence(participants brought their own "relaxing" music) and various factors were measured including skin temperature, muscle activity and HR[25]. Participants also rated both their anxiety and relaxation levels after either listening to music or remaining in silence[25]. The results indicated a significant finding (p=0.024) that music (self-chosen "relaxing" music) decreases anxiety and therefore may have an effect on the cognitive component in the stress response when compared to the other variations[25].

7.7 Modified Stroop Task

The effect is named after John Ridley Stroop he was the first to publish the effect in 1935[83]. It evaluates the flexibility of the control of cognitive processes and behaviour, evaluates psychological capacities of an individual and lastly allows for a measure of reaction time of the task[83, 84]. There are two responses in the original design the congruent which is the natural response in reading the presented word and the incongruent which is the response to the un-associated colour of the word[59]. The incongruent requires cognitive control to prevent reading and to activate the colour-naming response[59]. The added time that is

required to process the colour in the incongruent response is known as the Stroop interference[59].

It is vital to observe that the nature of the stimulus plays an important role in attentional selection[85]. The Stroop task displays both relevant and irrelevant (word and colour or vice versa) information about the same object this requires a narrowing of attentional focus in order to respond correctly and ignore the distracting information[85, 86].

This is observed in a study of apparently healthy individuals (n=14) who underwent a fMRI while performing a Stroop task[86]. The results showed areas of the brain involvement in both processing attended and ignored features. Therefore there was both neural enhancement of the task relevant feature (colour) and neural suppression of the irrelevant feature (word) [86].

It has been researched whether exercise could maintain and enhance brain health in adults, however performance in tasks requiring little executive control was unaffected by exercise participation[87]. Significant improvements were found in Stroop task in the exercising group when compared the control group, therefore exercise may play a role in performance where increased executive control is required[87].

The effect HR and HRV response to both mental and physical stress was tested on 207 male and female participants (mean age ± Standard deviation 52 ± 3 years) [88]. Increased physical fitness, which is guided by lower exercise HR, showed a relationship ($p<0.02$) with HRV response to stress (mental stress, 5 minute Stroop task) [88]. The mechanism proposed is the association between lesser inflammatory cytokine response and acute mental stress, an effect which may be partly mediated through the PNS pathways[88]. This may be a contributing factor to the protection exercise and increased fitness against CV risk[88].

Smoking and smoking cessation (acute) can both effect the validity of the Stroop task. Smokers are less able to concentrate when abstinent from smoking and have an increased ability to focus after smoking[89, 90]. In a study where smokers and non-smokers performed a Stroop task on two consecutive days, where smokers refrained from smoking overnight between the two days, it was shown that the smokers demonstrated increased response times (decreased performance), whereas the non-smokers had no changes in performance[64].

7.8 Questionnaires

Spielberger State questionnaire (STAI-S) is a questionnaire which assesses the degree of anxiety of an individual using a scaled self-report structure. The Smith questionnaire is also a self-report questionnaire it is designed to assess various aspects of stress, relaxation, meditation, and mindfulness.

7.9 Conclusion

Our research aims at furthering the knowledge of the effect of biofeedback devices on autonomic reactivity (via HRV measures), affect and cognitive performance. There are limited studies which include both male and female participants working in the same environment. This study completed testing of participants at the place of employment and during office hours. This allows practical measures of work related stress as well implementing previously laboratory based research. This acute implementation of stress reducing techniques is practical, given that long intervention in the corporate environment is not realistic.

8. Experimental design and methods

The study protocol was approved by the Research and Ethics Committee of the University of Cape Town (Rec ref:296/2005) and conducted in accordance with the principles of the Declaration of Helsinki, ICH Good Clinic Practice and laws of South Africa. Subjects were only included in the study once they had signed a consent form issued to them and once the investigator had provided a full oral and written explanation of the study and risk factors. All subjects were informed that they were free to withdraw from the study at any time. All the data collected was locked in a safe environment, and was used for scientific purposes in a confidential manner.

8.1 Subject population

The target population was individuals from five different medium to large business corporations, who were randomly divided into two groups; a biofeedback group and control group. The sample size was 46 subjects, 23 in the control group and 23 in the biofeedback group. Initial questionnaires established eligibility to participate in the study. Exclusion criteria were previous diagnosed cardiac or psychiatric disorders or the current use of any psychotropic medications, heart rate altering medications, stimulants or recreational drugs. Volunteers who regularly participated in meditation techniques were also excluded. Only one subject was excluded from the testing trial due to a diagnosed cardiac disease. In the familiarisation subjects unable to understand the modified Stroop task would have also been excluded, however all participants were able to complete it.

8.2 Training and familiarisation protocol

The familiarisation protocol was standard for both groups and was completed during office hours in a dedicated office at the place of employment. At the familiarisation testing session all subjects signed informed consent forms and filled in a personal details questionnaire. Participants completed questionnaires which established exercise practices, previous breathing training, personal information and any meditation practices. The Trait component of the Spielberger State-Trait Anxiety Inventory (STAI-T) was then completed in order to establish baseline levels of anxiety. The STAI-T measures aspects of life stress and general personality traits. After the questionnaires were complete, three pre-gelled electrocardiogram (ECG) electrodes (Blue sensor, Ambu, Denmark) and a respiratory

transducer belt was attached to the subject, in order to measure physiological data. The ECG electrodes were placed in positions representing Eindhoven's triangle namely, on the sub-clavicular space bilaterally and over the last rib on the left-hand side. The skin surface was first cleaned and gently abraded with an alcohol swab before electrodes were attached. The 3 electrodes were wirelessly connected to a Biopac System MP150WSW (Biopac Systems, Goleta, CA, USA) ECG amplifier which was set to a band-pass filter between 0.5 and 35Hz and a sampling frequency of 1000Hz was used. The transducer belt also transferred data wirelessly to the Biopac to record respiratory frequency (RF). Subjects were seated at a desk in a comfortable chair and eight minute eyes closed recordings were made of the above physiological measures to establish base line physiology and to familiarize the participants with the testing equipment.

8.3 Training

The experimental group then underwent a 15 minute training session with a hand-held HRV device (StressEraser™,Helicor, USA) which was previously validated by Heilman, Handelman, Lewis and Porges[91]. The participants were required to place their right index finger on the infrared sensor, the device then displayed beat to beat changes in heart rate. The measure it reads is the interbeat-interval (IBI) which is transformed into a sinusoid wave which is displayed on the screen[92], enabling the user to see real time fluctuations in their heart rate.[92]. The device grades the users' sinusoid wave which can be altered when the users changes their breathing pattern, the grading is based on the wavelength for each sinusoid cycle. Points are given depending on the threshold targets that have been met. The StressEraser assists by predicting when the next exhalation should be taken with a triangle marker. The sinusoidal HR wave and respiration co-vary in a perfect phase relationship[92]. To achieve an optimal results the user should inhale until the wave peaks and exhale until the wave begins to rise again. Resonance between the main sources of cardiac vagal drive namely, the baroreflex and respiratory sinus arrhythmia (RSA) can be achieved when using the device correctly[93]. This breathing rate is referred to as resonant frequency, it differs between individuals but is usually between 4.5 and 7 breathes per minute. Large increases in both RSA and baroreflex gain are evident when done correctly[21] and there is a promotion of respiratory efficiency [92, 94]. Resonant frequency is the frequency where the response amplitude is a relative maximum. At these frequencies small forces can produce large

amplitude oscillations. After the training, all participants had to accumulate 30 points in ten minutes to demonstrate that they were using the device correctly[35].

The control group did not use any device, but they underwent the same familiarization process and they listened to relaxing music for ten minutes instead of doing biofeedback training. This music was by Frederic Chopin, Piano Concert No.1 in E minor, op 11 Romanza Larghetto.

During the familiarisation trial blood pressure and heart rate were recorded on two separate occasions by an automated blood pressure monitor (model HEM-705CP, Omron, Illinois, USA, validated by O'Brien, Mee, Atkins and Thomas[95]).

8.4 Experimental protocol

The experimental protocol was completed within one week of the familiarisation protocol in the same place also seated on a comfortable chair at a desk with a computer monitor. The timeline of the experimental protocol is illustrated in figure 1. All the subjects completed the State component of the Spielberger (STAI-S) and the Smiths Relaxation States Inventory 3 (SRSI3) both before and after the experimental trial. The STAI-S is a questionnaire which assesses the degree of state anxiety of an individual using a scaled self-report structure. The SRSI3 questionnaire is also a self-report questionnaire; it is designed to assess various aspects of stress, relaxation, meditation and mindfulness. Initial BP and HR were recorded. The subjects then underwent two familiarisation Stroop Tasks before taking part in the trial to minimise skill learning. The first session was a short explanation trial where the assessor talked the participant through the test and thereafter each subject completed a full familiarisation Stroop Task.

Electrodes for ECG and a respiratory transducer belt were then attached to the subject to record physiological data. The data was transferred wirelessly to a Biopac System to record ECG and RF. The subjects then completed a 5 minute eyes closed pre rest period (pre rest) and straight thereafter completed a 5 min 30sec pre-intervention Stroop Task (pre Stroop). The subjects then completed their respective 10 min interventions. The biofeedback group completed ten minutes with the StressEraser and the control group listened to a CD with classical music. Immediately after the intervention a second reading of BP and HR was recorded, and a Visual analogue scale (VAS) of sleepiness was administered. Both groups then completed a 5min post intervention eyes closed rest period (post rest) and a third

reading of BP and HR, and second VAS of sleepiness, before they completed the post-intervention 5min 30 sec Stroop Task (post Stroop). A final reading of BP and HR was recorded as well as a final VAS scale of sleepiness after the post Stroop.

After this the subjects completed the post-test questionnaires and they were asked to rate the subjective efficacy of the intervention using a VAS. The various VAS scales used were solid lines 20cm long, participants were instructed to make a mark on the line which was scaled from 0% to 100%, and the mark was measured and converted into a percentage.

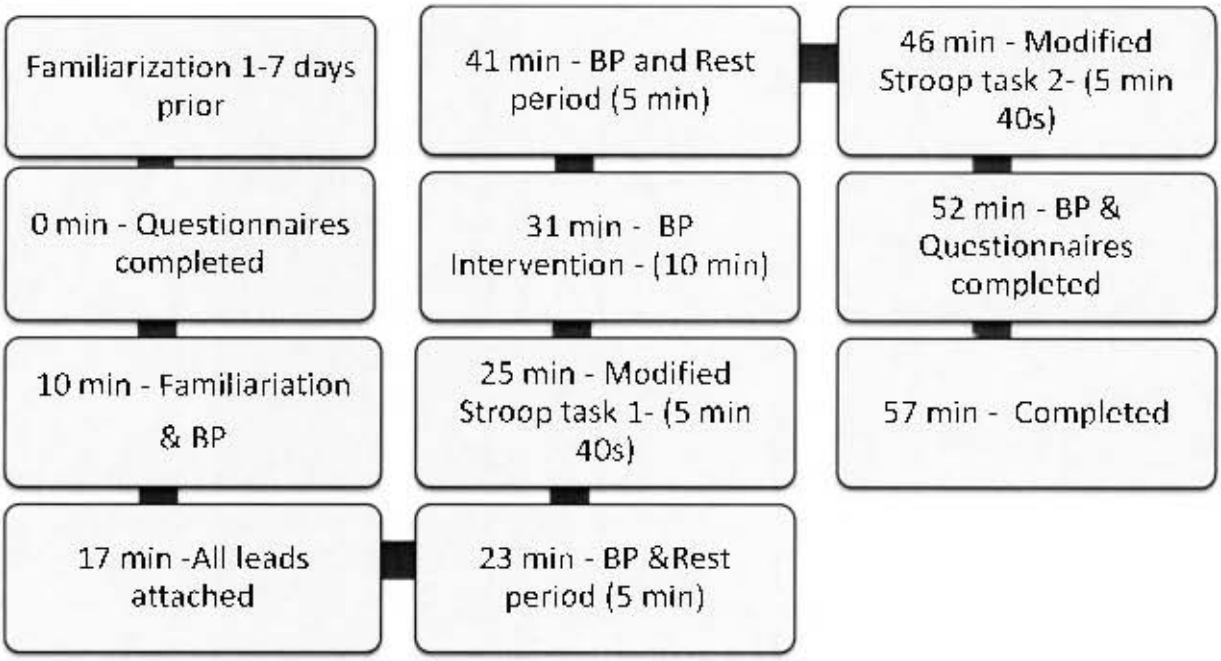


Figure 1: Flow diagram of timeline of the experimental trial from the initial familiarisation until completion of trial.

Abbreviations: min, minute; s, second; BP, blood pressure.

8.5 Modified Stroop Task:

The modifications of the original Stroop entailed electronically recorded responses using the computers keyboard instead of reading and the addition of a working memory component. The modified Stroop Task consisted of the individual appearance of 4 colour words (red /blue /yellow /green) 2cm in height and displayed in five different colours (red /blue /yellow /green /grey) in the centre of computer monitor on a black background. They appeared every 3 seconds for 400ms and were then replaced by a blank screen which was the response period that lasted 2600ms[96]. The word was displayed in either coloured (red /blue /yellow /green) ink or grey ink, but it was never displayed congruently in the same colour as

the written word (e.g. never written word: blue in blue ink). The subjects were instructed to respond using the response keys as quickly and accurately as possible. They were required to respond to the colour of the word and not the written word, except if the word was written in grey ink then they needed to respond to the word itself. By using the grey words it ensured that subjects had to read and recognize the colour-words rather than just noticing the colours, thereby invoking the Stroop effect. Responding to the colour of the word tests the inhibition of prepotent response while simultaneously testing reaction time[83]. Plain white squares (n = 20), presented randomly between the Stroop colour words were used for the working memory component; subjects had to continuously count the white squares throughout the modified Stroop task. The amount of square remained the same as each participant only completes the task 3 times; the participant is unaware of the number of squares throughout testing and each participant is told that the squares appear randomly. The total amount of squares counted was recorded after the modified Stroop task and accuracy was measured. Subjects responded using the following keys: 1- red, 2-blue, 7-green, and 8 -yellow. A total of 100 cues were thus presented randomly: 60 incongruent colour words (15 of each colour), 20 grey words and 20 white squares.

8.6 Data analysing:

The ECG recordings were analysed with AcqKnowledge for Macintosh OS X (version 3.9.0). This software uses a modified Pan and Tompkins algorithm to detect QRS complexes. The filtered ECG recording tachograms were then visually inspected to determine the correct recognition of QRS complexes and T waves. Missed and ectopic beats were corrected by either adding or spacing beats.

Only after each of the tachograms showed no false beats was the data analysed using HRV analysis software from the Biomedical Signal Analysis Group (Department of Applied Physics, University of Kuopio, Finland). Data was transformed using autoregressive (AR) analysis with an AR model order of 15 into low frequency (LF - 0.04 - 0.15 Hz) and high frequency (HF - 0.15 - 0.4 Hz) components. The software calculated the low LF power and HF power and we calculated the total frequency (TF) power (LF power + HF power) from this. Additional measurements in the time domain were calculated by the software, including: the square root of the mean squared differences of successive normal-to-normal intervals (RMSSD), the standard deviation of the normal-to-normal interval (SDNN) and approximate

entropy. The SDNN is calculated over short periods (5 minutes), it is therefore a measure of changes in heart rate due to cycles of 5 minutes, it reflects all the cyclic components responsible for variability in the period of recording and therefore it represents total variability. Approximate entropy is the presence of repetitive patterns of fluctuation in a time series which makes it more predictable than a time series in without patterns, it is the likelihood that similar patterns of observations will not be followed by additional similar observations[97]. The carry-over effect is the measure of whether the physiological effect is "carried over" into the subsequent rest period, which is important as that is the period where subjects would prepare for the following modified Stroop task.

During the 10 min intervention the ECG tachogram from minute 3 to minute 8 was used to calculate HRV measures. This provided the standard 5 minute period for comparison. Minute 3 to minute 8 was selected to allow the subjects 3 minutes to recompose their physiological state after they completed the modified Stroop task. The power values in the cardiac spectrograms were recorded in ms^2 and were then natural log transformed to normalise the variance.

The respiratory rate per minute was measured via a force transducer fixed to a belt placed around the chest wall. Subjects were asked to expel the air from their lungs when the transducer belt was first fitted and then they were instructed to breathe normally. The chest transducer was wirelessly connected to an amplifier with a low-pass 10Hz filter. Respiratory frequencies RF (i.e. breaths per second) were calculated from the respiratory rate. The HF component is mainly modulated by the PNS , whereas the LF component has mixed modulation from both parasympathetic and sympathetic neural activity[32]. The LF/HF ratio is typically a measure of sympathovagal balance[34].

The scores for the questionnaires were analysed using the associated scoring sheet. In the questions the higher the calculated value, the higher the level of anxiety, in both STAIT and STAI-S; or higher level of relaxation in the Smiths questionnaire.

8.7 Statistical analysis:

To determine the differences over the five time periods repeated measures ANOVA (analysis of variance) were used to establish time and group effects, as well as time-group interactions. When significant differences were established, Tukey's post-hoc test was used to find specific differences.

Independent t-test and ANOVA's were used to look for BIO and CON group differences in the subject characteristics and questionnaires.

Percentage decrease in mistakes and reaction time in the cognitive performance test was compared by using t-test (independent by groups).

Pairwise product moment and partial correlation was used to find correlations between data. All data is described as means ± standard deviation (SD) in the text and tables, and as SEM in the figures. A P value of < 0.05 was considered to be statistically significant.

9.Results

9.1 Part 1 - Initial measurements

9.1.1 Subject characteristics

The BIO and CON subject characteristics are displayed in table 1. The groups had a significant difference in their ages, but there were no other significant differences between the groups. The types of jobs ranged from managerial positions (50%), administration jobs (consultants, private bankers, executive consultants; 26%) and other white-collar jobs (airline load control agents, human relations, IT co-ordinator; 24%).

Table 1. Subject characteristics of BIO (n=23) and CON (n=23)

	BIO	CON
Age	41 ± 8.8	36 ± 8.9 *
Females	3	9
Males	20	14
Exercise (hours/week)	3 ± 3	3.3 ± 2.3
Smokers	3	8

BIO- Biofeedback group; CON- control group

Group effect

* p = 0.047 BIO age vs. CON Age

9.1.2 Familiarisation trial

The physiological and questionnaire data from the familiarisation trial is represented in table 2. There were no differences between groups in any of the variables.

The STAIT scores had a significant negative correlation with both RMSSD (r = -0.39, p=0.029) and SD NN (r = -0.53, p=0.002), displayed on figure 2.

BP was measured twice and averaged. According to the guidelines of the American College of Sports Medicine[98] eight participants were classified as having normal BP, while 27 were classified as pre-hypertensive and ten as having stage 1 hypertension. One participant was classified as having stage 2 hypertension. Systolic BP was negatively correlated with RMSSD (r=-0.32, p=0.049), presented on figure 2.

Table 2. Heart rate, Respiratory rate, blood pressure recordings and heart rate variability values of BIO (n=19) and CON (n=19) in the familiarization trials

	BIO	Range	CON	Range
Heart rate (beats/min)	74 ± 13	57 - 101	72 ± 12	56-106
Blood pressure systolic (mmHg)	134± 13	105-163	128± 13	103-146
Blood pressure diastolic (mmHg)	83 ± 18	64-106	78 ± 9	66-91
Respiratory rate (breaths/min)	15 ± 4	8 - 19	17 ± 4	9 - 25
STAIT	35.8 ± 8.3	23 -48	36.4 ± 8.8	23 - 54
LF power	6.9 ± 0.7	5.8 -8.1	7.0 ± 0.7	5.6 – 8.3
HF power	5.6 ± 0.9	4.2 – 7.8	5.8 ± 0.9	5.5 – 7.5
TF power	7.2 ± 0.6	6.0 – 8.3	7.2 ±0.7	5.9 – 8.7
SDNN	44 ± 12	25 - 66	59 ± 21	32 - 87
Entropy	1.04 ± 0.12	0.7 -1.22	1.22 ± 0.10	0.8 - 1.23
RMSSD	29 ± 12	10 - 62	33 ± 15	13 - 75

BIO- Biofeedback group; CON- control group

STAI-T- Spielberger trait questionnaire; LF- Low frequency power ; HF- High frequency power; TF- Total frequency power; Standard deviation of the normal-to-normal interval (SDNN); Square root of the mean squared differences of successive NN intervals (RMSSD); milliseconds (ms); millimetres mercury (mmHG)

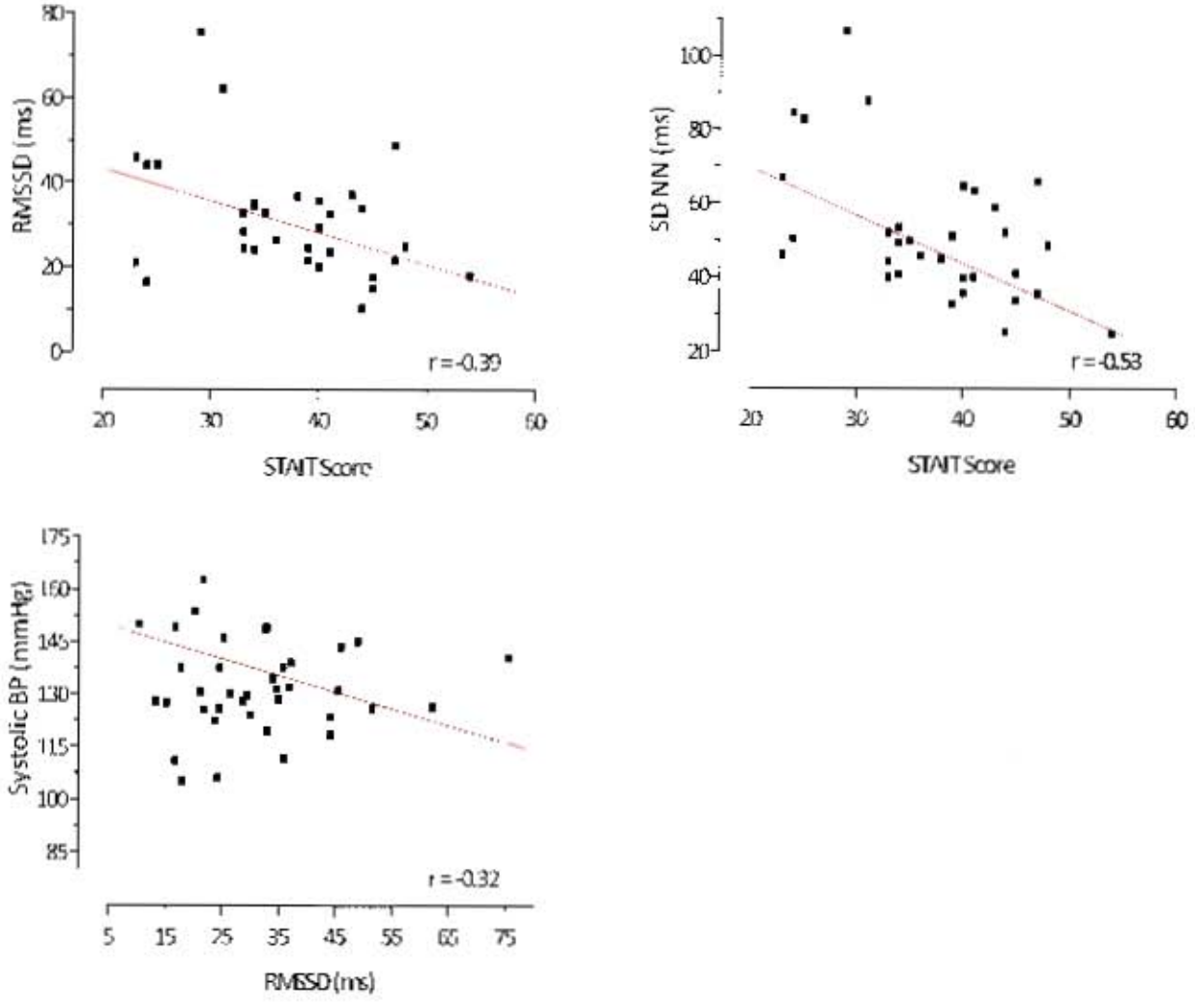


Figure 2: Correlation between STAIT scores, RMSSD, SDNN and Systolic BP in the familiarisation trial.

Abbreviations: STAIT- Spielberger trait questionnaires; Standard deviation of the normal-to-normal interval (SDNN); Square root of the mean squared differences of successive NN intervals (RMSSD); milliseconds (ms); BP- blood pressure; mmHg- millimetres mercury.

9.2 Part 2 - Intervention trial

9.2.1 Questionnaires

The results from the STAI-S, SRSI3 and VAS questionnaires are shown in table 3. The STAIS scores were not different between the groups, however there was a significant decrease over time ($F_{1,37} = 14.453$, $p = 0.000$). The BIO group displayed a significant decrease from pre STAIS to post STAIS ($p= 0.009$), which was not evident in the CON group. The total score of the Smith relaxation inventory were not significantly different between groups, however there was a time effect ($F_{1,37} = 20.754$, $p = 0.000$). The BIO group displayed a significant decrease from pre to post ($p=0.001$), this was not displayed in the CON group. Furthermore in the energised elements of the SRIS3, there was a significant interaction effect ($F\ 1, 37 = 4.2892$, $p=0.045$). The BIO group also felt significantly more energised after the experimental trial was completed compared to how they felt at the start ($p= 0.000$). There were no differences in either of the visual analogue scales (VAS) measuring feelings of sleepiness and subjective measures of the helpfulness of the intervention.

Table 3. Results of the questionnaires of BIO (n=23) and CON (n=23)

	BIO	CON
STAIS- Pre	36.9 ± 8.3	36.2 ± 10.1
STAIS – Post	31.9 ± 6.5#	33.9 ± 8.4
SRSI3- Pre	49 ± 19.6	54.4 ± 21.3
SRSI3- Post	59.8 ±20 #	51.0 ± 31.1
SRSI3- Pre Energised	3.1 ± 1.2	3.6 ± 1.4
SRSI3- Post Energised	4.9 ± 1.2 #	3.9 ± 1.1 *
VAS- helpful	5.4 ± 3.2	6.5 ± 2.6
VAS- Sleepy immediate	4.9 ± 2.8	4.5 ± 2.8
VAS –Sleepy after 5 minutes	4.4 ± 2.3	4.3 ± 3.0
VAS –Sleepy after 5 minutes	4.4 ± 2.7	4.2 ± 3.4

BIO- Biofeedback group; CON- control group
STAI-S- Spielberger state questionnaire; SRI3- Smiths relaxation inventory 3; VAS- visual analogue scale
Group effect
* $p < 0.05$ BIO vs. CON
Time effect
# $p < 0.05$ BIO Pre vs. BIO Post

9.2.2 Physiological data

BP and RF measures for the rest and Stroop periods are displayed in Table 4. Blood pressure (BP) was similar in both groups throughout the testing except for the initial systolic value, the BIO group had a significantly higher systolic BP reading compared to the CON group (134 ±13 vs. 128 ± 14 respectively , p= 0.046). RF displayed significant increases from post rest to post Stroop in both groups (BIO, p= 0.005 and CON, p=0.004). Respiratory rate (RR) increased significantly in the CON from rest to Stroop, both pre (p= 0.000) and post (p=0.001). RR displayed the same effect in the BIO group increasing from rest to Stroop task (p= 0.000, for both pre and post).

Table 4. Respiratory frequency, Respiratory rate and blood pressure recordings throughout the experimental trial in the BIO (n=23) and CON (n=23).

	BIO			CON		
	RF	Resp	BP	RF	Resp	BP
Rest 1	0.22 ± 0.07	14 ± 3	136*/86 ± 13/8	0.24 ± 0.10	16 ± 4	128/79 ± 14/10
Stroop 1	0.27 ± 0.08	17 ± 3#		0.28 ± 0.10	19 ± 3#	
Rest 2	0.21 ± 0.05	13 ± 3	129/82 ± 12/8	0.23 ± 0.09	15 ± 4	120/79 ± 24/11
Stroop 2	0.27 ± 0.05 #	17 ± 3 #	132/82 ± 17/10	0.29 ± 0.09 #	18 ± 4#	128/81 ± 14/10

BIO- Biofeedback group; CON- control group
RF- Respiratory Frequency (respiratory rate per second); Resp, respiratory rate (breaths per minute); BP Blood pressure(mmHg millimetres mercury)

Group effect

* p < 0.05	Pre rest	Systolic BP	Bio vs. CON

Time effect

# p < 0.05	Pre rest	RR	BIO pre rest < pre Stroop
			CON pre rest < pre Stroop
	Post rest	RR	BIO post rest < post Stroop
			CON post rest < post Stroop
		RF	BIO post rest < post Stroop
			CON post rest < post Stroop

HR changes throughout the trial are displayed graphically in figure 3. There was a significance difference in HR between groups over time ($F_{4, 128} = 4.2347$, $p = 0.003$), as well as a significant time effect ($F_{4, 128} = 6.8772$, $p = 0.000$). HR decreased significantly in the CON group pre Stroop to the intervention (p = 0.000). In the BIO group there was a significant decrease from both the pre Stroop to the post rest period (p=0.002), and intervention to post rest (p=0.04).

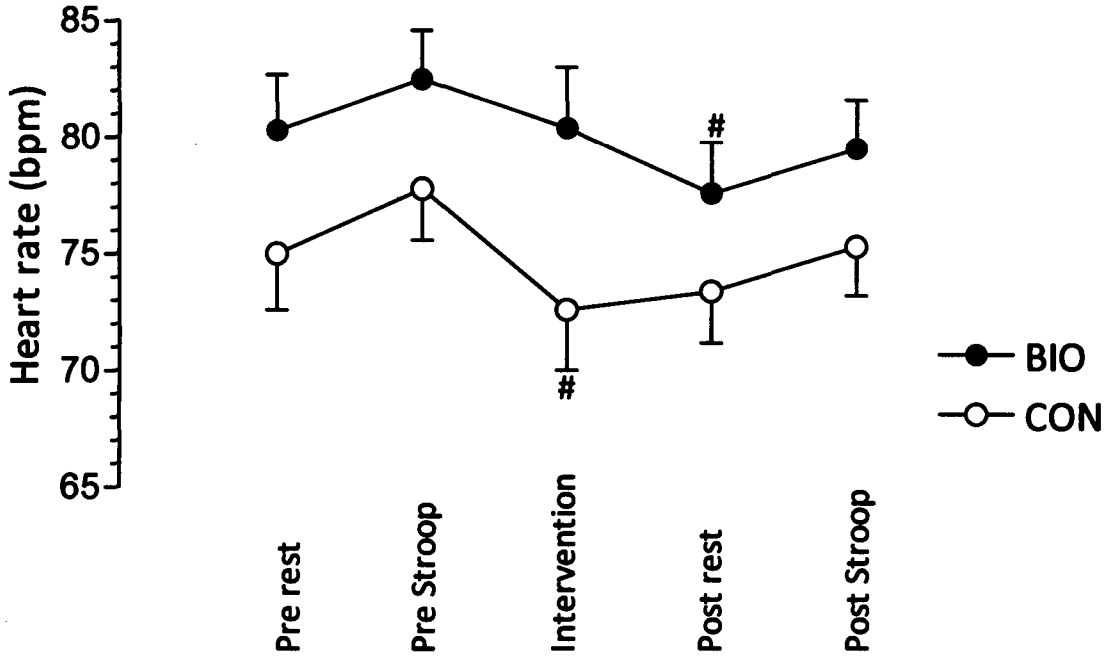


Figure 3: Heart rate changes throughout the trial between the BIO (n = 19) and CON (n = 20) group.

Abbreviations: BIO, biofeedback group; CON, control group; bpm, beats per minute.

Time effect	# p < 0.005	CON	Pre Stroop > intervention
		BIO	Pre Stroop > Post rest
			Intervention > Post rest

HRV data is displayed on table 5. The intervention data was removed from table 5 for ease of interpretation and displayed graphically in figure 4 as a carry-over effect from the intervention period into the post rest period.

HF power remained similar in the BIO group. The CON group had a significant decrease in HF power from the pre rest period to pre Stroop (p = 0.013) and from the post rest to post Stroop (p=0.022).

Both the BIO and CON groups displayed significant decreases from pre rest to pre Stroop in the LF power (p = 0.000 for both groups) as well as from post rest to post Stroop (p=0.000 for both groups).

TF power had a significant overall interaction effect ($F_{4,128} = 3.5281$, p = 0.009), as well as a significant time effect ($F_{4,128} = 32.286$, p = 0.000). Both groups displayed a decrease in TF

power from pre rest to pre Stroop (BIO, p= 0.002 and CON, p= 0.000) and from post rest to post Stroop (BIO, p= 0.017 and CON, p= 0.000).

A significant time effect was displayed in the SDNN measures ($F_{4,124} = 27.520$, $p = 0.000$). SDNN significantly decreased from pre rest to pre Stroop in the CON group (p=0.000), and BIO group (p=0.030). SDNN also significantly decreased from post rest to post Stroop in both groups (BIO, p= 0.001 and CON, p=0.000).

RMSSD showed a significant decrease from pre rest to pre Stroop in both the CON (p=0.000) and BIO (p=0.029) groups. RMSSD decreased significantly in the CON group form the post rest to post Stroop (p=0.001) period, this was not demonstrated in the BIO group.

Entropy displayed a significant interaction effect ($F_{4, 128} = 10.765$, $p = 0.000$). In the CON group entropy increased significantly from pre rest to pre Stroop (p=0.005). Entropy increased from post rest to post Stroop in both the BIO (p=0.000) and CON (p=0.007) group.

9.2.3 Carry over effect from intervention into post Rest

The HF power was significantly lower in the BIO group during post rest period after the intervention (p=0.05) in comparison to the CON group. TF power was significantly decreased in the BIO group from the intervention to post rest (p=0.005), this effect was not seen in the CON group. Entropy during the BIO intervention was significantly lower than during the CON intervention ($p \leq 0.000$) (Figure 4).

The data of five subjects were excluded from the HRV analyses due to equipment failure during recording.

Table 5. Heart rate variability data recordings throughout the experimental trial in the EXP (n=23) and CON (n=23).

	BIO				CON			
	Pre rest	Pre Stroop	Post rest	Post Stroop	Pre rest	Pre Stroop	Post rest	Post Stroop
LF Power	7 ± 0.83	6.25 ± 0.8 3#	6.94 ± 0,81	6.13 ± 0.83 #	7.29 ± 0.92	6.18 ± 0.89 #	7.45 ± 0.71	6.33 ± 0.79 #
HF Power	5.38 ± 0.86	4.95 ± 1.01	5.14 ± 0.76	4.96 ± 0.87	5.97 ±1.11	5.49 ± 1.08 #	6.23 ± 0.91*	5.62 ± 0.89 #
TF Power	7.2 ± 0.8	6.5 ± 0.9#	7.1 ± 0.7	6.5 ± 0.8#	7.7 ± 0.9	6.7 ± 0.9#	7.7 ± 0.7	6.8 ± 0.7#
SD NN	52.2 ± 4.4	39.0 ± 3.5 #	53.5 ± 3.7	37.4 ± 3.2 #	67.4 ± 6.2	43.8 ± 3.9 #	70.4 ± 5.0	43.9 ± 3.2 #
RMSSD	27.8 ± 15.6	21.2 ± 12.9 #	23.9 ± 9.7	21.4 ± 10.6	36.9 ± 19.5	28.3 ± 15.3 #	39.7 ± 16.3	29.4 ± 12.3 #
Entropy	0.98 ± 0.10	1.05 ± 0.14	0.94 ± 0.12	1.10 ± 0.13#	1.05 ± 0.11	1.15 ± 0.10 #	1.05 ± 0.13	1.16 ± 0.09 #

BIO- Biofeedback group; CON- control group

HF- High frequency; LF- Low frequency; Square root of the mean squared differences of successive NN intervals (RMSSD); milliseconds (ms)

Group effect

* p < 0.05	Post Rest	HF	BIO < CON

Time effect

	Pre Rest vs. pre Stroop		Post rest vs. post Stroop	
	BIO		**BIO**	
# p < 0.05	LF, TF, SDNN, RMSSD	pre rest < pre Stroop	LF, TF, SD NN	post rest < post Stroop
	CON		Entropy	post rest > post Stroop
	LF, HF, TF, SD NN, RMSSD	pre rest < pre Stroop	**CON**	
	Entropy	pre rest > pre Stroop	LF, HF, TF, SD NN, RMSSD	post rest < post Stroop
			Entropy	post rest > post Stroop

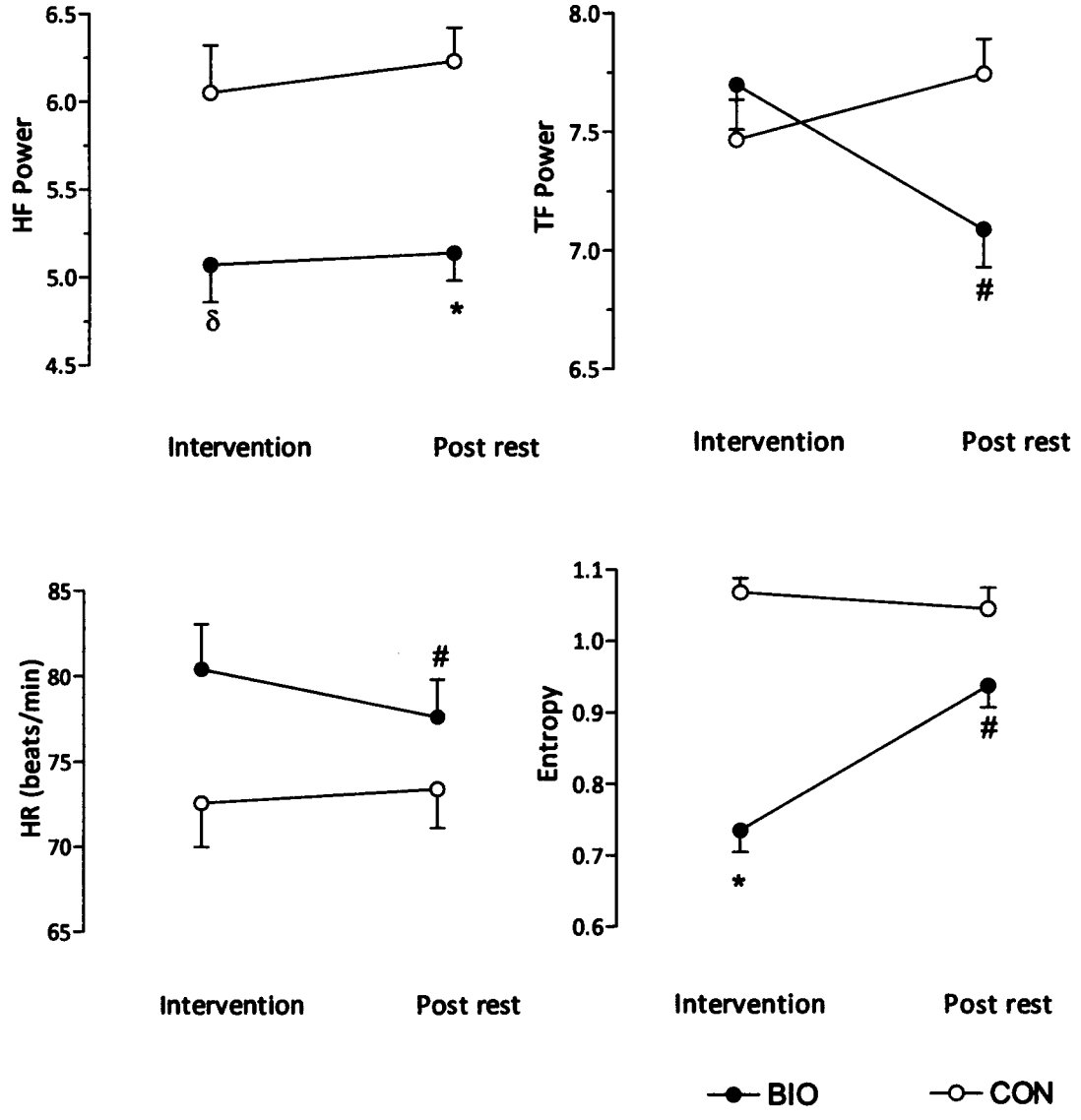


Figure 4: Carry over effect from the intervention to the post rest period in HR, HF power, TF power and Entropy between the BIO (n = 19) and CON (n = 20) group.

Abbreviations: BIO, biofeedback group; CON, control group; HR, heart rate; HF, high frequency and TF, total frequency

Group effect

* $p < 0.05$ BIO vs. CON

δ trend $p = 0.058$

Time effect

# $p < 0.05$ Intervention vs. post rest

9.2.4 Cognitive performance

Reaction time

A time effect was observed, both groups significantly decreased their reaction time ($F_{1, 37}$ = 55.185, p = 0.000) when performing the second modified Stroop task. The BIO and CON groups both had similar decreases (BIO group: 1.24 ± 0.18s vs. 1.13 ± 0.15s, p= 0.000, pre vs. post; CON group 1.35 ± 0.19s vs. 1.24 ± 0.16s, p= 0.002, pre vs. post).

Mistakes made

There were no differences between groups in the total amount of mistakes, amount of square mistake or amount of word mistakes. The data from two subjects (one in the BIO group and one in the CON group) were excluded as the standard deviation greatly differed from that of the mean.

10. Discussion and conclusion

10.1 Familiarisation

The familiarisation data was documented in order to establish basic information of the participant's tested as well as to familiarise them to the testing protocol. The STAIT was utilized in the familiarisation trial in order to establish levels of anxiety; all testing was done at the different places of work and during office hours in order to establish work-related anxiety. In a review article which aimed at establishing the reliability of STAIT and STAIS scores, it was stated that higher scores were recorded with high external stress levels compared to lower external stress level[99]. The mean scores were established as high stimulus at a score of 45.10 versus 39.19 for a low stimulus (Cohen's d = .91)[99]. In a recent study which measured a HRV biofeedback device in a stressed work population STAIT scores were utilized to measure perceived life stress, the STAIT scores were 48 ± 6 and 44 ± 7 respectively in the two groups[96]. In our study the STAIT scores were slightly lower (36 ± 8 and 36 ± 9, in the BIO and CON groups' respectively). According to the reliability data[99] our subject population is classified in the low stimulus, or low anxiety group. However all participants were volunteers who readily got involved in research aimed at assessing and reducing work-related stress.

SDNN reflects all the cyclic components in the heart rate responsible for variability in the period of recording[38]. The inverse correlation between SDNN and STAIT scores imply that increases in anxiety (STAIT score) is related to a decrease in HRV measures (Figure 2). This corresponds with the current literature as increases in stress and anxiety affect HRV negatively, reducing variability[34, 42]. This was also displayed in the inverse relationship between RMSSD and STAIT scores (Figure 2).

A significant inverse correlation was also found between RMSSD and BP, indicating that increases in BP are correlated with decreases in HRV (Figure 2); this has been established in previous comparable research[39].

This suggests that stress reduction interventions should be aimed at increasing HRV measures in order to obtain relative decreases in anxiety and BP.

10.2 Intervention trial

Resonance between the main sources of cardiac vagal drive namely, the baroreflex and respiratory sinus arrhythmia (RSA) can be achieved when using the biofeedback device correctly[93]. This breathing rate is referred to as resonant frequency, it differs between individuals but is usually between 4.5 and 7 breathes per minute. The implementation of the biofeedback device was successful in the BIO group, as they demonstrated a mean respiratory rate of 7±3 breaths per minute during the intervention.

The key finding in this study lies in the carry over effect of the two interventions. Differences in physiological variables were evident between the two groups. HR (Figure 3) dropped significantly from pre Stroop to the intervention period in the CON group; this was expected given that classical music has been shown to cause increased vagal modulation and is associated with decreases in HR, BP and respiratory rate[22, 23]. The RF on the other hand did not change during the music intervention.

The BIO group did not display this effect; HR throughout the intervention remained similar to the pre Stroop HR and only dropped significantly during the post rest period, which illustrates a disparity in the modulation of HR in the BIO and CON groups. The mechanism of classical music works though a relaxation response; the mechanism appears to be a direct physiologic effect through the ANS by decreasing BP, HR and respiratory rate [22-24]. Classical music has also been shown to decrease SNS activation[30], reduce anxiety and may have an effect on the cognitive component in the stress response[25].

The BIO group displayed a significant decrease in TF power from the intervention to the second rest period, an effect which was not seen in the CON group.
Furthermore, in the BIO group HF power did not change from the intervention period to the post rest period, and in fact the HF power in the post rest period was significantly lower in the BIO group compared to the CON group (Figure 4). In this regard, there was also a trend ($P = 0.058$) for the HF power to be lower in the BIO than in the CON groups during the intervention. HF power is modulated by efferent vagal activity. This demonstrates a selective withdrawal of vagal modulation during the intervention in the BIO group which was not evident in the CON group. Porges found that nicotinic blockade selectively blocks

the RSA without affecting the LF power[100]. In contrast both low and respiratory frequencies can be removed using atropine to block the muscarinic receptors[100]. The use of the HRV biofeedback device may elicit a similar response, causing withdrawal of HF vagal modulation only.

As illustrated graphically in Figure 4 the physiological and HRV measures of the CON group remained essentially unchanged from the intervention period to the post rest period. The changes displayed between pre and post intervention in the CON group are displayed in table 5 and are unanimous between time periods. All measures of HRV displayed the same pattern of stress reactivity pre and post intervention. LF power, HF power, TF power, RMSSD and SDNN all decreased significant from the rest period to the Stroop Task, both pre and post intervention. Entropy on the other hand increased significantly from the rest period to the Stroop task, both pre and post intervention in the CON group. The CON group displayed the relaxation response[22, 23] during the intervention; HRV measures increased, while HR decreased.

The BIO group demonstrated several differences in HRV measures to stress reactivity pre and post intervention. The BIO group did not display the initial decrease in HF power from pre rest to pre Stroop. This was not expected, as both the BIO and CON were expected to have the same initial responses. The BIO group did however start with a significantly higher systolic BP, which may illustrate increased performance anxiety that affected the HF power of the cardiac spectrogram. This was the only disparity between the pre intervention HRV measures to stress reactivity.

The BIO group had significantly lower HRV entropy during the intervention compared to the CON group (Figure 4). Approximate entropy is an estimation technique and is a nonlinear measure of HRV[101]. It estimates the average uncertainty of the HRV. If the intervals between the heart beats are equally spaced, entropy is low. Whereas, if the beat-to-beat intervals are randomly distributed, the next beat cannot be easily anticipated and entropy increases [102]. The biofeedback device induced a significant decrease in entropy during the intervention, highlighting the effect it has on the regularity of the beat-to-beat interval.

The BIO group did display similar decreases in certain HRV measures to stress reactivity as found in the CON group. Both pre and post intervention, SDNN, LF power and TF power

decreased from rest to Stroop. However, in contrast to the pre rest to pre Stroop period, the BIO group did not show a decrease in the RMSSD from the post rest to the post Stroop period. The RMSSD measures remained similar to the pre Stroop value, which significantly decreased from the pre rest period. This further illustrates the decreased HRV evident in the post rest period (carry-over effect) in the BIO group.

The BIO subjects had significant decreases in anxiety (STAIS) and significant increases in feelings' of relaxation (SRSI3) after the testing protocol, which was not found in the CON group. The BIO subjects also felt significantly more energised after the intervention compared to both the CON group and to their pre intervention measures.

The carry over HRV pattern facilitated by the biofeedback intervention during the post rest period illustrated significant vagal withdrawal; this finding is seemingly contrary to their subjective feelings post testing. Increased vagal modulation (for which increased HRV is a marker) is responsible for the relaxation response[22, 23, 103], which results in decreases in HR, BP and RF. The CON group displayed this relaxation response, both during the music intervention and the post rest period, showing increases in all HRV measures and a decrease in HR, but they did not feel more relaxed or less anxious after the testing.

10.3 Cognitive performance

Mature mental functioning is characterised by holding information, exercising self-control and quickly and flexibly adapting to changing situations[104]. These are referred as working memory, inhibition, and cognitive flexibility respectively[104]. Mental set shifting, which is required in the modified Stroop task, is a fundamentally difficult executive control as it cannot be done "automatically". It requires both working memory and inhibition; relevant information must be responded to and irrelevant information needs to be suppressed[86]. The modified Stroop task evaluates the flexibility of the control of cognitive processes and behaviour, evaluates psychological capacities of an individual and lastly allows for a measure of reaction time of the task[83, 84]. It is vital to observe that the nature of the stimulus plays an important role in attention selection[85]. Our modified Stroop Task also evaluated working memory, i.e. the continuous counting of squares forced the subjects to change their mental

focus between squares and words and also tested their ability to inhibit prepotent responses[56, 96]. Both the BIO and CON group interventions improved their reaction time over time, and this is not likely attributable to skill learning. It has been shown that a learning effect is evident between the first and second modified Stroop Tasks but not between the second and third, as established in a previous validation trial[105]. This is why it is vital for participants to complete a full familiarisation trial beforehand. There was no significant difference between the two groups showing that both interventions had a positive effect on reaction time between pre Stroop and post Stroop.

Working memory was not changed significantly between groups or over time, which demonstrates that neither group's intervention had any effect on working memory. Present moment attention was not significantly altered; this may be due to the high number of participants, in both groups, who made no mistakes in the initial modified Stroop Task which left little room for improvement. The speed accuracy trade-off is the inverse relationship between speed and accuracy[105-107]. With increasing accuracy you have a decrease in reaction time and vice versa. An important factor about the speed accuracy trade-off is that they are the main determinants of one another and are negatively related. Thus choosing to increase reaction time one's accuracy necessarily decreases[107]. It is critical to note that both the BIO and CON subjects decreased their reaction times (responded faster) and maintained their mistakes in the modified Stroop Tasks. Age and sex has demonstrated effects on cognitive performance[108, 109], although participants were randomised into groups, there were differences between the groups. These factors may have affected cognitive performance.

The main limitation to this study was the level of stress observed in the participants; the Spielberger STAI-T values were slightly below the values established as "high stress" found in previous research. Participants may experience work related stress but the STAI-T questionnaire was unable to depict this. Another limitation was the differences in age between the groups which may have affected the cognitive performance test. Males and females were included in this study which may also have an effect on response to stress as well as aspects of cognitive functioning, which may impact cognitive performance results.

11 Conclusion

In conclusion, contrasting physiological reactivity to stress was found between the BIO and CON groups. Both groups displayed seemingly conflicting physiological versus subjective measures. The BIO subjects felt significantly more relaxed and less anxious after the testing, even though the carry over effect of their intervention facilitated significant decreases in HRV during the post rest period. This suggests that the ability to maintain vagal withdrawal during both the 5 min preparation period and during the execution of a stressful cognitive task facilitates subjective relaxation and decreases felt anxiety after task completion. This effect was displayed together with increases in cognitive performance as evidenced by quicker reaction times without making more mistakes. The CON subjects also displayed the same increased cognitive performance, however they showed no improvement in their subjective measures of relaxation and anxiety despite the fact they effectively implemented the relaxation response during the intervention period and in the 5 min rest period immediately prior to the post Stroop Task. Being prepared for optimal performance is thus a balance between over excitation and excess relaxation. While the CON group displayed significant increases in physiological measures linked with relaxation, they did not report this subjectively after task completion.

Music shows positive physiological response to reducing stress, which has been previously highlighted in research. However in this study aimed at establishing which intervention would optimally prepare you for a stressful performance environment, and although there were no significant differences in cognitive performance, the physiology which resulted from the use of the biofeedback devise may been seen as more optimal when dealing with stress.

12. Practical application

Stress is an independent contributing risk factor for the development of CVD[1, 3, 5, 9-12]. Job strain is one of the most common causes of stress and has also been shown to contribute to the development of CVD [1, 3, 5, 9-12]. Stress reduction techniques are vital in order reduce adverse effects and decrease risk. It is critical to highlight how stress is reduced, as interventions which only display an effect during implementation may not be as effective as techniques which have lasting effects. Stress reactivity in autonomic variables occurs with

exposure to acute stress[34]. Interventions which aim at acutely modulating the ANS in preparation to stress may be a more effective way to cope with stress. The biofeedback intervention engendered the same improvements in cognitive performance, but the participants had significant positive changes in subjective stress perceptions.

Stress is a response, or process and not strictly an emotion[110]. Not all stress is viewed negatively; stress may be a motivating force to action[110]. However the psychological or physiological hyper-arousal which is caused by a perceived imbalance between the situational demands and the coping mechanisms, leads to a wide range of subjective feelings[110]. The music intervention only facilitated a relaxation response (decreased HR, BP and RF), whereas the biofeedback device engendered a significant decrease in subjective anxiety and an increase in relaxation. This implies that the BIO intervention may "utilise" stress positively in the preparation for a stressful task. Altering perceived stress may be as important as effecting physiological variables; in the long term the subjective feelings of stress may manifest in increased sympathetic drive, an associated risk factor for life style diseases[16].

Interventions at the work place, although necessary are sometimes difficult to implement. Many barriers from both managers and employees need to be overcome before effective implementation can occur. These barriers include lack of access to company management and perceptions of contract vulnerability[111]. Personal problems such as coping with change, time management as well as fear for loss of job if unable to change[111]. The crucial aspect for the program is management support[111]. Education about workplace stress interventions may be most effectively directed integrated with company level work groups[111]. A combination of person-focused and organization-focused approaches is the most promising[111].

Reference List

1. Fauvel JP, Quelin P, Ducher M, Rakotomalala H, Laville M. Perceived job stress but not individual cardiovascular reactivity to stress is related to higher blood pressure at work. Hypertension 2001; 38:71-75.

2. Rosmond R, Dallman MF, Bjorntorp P. Stress-related cortisol secretion in men: relationships with abdominal obesity and endocrine, metabolic and hemodynamic abnormalities. J Clin Endocrinol Metab 1998; 83:1853-1859.

3. Chandola T, Heraclides A, Kumari M. Psychophysiological biomarkers of workplace stressors. Neurosci Biobehav Rev 2010; 35:51-57.

4. Lucini D, Riva S, Pizzinelli P, Pagani M. Stress management at the worksite: reversal of symptoms profile and cardiovascular dysregulation. Hypertension 2007; 49:291-297.

5. Chandola T, Britton A, Brunner E, Hemingway H, Malik M, Kumari M, Badrick E, Kivimaki M, Marmot M. Work stress and coronary heart disease: what are the mechanisms? Eur Heart J 2008; 29:640-648.

6. Lucini D, Norbiato G, Clerici M, Pagani M. Hemodynamic and autonomic adjustments to real life stress conditions in humans. Hypertension 2002; 39:184-188.

7. Hughes JW, Stoney CM. Depressed mood is related to high-frequency heart rate variability during stressors. Psychosom Med 2000; 62:796-803.

8. Saito K, Hiya A, Uemura Y, Furuta M. Clinical training stress and autonomic nervous function in female medical technology students: analysis of heart rate variability and 1/f fluctuation. J Med Invest 2008; 55:227-230.

9. Kang MG, Koh SB, Cha BS, Park JK, Woo JM, Chang SJ. Association between job stress on heart rate variability and metabolic syndrome in shipyard male workers. Yonsei Med J 2004; 45:838-846.

10. Riese H, van Doornen LJ, Houtman IL, de Geus EJ. Job strain in relation to ambulatory blood pressure, heart rate, and heart rate variability among female nurses. Scand J Work Environ Health 2004; 30:477-485.

11. Vrijkotte TG, van Doornen LJ, de Geus EJ. Overcommitment to work is associated with changes in cardiac sympathetic regulation. Psychosom Med 2004; 66:656-663.

12. Schubert C, Lambertz M, Nelesen RA, Bardwell W, Choi JB, Dimsdale JE. Effects of stress on heart rate complexity--a comparison between short-term and chronic stress. Biol Psychol 2009; 80:325-332.

13. Le Fevre M, Matheny J, Kolt GS. Eustress, distress, and interpretation in occupational stress. Journal of Managerial Psychology 2003; 18:726-744.

14. Cooper CL, Carwright S. Healthy Mind; Healthy Organization - A Proactive Approach to Occupational Stress. Human Relations 1994; 47:455-471.

15. Mezzacappa ES, Kelsey RM, Katkin ES, Sloan RP. Vagal rebound and recovery from psychological stress. Psychosom Med 2001; 63:650-657.

16. Moravec CS. Biofeedback therapy in cardiovascular disease: rationale and research overview. Cleve Clin J Med 2008; 75 Suppl 2:S35-S38.

17. Melillo P, Bracale M, Pecchia L. Nonlinear Heart Rate Variability features for real-life stress detection. Case study: students under stress due to university examination. Biomed Eng Online 2011; 10:96.

18. McKee MG. Biofeedback: an overview in the context of heart-brain medicine. Cleve Clin J Med 2008; 75 Suppl 2:S31-S34.

19. Weiss T, Engel BT, Pickering TG, ., Mauck HP, Miller NE, Hockman CH, Lown B, Pickering T, Gorham G. Learnt voluntary control of heart rate and rhythm. Br Med J 1977; 1:1491.

20. Moravec CS, McKee MG. Biofeedback in the treatment of heart disease. Cleve Clin J Med 2011; 78 Suppl 1:S20-S23.

21. Lehrer PM, Vaschillo E, Vaschillo B, Lu SE, Eckberg DL, Edelberg R, Shih WJ, Lin Y, Kuusela TA, Tahvanainen KU, Hamer RM. Heart rate variability biofeedback increases baroreflex gain and peak expiratory flow. Psychosom Med 2003; 65:796-805.

22. Kemper KI, Danhauer SC. Music as Therapy. Featured CME Topic: Complementary and Alternative Medicine 2005;282-288.

23. Lee OK, Chung YF, Chan MF, Chan WM. Music and its effect on the physiological responses and anxiety levels of patients receiving mechanical ventilation: a pilot study. J Clin Nurs 2005; 14(5):609-620.

24. Camara JG, Ruszkowski JM, Worak SR. The effect of live classical piano music on the vital signs of patients undergoing ophthalmic surgery. Medscape J Med 2008; 10(6):149.

25. Burns LJ, Labbe E, Arke B, Capeless K, Cooksey B, Steadman A, Gonzales C. The effect of different types of music on perceived and physilogical measures of stress. Journal of Music Therapy 2002; XXXIX:101-116.

26. Sigafus P. Heart rate variability biofeedback and mindfulness: a functional neuroimaging study. Cleve Clin J Med 2011; 78 Suppl 1:S102.

27. Mancia G, Zanchetti A, Pedotti A, di Rienzo M, Grassi G, Ferreri A, Gregorini L, Parati G, Pomidossi G, Bertineri G. Blood Pressure and Heart Rate Variabilities in Normotensive and Hypertensive Human Beings. Journal of the American heart association 1983; 53:96-104.

28. Rajendra AU, Paul JK, Kannathal N, Lim CM, Suri JS. Heart rate variability: a review. Med Biol Eng Comput 2006; 44(12):1031-1051.

29. Kim GM, Woo JM. Determinants for heart rate variability in a normal Korean population. J Korean Med Sci 2011; 26:1293-1298.

30. Acharya UR, Kannathal N, Sing OW, Ping LY, Chua T. Heart rate analysis in normal subjects of various age groups. Biomed Eng Online 2004; 3:24.

31. Saul JP. Beat-to-Beat Variations of Heart Rate Reflect Modulation of Cardiac Autonomic Outflow. NIPS 1990; 5:32-37.

32. Wu SD, Lo PC. Inward-attention meditation increases parasympathetic activity: a study based on heart rate variability. Biomed Res 2008; 29:245-250.

33. van Ravenswaaij-Arts CMA, Kollée LAA, Hopman JCW, Stoelinga GBA, van Geijn HP. Heart rate variability. Annals of Internal Medicine 1993; 118:436-447.

34. Dikecligil GN, Mujica-Parodi LR. Ambulatory and challenge-associated heart rate variability measures predict cardiac responses to real-world acute emotional stress. Biol Psychiatry 2010; 67:1185-1190.

35. Saul JP, Rea RF, Eckberg DL, Berger RD, Cohen RJ. Heart rate and muscle sympathetic nerve variability during reflex changes of autonomic activity. Heart Circ Physio 1990; 27:713-721.

36. Sluiter JK, Guijt AM, Frings-Dresen MH. Reproducibility and validity of heart rate variability and respiration rate measurements in participants with prolonged fatigue complaints. Int Arch Occup Environ Health 2009; 82:623-630.

37. Hill LK, Siebenbrock A. Are all measures created equal? Heart rate variability and respiration - biomed 2009. Biomed Sci Instrum 2009; 45:71-76.

38. Task Force of The European Society of Cardiology and The North AmericanSociety of Pacing and Electrophysiology. Heart rate variability: Standards of measurement, physiological interpretation, and clinical use. European Heart Journal 17, 354-381. 1996.

Ref Type: Journal (Full)

39. Bernardi L, Wdowczyk-Szulc J, Valenti C, Castoldi S, Passino C, Spadacini G, Sleight P. Effects of controlled breathing, mental activity and mental stress with or without verbalization on heart rate variability. J Am Coll Cardiol 2000; 35(6):1462-1469.

40. Furlan R, Guzzetti S, Crivellaro W, Tinelli M, Cerutti S, Lombardi F, Pagani M, Malliani A. Continuous 24-hour assessment of the neural regulation of systemic arterial pressure and RR variabilities in ambulant subjects. Circulation 1990; 81:537-547.

41. Hejjel L, Gal I. Heart rate variability analysis. Acta Physiol Hung 2001; 88:219-230.

42. Taylor AG, Goehler LE, Galper DI, Innes KE, Bourguignon C. Top-down and bottom-up mechanisms in mind-body medicine: development of an integrative framework for psychophysiological research. Explore (NY) 2010; 6:29-41.

43. Bernardi L, Porta C, Gabutti A, Spicuzza L, Sleight P. Modulatory effects of respiration. Auton Neurosci 2001; %20;90(1-2):47-56.

44. Eckberg DL, Nerhed C, Wallin G. Respiratory Modulation of muscle Sympathetic and Vagal Cardiac outflow in man. J Physiol 1985;181-196.

45. Shields RW, Jr. Heart rate variability with deep breathing as a clinical test of cardiovagal function. Cleve Clin J Med 2009; 76 Suppl 2:37-40.

46. Akselrod S, Gordon D, Ubel FA, Shannon DC, Cohen RJ. Power spectrum of heart rate fluctuation: Aquantitive probe of beat-to-beat cardiovascular control. Science 1981; 213:220-222.

47. Novak V, Novak P, De Champlain J, Le Blanc R, Martin R, Nadeau R. Influence of respiration on heart rate and blood pressure fluctuations. J Appl Physiol 1993; 74:617-626.

48. Jerath R, Edry JW, Barnes VA, Jerath V. Physiology of long pranayamic breathing: neural respiratory elements may provide a mechanism that explains how slow deep breathing shifts the autonomic nervous system. Med Hypotheses 2006; 67:566-571.

49. Jovanov E. On Spectral Analysis of Heart Rate Variability during Very Slow Yogic Breathing. Conf Proc IEEE Eng Med Biol Soc 2005; 3:2467-2470.

50. Fang Y, Sun JT, Li C, Poon CS, Wu GQ. Effect of different breathing patterns on nonlinearity of heart rate variability. Conf Proc IEEE Eng Med Biol Soc 2008; 2008:3220-3223.

51. Lucini D, Bertocchi F, Malliani A, Pagani M. A controlled study of the autonomic changes produced by habitual cigarette smoking in healthy subjects. Cardiovasc Res 1996; 31(4):633-639.

52. Li Z, Snieder H, Su S, Ding X, Thayer JF, Treiber FA, Wang X. A longitudinal study in youth of heart rate variability at rest and in response to stress. Int J Psychophysiol 2009; 73:212-217.

53. Migliaro ER, Contreras P, Bech S, Etxagibel A, Castro M, Ricca R, Vicente K. Relative influence of age, resting heart rate and sedentary life style in short-term analysis of heart rate variability. Braz J Med Biol Res 2001; 34:493-500.

54. Rose AK, Duka T. The influence of alcohol on basic motoric and cognitive disinhibition. Alcohol Alcohol 2007; 42(6):544-551.

55. Jongen EM, Jonkman LM. Effects of concurrent working memory load on distractor and conflict processing in a name-face Stroop task. Psychophysiology 2010.

56. Heitz RP, Engle RW. Focusing the spotlight: individual differences in visual attention control. J Exp Psychol Gen 2007; 136(2):217-240.

57. Kane MJ, Engle RW. Working-memory capacity and the control of attention: the contributions of goal neglect, response competition, and task set to Stroop interference. J Exp Psychol Gen 2003; 132(1):47-70.

58. Long DL, Prat CS. Working memory and stroop interference: an individual differences investigation. Mem Cognit 2002; 30(2):294-301.

59. Hansen AL, Johnsen BH, Thayer JF. Vagal influence on working memory and attention. Int J Psychophysiol 2003; 48(3):263-274.

60. Hansen AL, Johnsen BH, Sollers JJ, III, Stenvik K, Thayer JF. Heart rate variability and its relation to prefrontal cognitive function: the effects of training and detraining. Eur J Appl Physiol 2004; 93(3):263-272.

61. Davy KP, DeSouza CA, Jones PP, Seals DR. Elevated heart rate variability in physically active young and older adult women. Clin Sci (Lond) 1998; 94(6):579-584.

62. Lee KH, Yoon K, Ha M, Park J, Cho SH, Kang D. Heart rate variability and urinary catecholamines from job stress in korean male manufacturing workers according to work seniority. Ind Health 2010; 48:331-338.

63. Caspi A, Sugden K, Moffitt TE, Taylor A, Craig IW, Harrington H, McClay J, Mill J, Martin J, Braithwaite A, Poulton R. Influence of Life Stress on Depression: Moderation by a Polymorphism in the 5-HTT Gene. Science 2003; 301:386-389.

64. Brown RP, Gerbarg PL. Sudarshan Kriya Yogic breathing in the Treatment of Stress, Anxiety, and Depression: Part 2-Clinical Applications and Guidelines. The journal of Alternative and Complementary Medicine 2005; 11:711-717.

65. Ernst E, Rand JI, Stevinson C. Complemetary Therapies for Depression. American Medical Association 1998; 55:1026-1032.

66. Hamer M, Steptoe A. Association between physical fitness, parasympathetic control, and proinflammatory responses to mental stress. Psychosom Med 2007; 69:660-666.

67. Hall M, Vasko R, Buysse D, Ombao H, Chen Q, Cashmere JD, Kupfer D, Thayer JF. Acute stress affects heart rate variability during sleep. Psychosom Med 2004; 66:56-62.

68. Schwarz AM, Schachinger H, Adler RH, Goetz SM. Hopelessness is associated with decreased heart rate variability during championship chess games. Psychosom Med 2003; 65:658-661.

69. Fourie, M. M., Rauch, H. G. L., Morgan, B. E., Ellis, G. R., Jordaan, E. R., and Thomas, K. G. F. Guilt and pride are heartfelt, but not equally so. Psychophysiology 48, 888-899. 2011.

Ref Type: Journal (Full)

70. Pieper S, Brosschot JF, van der Leeden R, Thayer JF. Cardiac effects of momentary assessed worry episodes and stressful events. Psychosom Med 2007; 69:901-909.

71. Marques AH, Silverman MN, Sternberg EM. Evaluation of stress systems by applying noninvasive methodologies: measurements of neuroimmune biomarkers in the sweat, heart rate variability and salivary cortisol. Neuroimmunomodulation 2010; 17:205-208.

72. Thayer JF, Verkuil B, Brosschot JF, Kampschroer K, West A, Sterling C, Christie IC, Abernethy DR, Sollers JJ, Cizza G, Marques AH, Sternberg EM. Effects of the physical work environment on physiological measures of stress. Eur J Cardiovasc Prev Rehabil 2010; 17:431-439.

73. Sakakibara M, Hayano J. Effect of slowed respiration on cardiac parasympathetic response to threat. Psychosom Med 1996; 58:32-37.

74. Prinsloo GE, Rauch HGL, Lambert MI, Muench F, Noakes TD, Derman WE. The Effect of Short Duration Heart Rate Variability (HRV) Biofeedback on Cognitive Performance During Laboratory Induced Cognitive Stress. Applied Cognitive Psycology 2010.

75. Wahbeh H, Elsas SM, Oken BS. Mind-body interventions: applications in neurology. Neurology 2008; 70:2321-2328.

76. Lehrer PM, Vaschillo E, Vaschillo B, Lu SE, Eckberg DL, Edelberg R, Shih WJ, Lin Y, Kuusela TA, Tahvanainen KU, Hamer RM. Heart rate variability biofeedback increases baroreflex gain and peak expiratory flow. Psychosom Med 2003; 65:796-805.

77. Kaushik RM, Kaushik R, Mahajan SK, Rajesh V. Effects of mental relaxation and slow breathing in essential hypertension. Complement Ther Med 2006; 14:120-126.

78. Grossman E, Grossman A, Schein MH, Zimlichman R, Gavish B. Breathing-control lowers blood pressure. J Hum Hypertens 2001; 15:263-269.

79. Chiang LC, Ma WF, Huang JL, Tseng LF, Hsueh KC. Effect of relaxation-breathing training on anxiety and asthma signs/symptoms of children with moderate-to-severe asthma: a randomized controlled trial. Int J Nurs Stud 2009; 46:1061-1070.

80. Schein MH, Gavish B, Herz M, Rosner-Kahana D, Naveh P, Knishkowy B, Zlotnikov E, Ben-Zvi N, Melmed RN. Treating hypertension with a device that slows and regularises breathing: a randomised, double-blind controlled study. J Hum Hypertens 2001; 15:271-278.

81. Lee JS, Lee MS, Lee JY, Cornelissen G, Otsuka K, Halberg F. Effects of diaphragmatic breathing on ambulatory blood pressure and heart rate. Biomed Pharmacother 2003; 57 Suppl 1:87s-91s.

82. Jagomagi K, Raamat R, Talts J, Lansimies E, Jurvelin J. Effect of deep breathing test on finger blood pressure. Blood Press Monit 2003; 8(5):211-214.

83. Stroop JR. Studies of Interference in serial verbal reactions. Journal of experimental Psychology: Human perception and Performance 1935; 18:643-662.

84. Bugg JM, Jacoby LL, Toth JP. Multiple levels of control in the Stroop task. Mem Cognit 2008; 36(8):1484-1494.

85. Chen Z. Attentional focus, processing load, and Stroop interference. Perception & Psychophysics 2003; 65:888-900.

86. Polk TA, Drake RM, Jonides JJ, Smith MR, Smith EE. Attention enhances the neural processing of relevant features and suppresses the processing of irrelevant features in humans: a functional magnetic resonance imaging study of the Stroop task. J Neurosci 2008; 28(51):13786-13792.

87. Smiley-Oyen AL, Lowry KA, Francois SJ, Kohut ML, Ekkekakis P. Exercise, fitness, and neurocognitive function in older adults: the "selective improvement" and "cardiovascular fitness" hypotheses. Ann Behav Med 2008; 36(3):280-291.

88. Hamer M, Steptoe A. Association between physical fitness, parasympathetic control, and proinflammatory responses to mental stress. Psychosom Med 2007; 69(7):660-666.

89. Domier CP, Monterosso JR, Brody AL, Simon SL, Mendrek A, Olmstead R, Jarvik ME, Cohen MS, London ED. Effects of cigarette smoking and abstinence on Stroop task performance. Psychopharmacology (Berl) 2007; 195(1):1-9.

90. Xu J, Mendrek A, Cohen MS, Monterosso J, Simon S, Jarvik M, Olmstead R, Brody AL, Ernst M, London ED. Effect of cigarette smoking on prefrontal cortical function in nondeprived smokers performing the Stroop Task. Neuropsychopharmacology 2007; 32(6):1421-1428.

91. Heilman KJ, Handelman M, Lewis G, Porges SW. Accuracy of the StressEraser in the detection of cardiac rhythms. Appl Psychophysiol Biofeedback 2008; 33:83-89.

92. Muench F. The Portable StressEraser Heart Rate Variability Biofeedback Device: Background and Research. Biofeedback 2008; 36:35-39.

93. Vaschillo E, Vaschillo B, Lehrer P. Heartbeat Synchronizes With Respiratory Rhythm Only Under Specific Circumstances. Chest 2004; 126:1385-1387.

94. Giardino ND, Chan L, Borson S. Combined Heart Rate Variability and Pulse Oximetry Biofeedback for Chronic Obstructive Pulmonary Disease: Preliminary Findings. Applied Psychophysiology and Biofeedback 2004; 29:121-133.

95. O'Brien E, Mee F, Atkins N, Thomas M. Evaluation of three devices for self-measurement of blood pressure according to the revised British Hypertension Society Protocol: The Omron HEM-705CP, Philips HP5332, and Nissei DS-175. Blood pressure monitoring 1996; 1:55-61.

96. Prinsloo, G. E., Rauch, H., Lambert, M. I., Noakes, T. D., and Derman, W. E. The effect of short duration heart rate variability (HRV) biofeedback on cognitive performance during laboratory induced cognitive stress. Applied Cognitive Psychology . 2010.

Ref Type: In Press

97. Ho KK, Moody GB, Peng CK, Mietus JE, Larson MG, Levy D, Goldberger AL. Predicting survival in heart failure case and control subjects by use of fully automated methods for deriving nonlinear and conventional indices of heart rate dynamics. Circulation 1997; 96:842-848.

98. ACSM's Guidelines for Exercise Testing and Prescription. Wolters Kluwer; Lippincott Williams & Wilkins; 2009. 47 p.

99. Barnes LL, Harp D, Jung WS. Reliability Generalization of Scores on the Spielberger State-Trait Anxiety Inventory. Educational and Psychological Measurement 2002; 62.

100. Porges, S. W. The polyvagal theory: New insights into adaptive reactions of the autonomic nervous system. Cleve.Clin.J.Med. 76, 86-90. 2009.

Ref Type: Journal (Full)

101. Hanumantha Rao, T. V. K and Ravindran, G. Entropy Estimation of Heart Rate Variability and Computation of its Multiscale Entropy. European Journal of Scientific Research 47[4], 517-530. 2010.

Ref Type: Journal (Full)

102. Wu G, Arzeno NM, Shen L, Tang D, Zhao D, Zheng D, Eckberg DL, Poon C. Chaotic Signatures of Heart Rate Variability and Its Power Spectrum in Health, Aging and Heart Failure. Aging and Heart failure 2009;2.

103. Benson H. The Relaxation Response. Mind Body Medicine: How to use your Mind for Better Health. 1993.

104. Davidson MC, Amso D, Anderson LC, Diamond A. Development of cognitive control and executive functions from 4 to 13 years: evidence from manipulations of memory, inhibition, and task switching. Neuropsychologia 2006; 44(11):2037-2078.

105. Rauch H, John L, St Clair Gibson A, Noakes TD, Vaughan C. Validation of EEG response during modified stroop colour word test. Proceedings of the Physiology Society of Southern Africa 2005.

106. Franzon M, Hugdahl K. Effects of speed vs. accuracy in vocal reaction time to visual half-field presentations of incongruent (Stroop) colour-words. Cortex 1987; 23:615-629.

107. Klein Entink RH, Fox JP, van der Linden WJ. A Multivariate Multilevel Approach to the Modeling of Accuracy and Speed of Test Takers. Psychometrika 2009; 74(1):21-48.

108. Salthouse TA. Mediation of Adult Age Differences in Cognition By reductions in working Memory and speed of processing. American Psychological Society 19910; 2:179-183.

109. Gur RC, Turetsky BI, Matsui M, Yan M, Bilker W, Hughett P, Gur RE. Sex Differences in Brain Gray and White Matter in Healthy Young Adults: Correlations with Cognitive Performance. The journal of Neuroscience 19990; 19:4065-4072.

110. Stanley RO, Burrows GD. Chapter 1 Varieties and functions of human emotion. Emotions at work. 2001.

111. Semmer NK. Job stress interventions and the organization of work. Scand J Work Environ Health 2006; 32:515-527.

Office use ONLY
Subject code: _____

Personal Details

Name: _____

Surname: _____

Contact details: (Tel.) _____

(Email) _____

Age: _____

Sex: _____

Weight: _____

Meditation history:

Do you have any previous experience or training in breathing techniques?

_____

_____

Do you practise any meditation techniques, if yes what type and how often?

_____

_____

Exercise:

How often do exercise per week? _____

How many hours do you train per week? _____

Medical History:

Are you colour blind? _____

Do you suffer from any cardiac signs or symptoms (chest pain /dizziness /nausea)?

_____

_____

Have you been diagnosed with any cardiac problems? _____

_____

Do you suffer from any psychological problems? _____

_____

Have you been diagnosed with any psychological disorders? _____

_____

Do you take any medications, if so what type? _____

_____

Have you had caffeine or a cigarette in the last four hours? _____

_____

INFORMED CONSENT

Study on Heart Rate Variability, Breathing and Movement

I have read and understood the information sheet provided to me by the investigator. The procedures and risks have also been explained verbally to me and I have had the opportunity to ask questions about the study. The exact procedures used in the trial have been thoroughly explained to me. The aims of this study are to understand the physiological benefits of a breathing technique in affecting concentration, reaction time, test performance and heart rate variability.

I am aware that my respiration rate and heart rate will be measured, that the anticipated time required is approximately 1 x 60 min session, 1 week after the 30 min training. Testing is not invasive and the methodology will not expose me to any risk.

I understand that I am free to ask questions and withdraw from the study at any time, and without reason, at any time during the testing process. I agree to the information gathered in this study to be used for research, in a statistical format only. None of my personal information may be shared.

I agree to participate in this research study of the UCT/MRC Research Unit for Exercise Science and Sports Medicine.

Name of participant: .....

Signature: .....

Name of investigator: .....

Signature: .....

Name of witness: .....

Signature: .....

Date: .....

Principal investigator:
Dr Laurie Rauch
UCT/MRC Research Unit for Exercise Science and Sports Medicine, PO Box 155, Newlands 7725.
Email: Laurie.Rauch@uct.ac.za

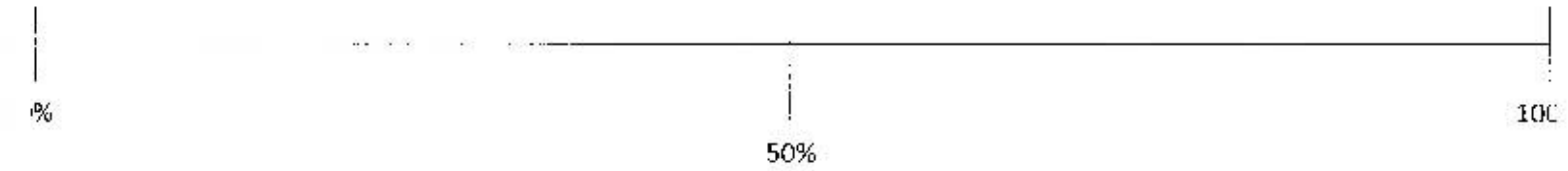
Co-investigators: Dr.G Prinsloo, Mr Stefano Scribani, Ms Sita Smit.

Heath Science Faculty
Research and Ethics Committee University of Cape Town
Room ES2-24 Groote Schuur Hospital, Old Main Building, Observatory, 7925
Tel. 021-406 6338 Facsimile: 021 406 6411

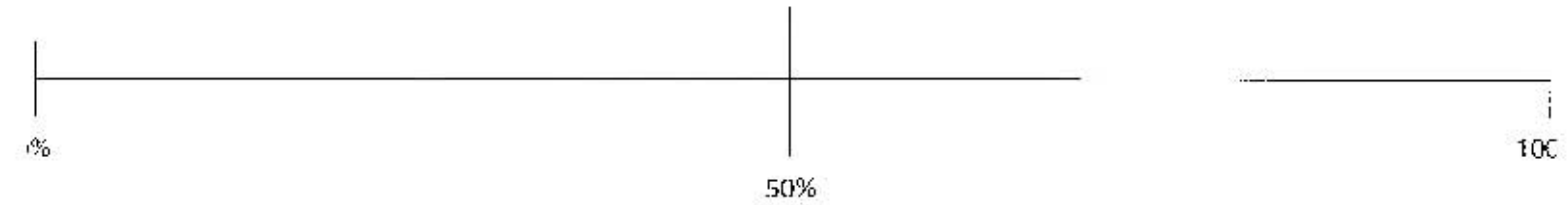
The Visual analogue scales: Name: .....

4 horizontal lines will be drawn one underneath the other. Each line is exactly 20cm long. According to each heading above the line, rate how effective you deemed the intervention to be. To rate how effective the intervention was, simply put a perpendicular line through the solid black line. The closer you put the mark to the right hand side, the more effective the intervention was. If you put your line in the middle, the intervention was only 50% effective.

1) How helpful was the intervention:

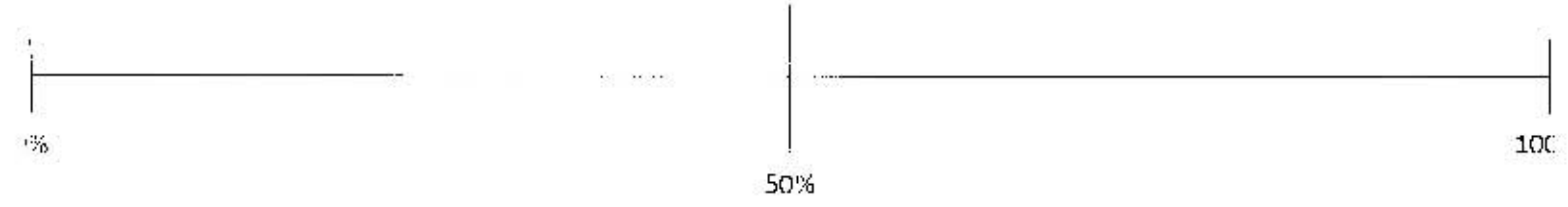


2) How sleepy do you feel now(immediately after the intervention)?



3) How sleepy do you feel now (immediately after post rest)?

4) How sleepy do you feel now (immediately after post Stroop)?



Patient Name _____ Date_____
Patient No. _____

SPIELBERGER STATE-TRAIT ANXIETY INVENTORY (STAI-T)

Self-Evaluation Questionnaire

Directions:
A number of statements which people have used to describe themselves are given below. Read each statement and then circle the appropriate answer to indicate how you generally feel. There are no right or wrong answers. Do not spend too much time on any one statement, but give the answer which seems to describe how you generally feel.

	Almost Never	Sometimes	Often	Almost Always
I feel pleasant	1	2	3	4
I feel nervous and restless	1	2	3	4
I feel satisfied with myself	1	2	3	4
I wish I could be as happy as others seem to be	1	2	3	4
I feel like a failure	1	2	3	4
I feel rested	1	2	3	4
I am "calm, cool, and collected"	1	2	3	4
I feel that difficulties are piling up so that I cannot overcome them	1	2	3	4
I worry too much over something that really doesn't matter	1	2	3	4
I am happy	1	2	3	4
I have disturbing thoughts	1	2	3	4
I lack self-confidence	1	2	3	4
I feel secure	1	2	3	4
I make decisions easily	1	2	3	4
I feel inadequate	1	2	3	4
I am content	1	2	3	4
Some unimportant thought runs through my mind and bothers me	1	2	3	4
I take disappointments so keenly that I can't put them out of my mind	1	2	3	4
I am a steady person	1	2	3	4
I get in a state of tension or turmoil as I think over my recent concerns and interests	1	2	3	4
	Almost Never	Sometimes	Often	Almost Always

Patient Name _____ Date _____
Patient Nr. _____

SPIELBERGER STATE-TRAIT ANXIETY INVENTORY (STAI – S / Part 1)

Instructions:
A number of statements which people have used to describe themselves are given below. Read each statement and then circle the appropriate number to the right of the statement to indicate how you feel right now, that is, at this moment. There are no right or wrong answers. Do not spend too much time on any statement, but give the answer which seems to describe your present feelings best.

	Not at all	Somewhat	Moderately so	Very much so
1. I feel calm.	1	2	3	4
2. I feel secure.	1	2	3	4
3. I am tense.	1	2	3	4
4. I feel strained.	1	2	3	4
5. I feel at ease.	1	2	3	4
6. I feel upset.	1	2	3	4
7 I am presently worrying over possible misfortune.	1	2	3	4
8. I feel satisfied.	1	2	3	4
9. I feel frightened.	1	2	3	4
10. I feel comfortable.	1	2	3	4
11. I feel self-confident.	1	2	3	4
12 I feel nervous	1	2	3	4
13. I am jittery.	1	2	3	4
14. I feel indecisive.	1	2	3	4
15. I am relaxed.	1	2	3	4
16. I feel content.	1	2	3	4
17. I am worried.	1	2	3	4
18. I feel confused.	1	2	3	4
19. I feel steady.	1	2	3	4
20. I feel pleasant.	1	2	3	4

Analysis of Spielberger questionnaires

Column number	**1**	**2**	**3**	**4**
Anxiety present item score	1	2	3	4
Anxiety absent item score	4	3	2	1

STAIS
Reverse scoring (anxiety absent questions): 1, 2, 5, 8, 10, 11, 15, 16, 19 and 20
Normal scoring (anxiety present questions): 3, 4, 6, 7, 9, 12, 13, 14, 17 and 18

STAIT
Reversed scoring: 1, 3, 6, 7, 10, 13, 14, 16, 19
Normal scoring: 2, 4, 5, 8, 9, 11, 12, 15, 17, 18, 20

Then add all of the scores together – total can be between 20 and 80.

SRSI3

HOW DO YOU FEEL RIGHT NOW? PLEASE CHECK ALL THE ITEMS USING THIS KEY.

RIGHT NOW, I FEEL THIS

①	②	③	④	⑤	⑥
Not at All	A Little	. . Moderately . .		A Lot	Maximum

①	②	③	④	⑤	⑥	1. My mind is SILENT and calm (I am not thinking about anything).
①	②	③	④	⑤	⑥	2. My muscles feel TIGHT and TENSE (clenched fist or jaws; furrowed brow).
①	②	③	④	⑤	⑥	3. I feel AT PEACE.
①	②	③	④	⑤	⑥	4. I feel DROWSY and SLEEPY.
①	②	③	④	⑤	⑥	5. Things seem AMAZING, AWESOME, and EXTRAORDINARY.
①	②	③	④	⑤	⑥	6. Right now I recognize the wisdom of sometimes ACCEPTING things as they are.
①	②	③	④	⑤	⑥	7. My muscles are SO RELAXED that they feel LIMP.
①	②	③	④	⑤	⑥	8. I am HAPPY.
①	②	③	④	⑤	⑥	9. I am WORRYING
①	②	③	④	⑤	⑥	10. I feel AT EASE.
①	②	③	④	⑤	⑥	11. I feel DISTANT and FAR AWAY from my cares and concerns.
①	②	③	④	⑤	⑥	12. I feel ENERGIZED, CONFIDENT, and STRENGTHENED.
①	②	③	④	⑤	⑥	13. I am DOZING OFF or NAPPING.
①	②	③	④	⑤	⑥	14. I feel THANKFUL.
①	②	③	④	⑤	⑥	15. I feel like I am living fully and SIMPLY in the PRESENT, not distracted by past or future concerns.
①	②	③	④	⑤	⑥	16. Things seem TIMELESS, BOUNDLESS, or INFINITE
①	②	③	④	⑤	⑥	17. I feel IRRITATED or ANGRY.
①	②	③	④	⑤	⑥	18. I feel JOYFUL.
①	②	③	④	⑤	⑥	19. I feel SAD, DEPRESSED, or BLUE.
①	②	③	④	⑤	⑥	20. I feel AWARE, FOCUSED, and CLEAR.
①	②	③	④	⑤	⑥	21. My hands, arms, or legs are SO RELAXED that they feel WARM and HEAVY.
①	②	③	④	⑤	⑥	22. I feel INNOCENT and CHILDLIKE.
①	②	③	④	⑤	⑥	23. My BREATHING is NERVOUS and UNEVEN (Or shallow and hurried).
①	②	③	④	⑤	⑥	24. I feel LOVING.
①	②	③	④	⑤	⑥	25. Things seem FRESH and NEW, as if I am seeing them for the first time.
①	②	③	④	⑤	⑥	26. I feel INDIFFERENT and DETACHED from my cares and concerns.
①	②	③	④	⑤	⑥	27. I feel PRAYERFUL or REVERENT.
①	②	③	④	⑤	⑥	28. I feel PHYSICAL DISCOMFORT or PAIN (backaches, headaches, fatigue)
①	②	③	④	⑤	⑥	29. My mind is QUIET and STILL.
①	②	③	④	⑤	⑥	30. I feel ANXIOUS.
①	②	③	④	⑤	⑥	31. I sense the DEEP MYSTERY of things beyond my understanding.
①	②	③	④	⑤	⑥	32. I feel RESTED and REFRESHED
①	②	③	④	⑤	⑥	33. I feel CARE-FREE.
①	②	③	④	⑤	⑥	34. TROUBLESOME THOUGHTS are going through my mind
①	②	③	④	⑤	⑥	35. My body is PHYSICALLY RELAXED.
①	②	③	④	⑤	⑥	36. Presently I feel there's no need to try to change things that simply can't be changed.
①	②	③	④	⑤	⑥	37. I feel fully focused and ABSORBED in what I am doing
①	②	③	④	⑤	⑥	38. I feel OPTIMISTIC, HOPEFUL, or TRUSTING that I can rely on someone or something.

Your age: _____ Gender: ☐ M ☐ F

SCORING KEY

NEW SCALES	TRADITIONAL SCALES	SCORING
SLEEPINESS		
SLEEPINESS	Sleepiness	4 + 13
BASIC RELAXATION		
DISENGAGEMENT	Disengagement	11 + 26
PHYSICAL RELAXATION	Physical Relaxation	7 + 21 + 35
MENTAL RELAXATION		
RESTED / REFRESHED	Rested / Refreshed	30
MENTAL RELAXATION	At ease / Peace	3 + 10 + 33
CORE MINDFULNESS		
MINDFUL ACCEPTANCE		6 + 36
MINDFUL QUIET	Mental Quiet	1 + 29
MINDFUL CENTERING		15 + 37
MINDFUL AWARENESS	Aware	20
MINDFUL AWAKENING		25
MINDFUL INNOCENCE	Childlike Innocence	22
ENERGIZED POSITIVE FEELINGS		
OPTIMISM / HOPE / TRUST		38
ENERGIZED	Energized	12
HAPPY	Joyful	8 + 18
THANKFUL AND LOVING	Thankful, Loving	14 + 24
TRANSCENDENCE		
AWE AND WONDER	Awe / Wonder	5
PRAYERFUL	Prayerful	27
DEEP MYSTERY	Deep Mystery	31
TIMELESS, BOUNDLESS, INFINITE	Timeless . . .	16

Abbreviations

ANOVA – analysis of variance

ANS – Autonomic nervous system

BFSM – Biofeedback assisted stress management

BIM – Breathe with interactive music

BIO- Biofeedback group

BP – Blood pressure

BR – breathing rate

CON- Control group

CV - Cardiovascular

CVD – Cardiovascular disease

CVS – Cardiovascular system

DM – Diaphragmatic breathing

ECG – Electrocardiogram

fMRI- Functional magnetic resonance imaging

HF- High frequency

HPA – hypothalamic- pituitary- adrenal

HR – Heart rate

HRV – Heart rate variability

IBI – Interbeat-interval

LF- Low frequency

MI – Myocardial infarction

NREM – Non rapid eye movement

PNS – Parasympathetic nervous system

RF – Respiratory frequency

RMSSD – Square root of the mean squared differences of successive normal-to-normal intervals

RSA – Respiratory sinus arrhythmia

SA- Sinus arterial

SD – Standard deviation

SDNN – the standard deviation of the normal-to-normal interval

SEM – Standard error measurement

SNS- Sympathetic nervous system

SRSI3 – Smiths relaxation inventory 3

STAI-S State aspect of the STAI-T

STAI-T - Spielberger State-Trait Anxiety Inventory

TF- Total Frequency

VAS – Visual analogue scale