

# A Novel Breathing Retraining and Osteopathic Manual Therapy Intervention, and its Effect on Cardiac Autonomic Activity and Breathing Symptoms

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## Declaration

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This Research Project entitled “The effect of osteopathic manual therapy with breathing re-training on the autonomic nervous system and dysfunctional breathing; an RCT” is submitted in partial fulfilment for the requirements for the Unitec degree of Master of Osteopathy.

## Candidate’s declaration

I confirm that:

- This Research Project represents my own work;
- Research for this work has been conducted in accordance with the Unitec Research Ethics Committee Policy and Procedures, and has fulfilled any requirements set for this project by the Unitec Research Ethics Committee.
- Research Ethics Committee Approval Number:

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Date:

## ABSTRACT:

Interventions entailing clinical management of dysfunctional breathing are thought to improve cardiac autonomic activity and breathing symptoms. Literature investigating such interventions in healthy individuals or in those with subclinical breathing disorders is sparse. The aims of this thesis were to develop a complex breathing management intervention and analyse its effects on cardiac autonomic measures and breathing symptom questionnaires in healthy adults. This thesis comprises three chapters. The first details an exploration of literature surrounding breathing retraining (BRT) and manual therapy (MT) directed at breathing in healthy individuals. In this review, an organisational structure for categorising relevant intervention studies is presented, setting a context for the clinical trial undertaken as part of this thesis. The second chapter describes an iterative process employed in the development and evaluation of a dual-protocol complex intervention administering BRT and MT. The third chapter is a report of randomised controlled trial, in which the complex intervention was carried out on a cohort of healthy active individuals, who perceived that breathing might be limiting their sporting performance. Outcomes presented in this report are cardiac autonomic measures and breathing questionnaire scores. Participants randomised to receive the complex intervention immediately demonstrated 3 – 4% improvements in cardiac autonomic variables, compared to reductions of 2% in the same variables for those in the control group who had not received treatment up to that point. This thesis resulted in the development of a clinically-applicable complex intervention for management of breathing, which was shown to improve cardiac autonomic measures in healthy active individuals. Further implementation of the BRT and MT protocols in clinical practice to alter cardiac autonomic measures is warranted. Further research is required to examine the efficacy and effectiveness of this intervention on various outcome measures and health states.

Keywords:

Abnormal Breathing Pattern Disorders, Breathing Dysfunction, Breathing Exercises, Physical Therapy Modalities, Osteopathic Manipulation, Heart rate variability, Heart rate recovery.

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## ABBREVIATIONS

ANS	Autonomic nervous system
BRT	Breathing retraining
CO <sub>2</sub>	Carbon dioxide
DB	Dysfunctional breathing
FEV	Forced expiratory volume
FVC	Forced vital capacity
HF	High frequency
HR	Heart rate
HRR	Heart rate recovery
HRV	Heart rate variability
Ln	Logarithm
ms	Milliseconds
MT	Manual Therapy
NQ	Nijmegen Questionnaire
O <sub>2</sub>	Oxygen
OMT	Osteopathic manual therapy
PSNS	Parasympathetic nervous system
RMSSD	Root mean square of the successive differences
SEBQ	Self-Evaluation of Breathing Questionnaire
SNS	Sympathetic nervous system
VO <sub>2</sub>	Volume of oxygen
W	Watts

## CHAPTER 1: INTRODUCTION

The supply of oxygen to the brain is the most immediate life-supporting need of the human body, and cessation of breathing is said to cause brain death after minutes. Comparatively, humans can survive complete water deprivation for days or food deprivation for weeks. The importance of breathing to sustain life is however at polar opposites to the current lack of literature surrounding breathing.

Within the spectrum of breathing health, from optimum to worst, breathing becomes dysfunctional when it is no longer adaptive, appropriate or responsive. Dysfunctional breathing (DB) results in intermittent or chronic symptoms mediated through biomechanical, biochemical and psychological mechanisms (Kulur, Haleagrahara, Adhikary, & Jeganathan, 2009). DB symptoms are diverse and include respiratory, cardiac, neurological and gastrointestinal presentations (de Groot, 2011). Furthermore, interventions in which breathing is consciously altered have been shown to positively affect the autonomic nervous system (ANS), type 2 diabetes (Kulur et al., 2009), kidney disease (Esposito et al., 2015), cardiovascular disease, gastrointestinal health (Silva & Motta, 2013), immune disorders (Cahalin, Arena, Lavie, & Forman, 2013), psychological disorders (Sánchez-Meca, Rosa-Alcázar, Marín-Martínez, & Gómez-Conesa, 2010) and musculoskeletal dysfunctions (McLaughlin, Goldsmith, & Coleman, 2011).

Reduced cardiac autonomic activity is associated with the same conditions improved through DB, i.e. cardiac failure, hypertension, type 2 diabetes and kidney disease. Autonomic activity affects and is affected by breathing. However, DB has not been shown to be strongly correlated with indices of low autonomic activity, though improvements in both DB and autonomic activity reduce the severity of associated conditions (Chaddha, 2015; Kishi, 2012). Therefore a treatment that affects both DB and autonomic activity could hold further health benefits, than a treatment that effects only one of these.

Treatment for DB is predominantly implemented through breathing retraining (BRT) and/or manual therapy (MT), yet uncertainty surrounds the nature of such interventions, their effects on

the autonomic nervous system, and how they can be incorporated into osteopathic practice. The aims of this thesis are to develop then investigate the effects of a dual-protocol complex breathing-specific intervention, comprising BRT and MT, on cardiac autonomic variables and breathing symptom questionnaire scores.

The thesis comprises three chapters. The first details an exploration of literature surrounding BRT and MT directed at breathing in healthy individuals. The second describes the development of a complex intervention involving BRT and MT. The third reports on a randomised control trial investigating the implementation of the aforementioned complex intervention on a cohort of healthy active individuals, who perceived that their breathing might have been limiting their sporting performance. Outcome measures included cardiac autonomic measures and breathing questionnaire scores.

## CHAPTER 2: LITERATURE REVIEW

### SECTION 1: STRUCTURE AND FUNCTION OF THE RESPIRATORY SYSTEM

#### 1.1 Respiratory Anatomy:

The anatomy of respiration can be subdivided into two systems, the structures that form a conduit for airflow, and structures responsible for creating air pressure changes. During inhalation, air enters the respiratory system through the nasal or oral cavities, travelling through the pharynx, larynx and trachea, halting at alveolar terminal air sacs (Hall, 2010). Within the lungs, oxygen and carbon dioxide are transferred at the alveoli to and from the circulatory system. A reverse flow then occurs during exhalation as air exits the lungs.

The mechanics involved in breathing involve a large number of musculoskeletal structures including multiple cervical, thoracic, costal and lumbar regions. The thoracic rib cage serves a protective function for the respiratory organs, whilst maintaining mobility required for respiratory movements. The classical description of inhalation involves the lower thorax/rib cage expanding anterolateral and superiorly, whereas the upper thorax/ribcage expands anteriorly and superiorly. During exhalation the thorax retracts either through passive elastic recoil or muscular contraction. Skeletal muscles involved in breathing can be categorised as primary, or secondary ('accessory') muscles of respiration (Standring, 2008). Primary muscles (Table 2.1 & Figure 2.1) are the most efficient, effective muscles of respiration, of which their principal role is respiration. Accessory muscles (Table 2.1) supplement the work of primary muscles during periods of increased metabolic demand (Caruana-Montaldo, Gleeson, & Zwillich, 2000), and have an alternate primary role of neck movement (Stone & Stone, 2011). During inhalation, reduction of lung/thoracic air pressure drives airflow from the higher ambient pressure into the lungs. Conversely, exhalation reverses this pressure gradient, and air flows out of the lungs and out of the individual (Hall, 2010; Standring, 2008).

Table 2.5: Primary and Secondary Muscles of Respiration.

<b>Muscle group:</b>	<b>Muscles utilised during inhalation:</b>	<b>Muscles utilised during exhalation:</b>
<b>Primary muscles</b>	Diaphragm External intercostals	Diaphragm (recoil) For forced exhalation: Internal intercostal Intercostalis intimi Subcostals muscles.
<b>Accessory muscles</b>	Sternocleidomastoid Scalenes Thoracic erector spinae Abdominals Pectoralis minor and major Inferior fibers of serratus anterior Latissimus dorsi Serratus posterior anterior Iliocostalis cervicis muscles	The abdominals (rectus and obliques) Iliocostalis Longissimus Serratus posterior inferior Quadratus lumborum

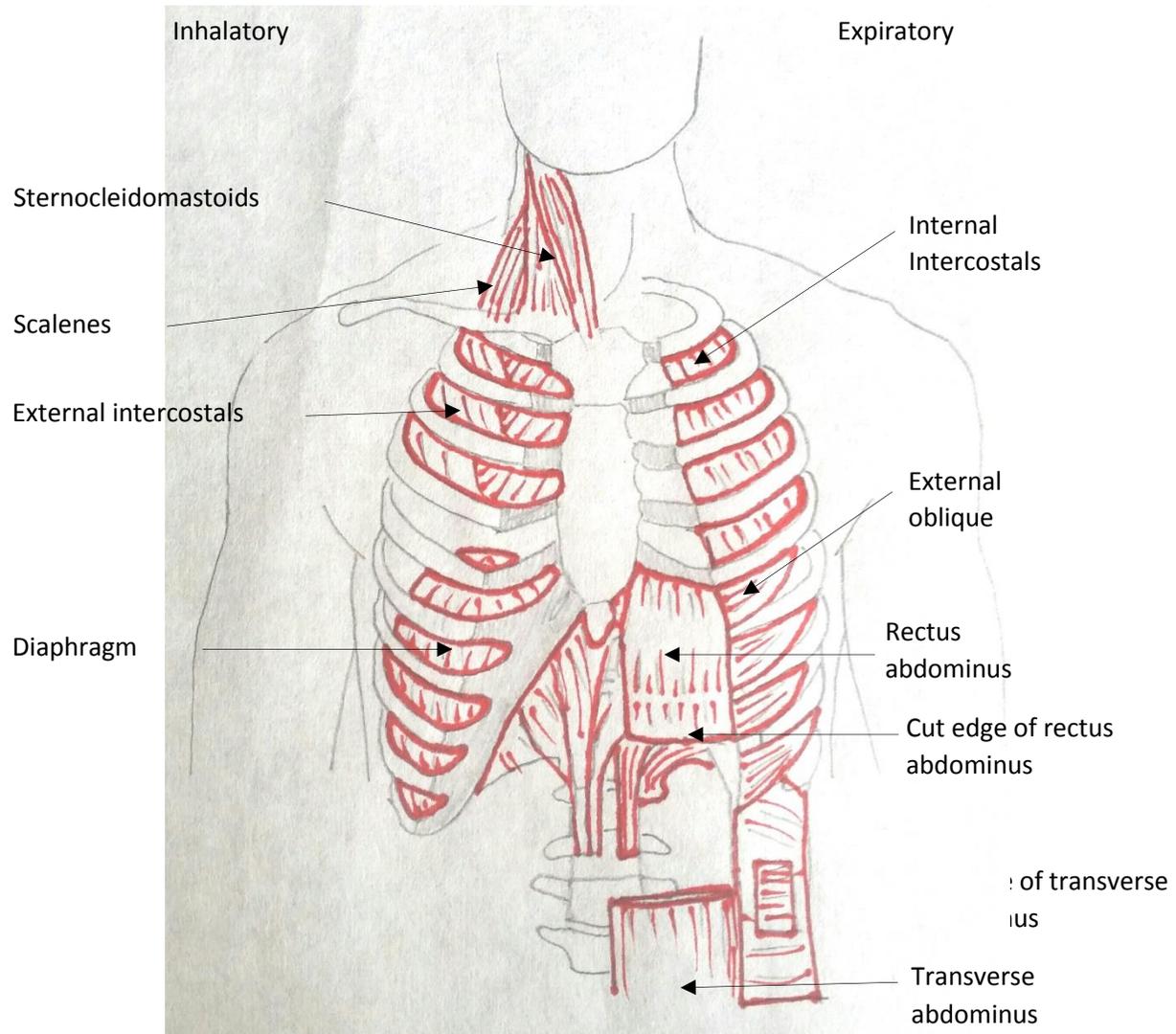


Figure 2.1: Muscles of Respiration. Muscles on the left are involved during inhalation. Exhalation on the right. Image adapted from humankinetics.com.

## SECTION 2: AUTONOMIC NERVOUS SYSTEM AND ITS NORMAL ROLE IN BREATHING

### 2.1 Definition of the Autonomic Nervous System

The autonomic nervous system (ANS) is the collection of nerves arising from the brain stem, which innervate smooth muscle and glands. The ANS is described in two division – the parasympathetic and sympathetic. Collectively, the parasympathetic and sympathetic nervous systems regulate blood pressure, breathing rate, arterial carbon dioxide levels, digestion, cardiac rhythm and pupil dilation (Hall, 2010). Body functions, such as arterial diameter regulation, require only the sympathetic nervous system (SNS), where increased SNS activity initiates vasoconstriction, and a sympathetic decrease, vasodilation. However, the majority of functions require a complex parasympathetic nervous system (PSNS) and SNS tonal balance (Hall, 2010). The combination of PSNS and SNS activity is remarkably effective at maintaining whole body functionality (Sleight, 1997).

Whilst both SNS and PSNS fluctuations occur in normal healthy states, chronic SNS dominance has been associated with pathophysiologic states such as type 2 diabetes, cardiovascular (heart failure, hypertension) and kidney disease (Bruno et al., 2012; Esler et al., 1995; Fisher, Young, & Fadel, 2009; Mueller, 2007; Thayer, Yamamoto, & Brosschot, 2010). In contrast, predominantly positive health outcomes are associated with increased PSNS activity, for example decreased risk of cardiovascular disease, type 2 diabetes, and psychological disorders (Berntson & Cacioppo, 2007; Gorman & Sloan, 2000; Quintana & Heathers, 2014) key body systems, respiratory and cardiac, effect and are affected by these complex interactions, which maintains their day-to-day activity. Several measures exist that aim to record the ANS.

### 2.2 State of ANS

Overall autonomic activity represents the balance between SNS PNS nerve impulses. Optimal autonomic state has been described as adaptive, responsive and appropriate to the given bodily task (Courtney, 2011). When a person is at rest parasympathetic dominance (increased parasympathetic activity) is typically observed (Thayer, Hansen, Saus-Rose, & Bjorn, 2009; Thayer & Lane, 2007). Increased sympathetic activity exists as a normal responsive mechanism during exercise, and is also associated with states of ill-health (Fisher et al., 2009). Regular physical exercise enables balanced autonomic activity, whereby short-term sympathetic

dominance stimulates central command sympathetic inhibition and precipitates longer-term parasympathetic responses (Nishime, Cole, Blackstone, Pashkow, & Lauer, 2000; Peçanha, Silva-Júnior, & Forjaz, 2013). Autonomic balance may become disturbed in pathological and non-pathological states (Thayer et al., 2009), for example, cardiac failure (Thayer et al., 2009), excess exercise (Carter & Ray, 2015) and long periods of sedentary activity (Hughson & Shoemaker, 2015). Ensuring an adaptive, responsive and appropriate ANS, is vital for whole body function as well as organs and organ systems. This is especially the case for the respiratory and cardiac systems, which are regulated through autonomic activity.

### 2.3 Respiratory Innervation

Innervation of the respiratory system consists of fibers from the autonomic and somatic nervous systems (Anon, 2011; Kirkman, 2014). Respiratory efferent innervation occurs for air conduits and musculoskeletal tissues involved in breathing. The brainstem permits automatic respiratory modulation, which regulates all aspects of subconscious breathing. Smooth muscle located in respiratory airways is innervated by the sympathetic branch of the ANS, creating bronchial constriction and dilation, whereas the somatic nervous system innervates striated muscle, contained within the primary and accessory muscles of respiration (Hall, 2010).

Predominantly the ANS and central respiratory muscle innervation fibers arise from cervical roots of C3, 4, 5 carried within the phrenic nerve, or in segmental peripheral fibers (Standing, 2008). Peripheral efferent fibers carry both conscious and subconscious instructions. Modulation takes place in the brainstem (pons and medulla), however, further modulation may occur from peripheral receptor feedback or higher central nervous system regulation (Hall, 2010; Anon, 2011). Cortical stimulation allows a conscious control of respiration, with signals transmitted within the pyramidal tracts and peripheral nerves.

### 2.4 Cardiac Innervation

The heart possesses self-regulatory beating mechanisms, although modulation predominantly occurs through cardiac autonomic innervation, and modulated at the hypothalamus and higher centres (Hall, 2010). Cardiac tissue is innervated by parasympathetic (via vagus nerve) and sympathetic (postganglionic) efferent fibers. Two innervated nodes (‘pacemakers’), release electrical impulses that stimulate coordinated cardiac muscle contraction, namely the sinoatrial and atrial-ventricular nodes. An electrical impulse originating from the sinoatrial node generates

the contraction of the right atrium, establishing cardiac muscle contraction (Ardell & Randall, 1986; Klabunde, 2013). Additionally, after a short pause, the sinoatrial node impulses contribute to atrial-ventricular node stimulation, which in turn initiates ventricular contraction. The sinoatrial node is primarily innervated by the right vagus nerve, with additional sympathetic efferent innervation (Lammert & Zeeb, 2014). The atrial-ventricular node is innervated by the left vagus nerve. The cardiac ventricles are indirectly innervated through the atrial-ventricular node impulses and directly through sympathetic innervation. Increased sympathetic activity raises heart rate and stroke volume, while decreased sympathetic and increased parasympathetic activity reduces both (Thayer et al., 2009). Furthermore, central nervous system modulation regulates cardiac function in response to emotion and physical stresses, such as temperature and exercise (Hall, 2010).

The cardiac and respiratory systems have a high level of complex autonomic innervation. Measurement of autonomic activity is often focused on the effects of such innervations, whereby cardiac and respiratory alterations are assumed indicative of autonomic activity.

## 2.5 Autonomic Measures

### *2.5.1 Introduction:*

More than 50 tools for measurement of the ANS have been described in the literature (Brierly-Bowers, Sexton, Brown, & Bates, 2011). These tools can be broadly placed into three categories: vascular, respiratory and cardiac (Brierly-Bowers et al., 2011). Vascular and respiratory measures such as temperature, blood, breath rate and air flow are revised elsewhere (Brierly-Bowers et al., 2011). Respiratory and cardiac autonomic measures are the focus of this thesis, and are the research standard for measuring cardiac autonomic activity (Al Haddad, Laursen, Chollet, Ahmaidi, & Buchheit, 2011), although confusion surrounds the validity of some cardiac autonomic measure indices (Billman, 2013).

Cardiac measures capitalise on autonomic innervation of cardiac tissue. A change in heart rate is representative of autonomic regulation (Hall, 2010). Observing natural beat to beat heart rate changes is the basis underlying the three main cardiac autonomic measures: heart rate (HR), heart rate variability (HRV) and heart rate recovery (HRR) (Buchheit, 2014). HR is measured as the number of beats per minute, with a higher number correlating to a higher sympathetic balance, and reversely so (Hall, 2010). HRV reports the beat to beat variance of heart rate, with a

greater variance indicative of a greater PSNS activity (Guidelines, 1996). HRR represents the strength of parasympathetic reactivation following exercise. A higher HRR represents a quicker PSNS reactivation (Peçanha et al., 2013).

### *2.5.2 Heart Rate*

Heart rate (HR) is documented as beats per minute (beats/min), increasing during activity and decreasing at rest. In resting conditions, HR is kept at a relatively constant low value (60–80 beats/min), due to a predominance of cardiac parasympathetic activity (Jensen-Urstad, Saltin, Ericson, Storck, & Jensen-Urstad, 1997; Peçanha et al., 2013). In contrast, during exercise sympathetic excitation and parasympathetic reduction increase HR, which elevates progressively with exercise intensity towards an individual's maximum HR (Macefield & Henderson, 2015). Beat-to-beat increases in heart rate are observed during inhalation, followed by a decrease during exhalation (Shields, 2009). This phenomenon of beat-to-beat variation in HR is termed the respiratory sinus arrhythmia (Grossman, Wilhelm, & Spoerle, 2004), the primarily mechanism driving HRV.

### *2.5.3 Heart Rate Variability*

Heart rate variability (HRV) represents cardiac beat-to-beat variances. Sympathetic dominance is observed during a constant uniform interval between heart beats, whereas an irregular heart beat interval (high variability) is indicative of adaptive parasympathetic activity (Anon, 1996). HRV is often used in medical clinics and research studies, and has been positively correlated to health issues such as cardiac failure (Thayer et al., 2010), type 2 diabetes (Almoznino-Sarafian et al., 2009), Alzheimer's disease, osteoporosis (Ershler & Keller, 2000), inflammatory arthritis, periodontal disease, decreased muscle strength and increased frailty (Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002; Thayer et al., 2010).

Several component HRV measures exist, originally believed to represent the PSNS and SNS's roles in cardiac autonomic activity. These components can be further split into those that are measured in the frequency domain (spectral analyses) and those recorded using the time domain (Anon, 1996). Whilst time domain measures are functions of the duration between heart beats, frequency domain measures are derived from the number of beats within pre-defined sampling durations (bands). Common frequency domain measures are low frequency, high frequency

(HF), very low frequency and low frequency/HF (Billman, 2013). Frequency domain (spectral analyses) is displayed through the three peak frequencies very low, low and high frequency, which are observed during HRV power recordings (Anon, 1996). Very low frequency are frequencies less than 0.04Hz. Low frequency between 0.04 and 0.15Hz. HF between 0.15–0.4Hz (Billman, 2013).

The most important time domain measure is the root mean square of the successive differences (RMSSD) in heart beats (R – R interval) (DeGiorgio et al., 2010; Halson, 2014).

#### 2.5.3.1 Very low Frequency

Oscillations in the very low frequency band have been attributed to thermoregulation, renin-angiotensin-aldosterone system and vagal innervation (Hughson et al., 1995; Shiraishi, Schou, Gybel, Christensen, & Norsk, 2002; Tripathi, 2011). These contributors often vary in potency, making very low frequency interpretation difficult (Kitney, 1974; J. A. Taylor, Carr, Myers, & Eckberg, 1998; Tripathi, 2011), and very low frequency does not solely represent PSNS activity (Tripathi, 2011).

#### 2.5.3.2 Low Frequency

Low frequency is also believed to represent a myriad of contributors. It is thought to reflect a mix of PSNS, SNS and yet undefined factors (Billman, 2013). Multiple representation has been identified through experiments involving surgical ablation of PSNS efferent fibers, resulting in a 50% decrease in low frequency (Akselrod et al., 1981; Houle & Billman, 1999; Randall, Brown, Raisch, Yingling, & Randall, 1991). When both PSNS and SNS are blocked, 25% of low frequency remains (Randall et al., 1991). This 25% is thought to consist of thermoregulation and renin-angiotensin-aldosterone system regulation. Importantly, these studies demonstrate that low frequency is not a reliable representation of any one influence (Billman, 2011) and therefore results based on low frequency should be interpreted as such.

#### 2.5.3.3 High Frequency

High frequency (HF) is likely constituted of a PSNS and a SNS component (S. Taylor, 2001). A sympathetic component has been reported in medical literature following a course of sympathetic receptors blockers, which reduced HF by 10%, at rest (S. Taylor, 2001). No other HF effectors are mentioned in the literature, this suggests that a PSNS involvement is potentially as large as

90%. The validation of HF's short recording period (60 seconds), allows for quick recordings, recommended in sporting research, where activity level are continuously changing and time is a constraint (Heathers, 2014). Therefore, although HF does not solely represent parasympathetic vagal activity, HF is a true representation of cardio vagal activity.

#### 2.5.3.4 Low Frequency/ High frequency

The ratio of low frequency to HF HRV was originally assumed to indicate the sympatho-vagal balance. This assumption was based on the idea that low frequency represents sympathetic activity and HF parasympathetic activity (Billman, 2011). While HF predominantly represents parasympathetic activity (Chess, Tam, & Calaresu, 1975; Piccirillo et al., 2009), low frequency does not solely represent sympathetic activity (Billman, 2013). Therefore, it was concluded by Billman (2013) that low frequency /HF cannot accurately quantify sympatho-vagal balance.

#### 2.5.3.5 Time Domain Measures:

The time domain represents the means and standard deviations of cardiac beat-to-beat intervals (R to R in the QRS heart beat complex). The normal-to-normal (NN) intervals represent all the R-R intervals within a given recording, after ectopic beats and artefacts are removed (Buchheit, 2014). Time domain measures are simple to compute, although the data provided is less descriptive, with less detailed data than frequency domain recordings (Billman, 2011). Time domain measures can be divided into statistical or geometrical indices (Table 2.2), the most popular being the RMSSD (Anon, 1996).

Table 6.2: Conventional Heart Rate Variability Measurements.

Variable	Units	Definition
<b>TIME DOMAIN MEASURES</b>		
<b>a. Statistical</b>		
SDNN	ms	SD of all normal R–R intervals
SDANN	ms	SD of the average normal R–R intervals calculated over short time periods (usually 5 min) for the entire recording period (usually 24 h)
RMSSD	ms	The square root of the mean squared differences between adjacent normal R–R intervals
SDNN index	ms	Mean of the SD of the normal R–R intervals calculated over short periods time (usually 5 min) for the entire recording period (usually 24 h)
NN50		The number of pairs of adjacent normal R–R intervals that differ by more than 50 ms
pNN50	%	NN50 divided by the total number of normal R–R intervals $\times$ 100
<b>b. Geometrical</b>		
HRV triangular index		Number of normal R–R intervals divided by the height of the histogram of all the normal R–R intervals measured on discrete scale with bins of 1/128 s (7.8125 ms)
TINN	ms	Baseline width of the minimum square difference of triangular interpolation of the highest peak of the histogram of all normal R–R intervals
<b>FREQUENCY DOMAIN MEASURES</b>		
Total	ms <sup>2</sup>	Area under the entire power spectral curve (usually $\leq 0.40$ ), variance of all normal R–R intervals
ULF	ms <sup>2</sup>	Ultra low frequency power ( $\leq 0.003$ Hz)
VLF	ms <sup>2</sup>	Very low frequency power (0.003–0.04 Hz)
LF	ms <sup>2</sup>	Low frequency power (0.04–0.15 Hz)
HF	ms <sup>2</sup>	High Frequency power (usually 0.15–0.40 Hz*)
LFnu	nu	Normalized low frequency power (LF/LF + HF)
HFnu	nu	Normalized high frequency power (HF/LF + HF)
LF/HF		Ratio of the low-to high frequency power

*Nu, normalized units; \*HF is shifted to higher ranges (0.24–1.04 Hz) in infants and exercising adults.*

List of time domain and frequency domain measures. Reproduced with permission from Billman (2011).

### 2.5.3.6 Root Mean Square of the Successive Differences

RMSSD has been reported as a valid representation of PSNS activity (Buchheit, 2014). The validity of RMSSD in studies is strengthened by RMSSD's low sensitivity to breath change (Penttilä et al., 2001), and short duration required for measurement (10 seconds – 1 minute) (Buchheit, 2014), with minimum 1 minute recordings often suggested (Esco & Flatt, 2014). Furthermore, frequency domain's low frequency /HF's coefficient of variation is higher ( $\approx 82\%$ ) than time domains RMSSD's ( $\approx 12\%$ ) (Al Haddad et al., 2011), which increases the likelihood that a change is not due to 'noise' (Buchheit, 2014). A further reduction in coefficient of variation has been reported after the natural logarithm of RMSSD (Ln RMSSD) is calculated (Heathers, 2014; Pinna et al., 2007). Therefore, Ln RMSSD is the recommended time domain index measure of PSNS (Halson, 2014).

### 2.5.4 Heart Rate Recovery

#### 2.5.4.1 Definition

HRR can be recorded at varying durations after exercise cessation. The most common are at 30 ( $HRR_{30}$ ), 60 ( $HRR_{60}$ ) and 120 ( $HRR_{120}$ ) seconds post exercise (Peçanha et al., 2013). HRR scores decrease in an inverse hyperbolic fashion (Perinil et al., 1993; Pierpont, Stolpman, & Gornick, 2000), with times post 60s plateauing horizontally (Buchheit, Papelier, Laursen, & Ahmaidi, 2007; Coote, 2010; Perinil et al., 1993). HRR has been shown to be reliable over the course of one year (Mellis, Ingle, & Carroll, 2014), with  $HRR_{60}$  reported as being more reliable than  $HRR_{120}$ , as parasympathetic innervation is more stable in this first 60 seconds following exercise, than post 60 seconds (Mellis et al., 2014).

HRR is often used to indicate cardiovascular fitness (Javorka, Zila, Balhárek, & Javorka, 2002; Peçanha et al., 2013) and is reported to strongly predict all-cause mortality (C. R. Cole, Blackstone, Pashkow, Snader, & Lauer, 1999; Nishime et al., 2000; Peçanha et al., 2013). HRR is faster in physically trained than sedentary individuals (Darr, Bassett, Morgan, & Thomas, 1988; Imai et al., 1994; Peçanha et al., 2013; Trevizani, Benchimol-Barbosa, & Nadal, 2012). Physical exercise programs are reported to improve HRR, in disease states (Kline et al., 2013; Legramante, Iellamo, Massaro, Sacco, & Galante, 2007; Myers et al., 2007), in cyclists (Lamberts, Swart, Noakes, & Lambert, 2009) and in healthy adolescents (Buchheit et al., 2008).

Factors known to effect HRR include exercise type undertaken, e.g. cycling or running (Maeder, Ammann, Rickli, & Brunner-La Rocca, 2009); body position during recording (Barak et al., 2010; Buchheit, Al Haddad, Laursen, & Ahmaidi, 2009); and intensity of exercise (Al Haddad et al., 2011; Imai et al., 1994). Because HRR is prolonged by an increase in exercise intensity, HRR recordings are suggested after submaximal rather than maximal or supra-maximal exercise (Al Haddad et al., 2011; Buchheit et al., 2007).

#### 2.5.4.2 Influences on Cardiac Autonomic Measurement

Many external and internal variables influence cardiac autonomic recordings. Such variables include age group, medication (cardiovascular, vasoactive, and psychotropic), circadian rhythm (Massin et al., 2000), wakefulness state (Walker, Muth, Odle-Dusseau, Moore de, & Pilcher, 2009), cardiovascular fitness (Carter & Ray, 2015; Hughson & Shoemaker, 2015), and breathing (Penttilä et al., 2001). These variables are generally controlled in research studies, however several acute variables are also problematic and often not controlled, such as the consumption of food and liquids (Heathers, 2014). These influencing factors confound interpretation of cardiac autonomic measures, and therefore conclusions can be vulnerable to interpretation error if experiments are not well controlled. Recently, it has been demonstrated that noise can be reduced by averaging 3 – 5 waking recordings (Plews et al., 2013), as day to day variations are smoothed out. As such, minimizing confounding variables and averaging waking recordings is advised to maintain data validity.

#### 2.5.4.3 Conclusion:

The cardiac autonomic measures HR, HRR and HRV are all considered to be valid measures of the ANS. Specifically, valid representations of PSNS include the HRV measures HF and RMSSD when averaged over multiple days, and HRR recordings at 30s and 60s. Despite their validity of measuring ANS activity, little to no relationship between HRR and HRV indices are reported in the literature (Dupuy et al., 2012; Esco, Olson, & Williford, 2010; Lee & Mendoza, 2012). The lack of relationship is believed to come from an inability to effectively control/allow for HRV's external and internal influences (Heathers, 2014).

Cardiac autonomic activity not only represents cardiac innervation, but can be utilised to represent other organs autonomic activity, including respiration. Additionally cardiac autonomic activity can be utilised to assess the effects of respiration on the ANS, for instance poor

breathing reduces HRV. Subsequently altered breathing may be observed through cardiac autonomic measures. Therefore, to ensure a healthy ANS it is suggested, amongst other methods that an individual maintain optimal breathing.

## SECTION 3: OPTIMAL BREATHING:

### 3.1 Introduction:

Breathing is the only vital function under both subconscious and conscious control (Ley, 1993; Ley & Timmons, 2013). Furthermore, the conscious manipulation of breathing affects the subconscious autonomic nervous system (ANS) (Hirsch & Bishop, 1981), giving rise to the implementation of conscious breathing exercises to influence autonomic activity. The ANS is also affected, negatively, by the day-to-day stresses of life, yet may be enhanced through adaptations of breathing. The functional, changeable aspects of breathing include: (1) controlling the orifice used for inhalation and exhalation (i.e. mouth or nose, or both); (2) the muscles which produce breath (primary and secondary muscles of respiration), the presence of pause (after inhalation, exhalation, or elsewhere in breathing cycle), the length of breath (breaths per minute and length of inhalation and exhalation phases) the depth of each breath, the consistency of breath to breath (for the above), and the body position held during respiration.

### 3.2 Nose and Mouth Breathing:

Normal function in humans is thought to be breathing through the nose rather than through the mouth, which is primarily used for eating and speaking (Petruson, 2007). Many clinical guidelines for breathing purport a similar emphasis on nose over mouth breathing for improving multiple health functions. Nasal breathing has long been recommended due to its abilities to raise air temperature and humidity (P. Cole, 1953; Ingelstedt & Ivstam, 1951; Proctor, 1977; Proctor & Lundqvist, 1973; Sivasankar & Fisher, 2002; Vass et al., 2003) and therefore water retention (Svensson, Olin, & Hellgren, 2006), as well as increase the filtration of contaminants and particulate matter, such as dust, pollen, and bacteria (P. Cole, 1953; Ingelstedt & Ivstam, 1951; Proctor, 1977; Proctor & Lundqvist, 1973; Sivasankar & Fisher, 2002). Furthermore, the transition of nasal diameter size, from 1.4 cm<sup>2</sup> at the nasal valve to 6 cm<sup>2</sup> in the nasal cavity interior, reduces intrathoracic negative pressure, permitting an easier inhalation (Petruson, 2007; Petruson & Theman, 1992).

Nasal breathing is associated with an increased lower lung lobe perfusion compared to mouth breathing, where alveoli are more potent (Swift, Campbell, & McKown, 1988); increasing total lung capacity by 5% (Swift et al., 1988) and arterial oxygen concentrations by 10% (Tanaka, Morikawa, & Honda, 1988).

The nose functions with valve like properties during exhalation acts to further prolong exhalation, ensuring adequate gas exchange takes place (Hairfeild, Warren, Hinton, & Seaton, 1987). Nasal breathing also increases nitric oxide production and distribution (Cardell, 2002; Jiang, Malavia, Suresh, & George, 2009; Kharitonov & Barnes, 1997), with production thought to occur in the predominantly in para-nasal sinuses (Andersson, Cervin, Lindberg, Uddman, & Cardell, 2002; Petruson, 2007). Nitric oxide aids the immune system by chemically killing invading microorganisms, decreasing SNS excitation, (Hughson & Shoemaker, 2015; S. A. Smith, Leal, Murphy, Downey, & Mizuno, 2015) creating vasodilation which improves alveoli oxygen uptake and improving the mucociliary system (Andersson et al., 2002). Mouth breathing does not result in increased nitric oxide production in the nasal passages, and therefore the health benefits associated with nitric oxide are not observed (Andersson et al., 2002).

‘Pursed lip’ mouth breathing, where inhalation is through the nose and exhalation through pursed lips creates a similar ‘valve like’ effect to nasal breathing. Several benefits have been reported for pursed lip breathing. Improvements include improved gas exchange in mild to severe chronic obstructive pulmonary disorder sufferers, with a quicker, more effective air transfer (Hellings & Trenité, 2013). Pursed lip breathing has also been used as a therapeutic intervention to reduce temporal mandibular joint and sinus problems (Kisner & Colby, 2012). However mouth breathing is suggested primarily as a temporary ‘backup’ for nasal breathing (Petruson, 2007) during dysfunctions such as nasal passage blockages. Chronic, constant mouth breathing is not advised, as malformation and dysfunctions of the mandible and maxilla have been reported in people who are long term mouth breathing (Franco et al., 2013; Harari, Redlich, Miri, Hamud, & Gross, 2010; Jefferson, 2009; Katherine, 1998; Lester & Hoit, 2014; Warren, 1990; Warren, Hairfield, & Dalston, 1990; Woodside, Linder-Aronson, Lundstrom, & McWilliam, 1991).

### 3.3 Abdomen and Upper Chest Breathing

‘Abdominal breathing’ is the term used to describe the movement of the abdomen during diaphragm contraction and relaxation. ‘Thoracic breathing’, or ‘upper thoracic breathing’ is the term used to describe the upper thorax rise and fall as the accessory muscles of respiration activate and relax. Abdominal breathing is considered desirable during quiet breathing, whereas secondary accessory muscles are important in situations where greater tidal volume, exhalation airflow rate or minute volume are required. The effects of abdominal and thoracic breathing can

be broadly categorised by either the pathway of respired air, musculoskeletal components used or pressure changes.

### *3.3.1 The Pathway of Airflow*

Abdominal and thoracic breathing, like nose and mouth breathing, direct air to different lung regions. Compared to thoracic breathing, abdominal breathing improves air distribution to the lungs (Gosselink, 2003) and is believed to enhance ventilation of the lower lung fields (Chuter, Weissman, Mathews, & Starker, 1990), reducing risk of atelectasis, infection and breathlessness (Breslin, 1996; Gosselink, 2003). Moreover, observations during abdominal breathing include a decrease in respiratory rate (Casciari, Fairshier, Morrison, & Wilson, 1981), minute ventilation (Casciari et al., 1981), tidal volume (Casciari et al., 1981) and following habitual abdominal breathing, improvements are also observed in forced vital capacity (FVC) (Celli, 1993; Orfanos, Ellis, & Johnston, 1999), and pre-hypertension symptoms (S.-Z. Wang et al., 2010).

### *3.3.2 Musculoskeletal Components of Respiration*

Diaphragmatic breathing, as with nose breathing, is suggested to reduce the energy cost of breathing (DiMarco, Connors, & Kowalski, 2004; Gosselink, 2003). Greater energy efficiency is likely to result from perfusion and structural adaptations, shaped by diaphragmatic breathing. Structural changes following habitual diaphragmatic breathing include a decrease in rib cage muscle involvement (Gosselink, 2003), an increase in inspiratory muscle strength (Buchheit, 2014; Mullen, 2002), thicker abdominal muscles (Ha, Kwon, Kim, & Choung, 2014), larger maximum lung expansion (Ha et al., 2014) and an increased lung volume (Martin, Ripley, Reynolds, & Best, 1976). In contrast, the regular recruitment of secondary muscles, seen during thoracic breathing, leads to secondary muscle overuse/straining and a weakening of the diaphragm (Ha et al., 2014).

### *3.3.3 Pressure Gradients Involved in Breathing*

During breathing, changes in abdominal and thoracic pressure facilitate gastrointestinal and circulatory function (Silva & Motta, 2013). The inferior vena-cava and esophagus pierce the dome shaped diaphragm muscle (Figure 2.2). During diaphragm contraction, the esophagus is constricted by the esophageal hiatus ensuring a food bolus does not reflux cephally (Eherer et al., 2012). Conversely, the inferior vena-cava passes through the central tendon and is not constricted during diaphragmatic contraction. The venous blood is propagated through the open

inferior vena-cava, by increased abdominal and decreased thoracic pressure, with the one-way valves of the venous system permitting only cephally directed venous flow (Figure 2.3). The increased abdominal pressure (Balzan et al., 2014), aids not only the inferior vena-cava flow, but blood flow within all veins in the abdomen and thorax; where an increased venous return is associated with an increased health of organs in this area (Byeon et al., 2012; S.-Z. Wang et al., 2010). Additionally, the pressurised abdomen, seen during inhalation, improves the stability of the lumbar spine (Hodges & Gandevia, 2000a, 2000b; Park, Kweon, & Hong, 2015).

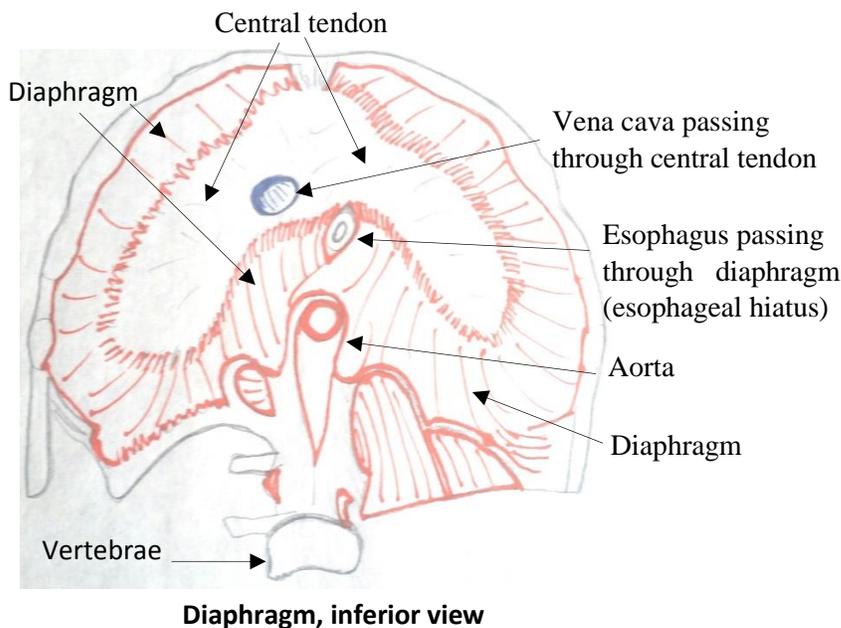


Figure 2.2: 1  
the central tendon  
diaphragm musculature.

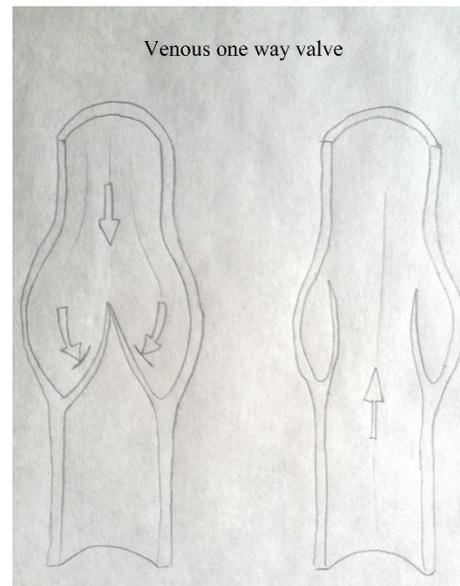


Figure 2.3: The Venous System's Omni-Dir but not caudad.

Furthermore, abdominal pressure also aid bolus distention, reducing constipation (Silva & Motta, 2013).

### 3.4 Depth of Breath

Breath frequency is inversely related to breath-depth, for the same metabolic demand and minute ventilation. Both depth and duration of breath alter the volume of air inhaled and exhaled. Rapid breathing frequency at rest is generally associated with negative outcomes, such as a decreased HRV, whereas positive outcomes are associated with predominantly slower breathing frequencies. Deep breathing is generally recommended over shallow breathing, (Kirkman, 2014),

with advantages of deep breathing overlapping those of nasal breathing. Deep breathing compared to shallow breathing has been reported to increase breathing efficiency (metabolic cost) at rest (Gosselink, 2003), lung volume (A. Jones, Tse, Cheung, To, & Lo, 1997), tidal volume (M. Thomas et al., 2003; Wald et al., 2005), oxygen saturation (Casciari et al., 1981; Gosselink, 2003; Manzano, Carvalho, Saraiva-Romanholo, & Vieira, 2008), ventilation (Menkes & Britt, 1980), efficiency of pulmonary mechanics (Celli, 1993; Chuter et al., 1990; Orfanos et al., 1999), surfactant secretion, improving lung compliance (Melendez, Alagesan, Reinsel, Weissman, & Burt, 1992), ventilation perfusion matching, and forced vital capacity (Celli, 1993; Chuter et al., 1990; Orfanos et al., 1999). Additionally, deep breathing decreases diffusion rate (Prabhu, Mink, Graham, & Cotton, 1990), the ratio of dead space to tidal volume, in patients with emphysema (Casciari et al., 1981), atelectasis, hypoxemia, pneumonia after abdominal surgery (Grams, Ono, Noronha, Schivinski, & Paulin, 2012) as well as constipation (Silva & Motta, 2013).

Conversely, benefits from slow breathing, greater than 3 breaths per minute (Song & Lehrer, 2003), include increased tidal volume (Gosselink, 2003), blood levels of carbon dioxide (Woolfolk, Sime, & Lehrer, 2007) alveolar ventilation (Gosselink, 2003), arterial oxygen saturation (in most patients with emphysema) (Gosselink, 2003), venous return (Byeon et al., 2012; S.-Z. Wang et al., 2010), ventilation of the basal lung lobes (Reid & Loveridge, 1983) and increased vagal activity (S.-Z. Wang et al., 2010). Furthermore, slow breathing decreases sympathetic activity (S.-Z. Wang et al., 2010), the ratio of dead space to tidal volume in emphysema patients (Casciari et al., 1981), pre-hypertension (S.-Z. Wang et al., 2010), atelectasis, hypoxemia and pneumonia after abdominal surgery (Grams et al., 2012), constipation (Silva & Motta, 2013) and a decrease in hyperventilation (Woolfolk et al., 2007). In conclusion, most evidence suggests slow deep breathing during rest is beneficial.

### 3.5 Breathing's Inhalation: Exhalation ratio

Gosselink (2003) suggests the inhalation phase of breathing should be shorter in duration than the exhalation phase. A longer exhalation should allow time for complete lung emptying, reducing dead space (Gilbert, Seals, Wyka, & Bradley, 1999; Wald et al., 2005), maintaining pH balance and the relaxation of inspiratory muscles (Chaitow, Bradley, & Gilbert, 2002; Gilbert et al., 1999; Guyton, 1967). A longer exhalation than inhalation phase has also been associated with

improved diaphragm function (Gosselink, 2003), an increase in maximum oxygen uptake (Gosselink, 2003) and increased parasympathetic activity (Pramanik, Pudasaini, & Prajapati, 2010).

### 3.6 Breath Pause

A breath pause after exhalation is suggested for optimum breathing (Hall, 2010). Additionally, the breathing retraining methods Buteyko and pranayama, suggest a second pause after inhalation (Gilbert, 1999). A post exhalation pause ensures a sufficient exhalation phase occurs. Woolfolk et al. (2007) have suggested a post exhalation pause should last for 5 – 10% of a breath's length (~1 s) compared to 3–5% for a post inhalation pause (~0.5 s). A pause after inhalation allows the even distribution of air from hyperventilated lung areas to areas of hypoventilation, homogenizing distribution throughout the lungs of ventilation to perfusion ratios for normal (Pillet, Choukroun, & Castaing, 1993) and obstructed lungs (Cormier, Laviolette, Atton, & Series, 1991).

### 3.7 Conclusion:

Optimal breathing should be adaptive, appropriate and responsive to the given task. During rest, optimal breathing has been described in the literature as nasal breathing, utilising the diaphragm, and accompanied by a longer exhalation phase followed by a pause. Each breath should then be repeated at a similar rhythm, duration and consistency as the last. The inability to perform optimal functional breathing is a characteristic aspect of dysfunctional breathing (DB), a concept discussed in the following section.

## SECTION 4: DYSFUNCTIONAL BREATHING.

### 4.1 Definition:

Dysfunctional breathing (DB) is a condition characterised by a range of symptoms of biochemical and biomechanical origin that are thought to be associated with inappropriate breathing (Chaitow et al., 2002). Although DB has been defined by multiple authors, to date no consensus based definitive definition exists (Barker & Everard, 2015). Multifactorial, diffuse, but cumulative pathological and pathophysiological changes make DB very difficult to diagnose with certainty, and a diagnosis is often arrived at by a process of exclusion (Howell, 1990; Warburton & Jack, 2006). Symptoms thought to result from DB are diverse and include respiratory, cardiac, neurological and gastrointestinal presentations (de Groot, 2011). Many of these symptoms may arise from respiratory alkalosis brought about by chronic or transient bouts of hyperventilation (hyperventilation syndrome) (Kern & Byrd, 2015). However, it is now commonly accepted that the manifestation of DB encompasses more than traditionally recognised hyperventilation syndrome (Courtney, 2011). Barker and Everard (2015) have recently discriminated between two forms of DB, thoracic and extra-thoracic (Barker & Everard, 2015) (Figure 2.4), further divided into the two subsets: functional and structural. Interventions are implemented with the aim of reducing these functional and structural based aspects of DB. Functional dysfunctions take the form of a non-optimal breath, as described in this section. Structural based dysfunctions include changes to musculoskeletal tissue that affect the mechanism of breathing, be it through inefficient movement/activation or altered circulatory, respiratory or neural conveyance.

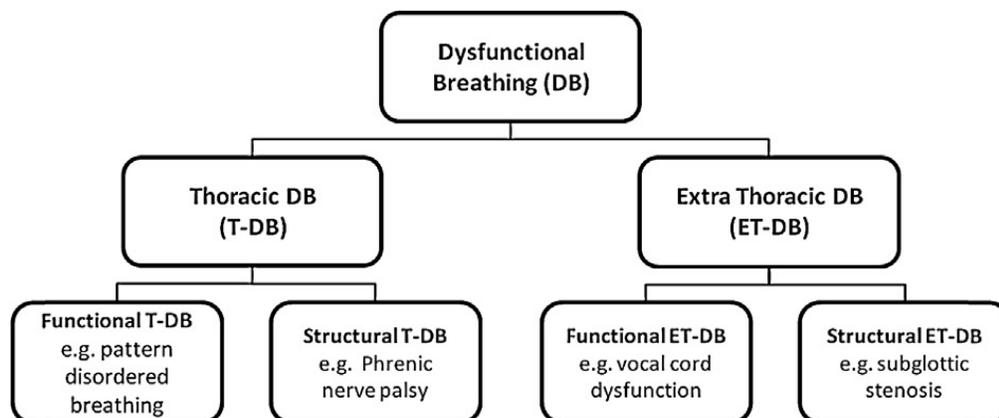


Figure 2.4: An Overview of Dysfunctional Breathing. Copied with permission from (Barker & Everard, 2015)

## 4.2 Clinical Presentation:

### 4.2.1 Signs and Symptoms:

Symptoms arising from DB may occur independently of other medical conditions or may exacerbate, or be exacerbated by, their similar symptoms (Grossman, De Swart, & Defares, 1983; M. Thomas, McKinley, Freeman, & Foy, 2001). Symptoms most commonly associated with DB include shortness of breath, wheeze/stridor, throat tightness, sighing, chest pain and difficulty on inhalation breath (Barker & Everard, 2015). Recently, Barker and Everard (2015) have categorised the common symptoms present in thoracic and extra thoracic DB. These are presented in Table 2.3.

### 4.2.2 The Aetiology of Dysfunctional Breathers:

Multiple non-pathological predisposing factors are thought to contribute to the aetiology of DB. Predisposing factors include smoking (Behera et al., 2013); exposure to airborne irritants (Magari et al., 2002; Prezant, Levin, Kelly, & Aldrich, 2008); high stress (Kunik et al., 2005; Masaoka & Homma, 2000, 2001); obesity, past and present (Aaron et al., 2008; Collins, Hoberty, Walker, Fletcher, & Peiris, 1995; Kamil, Teng, & Hassan, 2007); as neck and thorax

Table 2.7: Summary of symptoms associated with thoracic (T) and extra thoracic (ET) forms of dysfunctional breathing (DB).

Symptom	Key information	T-DB	ET-DB
Shortness of breath	At rest, with physical exertion or during performance	✓	✓
Wheeze/stridor	Predominantly inspiratory but can have an expiratory component	x	✓
Throat tightness	Commonly described as a choking sensation or throat closure	x	✓
Sighing	Generally frequent, deep or as yawning	✓	✓
Chest pain	At rest or with activity, can vary in location	✓	x
Difficulty breathing in	A sensation of not being able to get enough air in	✓	✓

Copied with permission from Barker and Everard (2015).

posture (Chaitow et al., 2002; Dean, 1985; Newsham, Klaben, Miller, & Saunders, 2002). A number of pathologies have also been associated with the aetiology of DB including the presence or history of respiratory, cardiac and autonomic diseases (Britton & Martinez, 1996; Engel & Vemulpad, 2007; Samet, Tager, & Speizer, 1983).

## 4.3 Diagnosis of Dysfunctional Breathing

Several methods exist to identify and monitor the signs of DB, namely symptom questionnaires, assessment of breathing related movement, and measurement of ventilatory parameters.

Individually, these methods are not considered to be reliable or valid at establishing the presence

of DB (Courtney, 2011), therefore, it has been recommended that a multicomponent assessment approach be taken when diagnosing DB, to reduce the risk of false positive DB diagnosis (Van Dixhoorn & Folgering, 2015).

#### *4.3.1 Questionnaires*

There are two main breathing symptom questionnaires in current clinical use, the Nijmegen Questionnaire (NQ) (Van Dixhoorn & Folgering, 2015), and the Self Evaluation of Breathing Questionnaire (SEBQ) (Courtney & Greenwood, 2009). The NQ (Appendix 1) contains 15 items aimed at assessing breathing symptoms. Initially, the NQ was developed in relation to HVS, however, it is now commonly used by practitioners and researchers as a diagnostic and prognostic measure of breathing symptoms (Han, Stegen, De Valck, Clément, & Van De Woestijne, 1996; M. Thomas et al., 2003). The NQ has been validated against the hyperventilation test (Van Dixhoorn & Duivenvoorden, 1985), with a sensitivity of 91% and a specificity of 95%. However, an elevated NQ score is not indicative of a specific syndrome other than hyperventilation syndrome, although elevated NQ scores have been shown to decrease following breathing retraining (Van Dixhoorn & Folgering, 2015). The author of the Nijmegen Questionnaire, Van Dixhoorn, recently stated that “the Nijmegen Questionnaire is useful to quantify and assess the normality of subjective sensations” and that NQ may be used concurrently with other breathing assessment methods to form a diagnosis of DB (Van Dixhoorn & Folgering, 2015). The SEBQ contains 25 DB symptom-associated items (Appendix 2). In a study reporting the initial development of the SEBQ questionnaire, two symptom based themes were identified as differing from the NQ, a “lack of air” and “perception of inappropriate or restricted breathing” (Courtney & Greenwood, 2009). The SEBQ has recently been shown to have high test re-test reliability, and that SEBQ scores were associated with current smoking and chronic and recent respiratory disease (Mitchell, Bacon, & Moran, 2015). Currently, neither the SEBQ nor the NQ have been sufficiently validated for diagnosing DB, and diagnosis remains dependent on the presence of other factors, such as the presence of chest and abdominal wall movement patterns.

### *4.3.2 Breathing Movement Parameters*

#### 4.3.2.1 Functional Methods

Common osteopathic clinical examination involves a combination of palpatory based and visual observational assessment procedures. There are two major palpatory manual assessment methods described for breathing assessment: the ‘HiLo’ and manual assessment of respiratory motion (Chaitow, Gilbert, & Morrison, 2014). The HiLo and manual assessment of respiratory motion were developed in an attempt to assess the functional and structural aspects of breathing in the clinical setting. The HiLo is a hands-on, active assessment of ‘upper thoracic’ versus ‘abdominal breathing’ patterns (Courtney, Cohen, & Reece, 2009). The manual assessment of respiratory motion technique aims to assess breathing rate, regularity and distribution of breathing motion through palpation of the lower lateral rib cage (Courtney, Van Dixhoorn, & Cohen, 2008). Observation-based breathing assessment monitors mouth and nose utilisation, abdomen or thorax movement, inhalation and exhalation duration, frequency of breathes per minute and the presence and placement of breath pauses, sighs, coughs or wheezes (Chaitow et al., 2002). However, these observations are subjective and depend on practitioner judgement, and are therefore vulnerable to poor inter-rater reliability (Van der Vleuten, Norman, & De Graaff, 1991). Furthermore, functional breathing assessments based on practitioner assessment of breathing movement in clinical populations, lack validation as effective methods for DB diagnosis.

#### 4.3.2.2 Manual Techniques to Assess Breathing

Assessment of respiratory structure involves the use of visual observation, patients’ movement (active) and practitioner facilitated movements (passive) and palpation examinations. Somatic dysfunction, defined as “Impaired or altered function of related components of the somatic (body framework) system: skeletal, arthrodial and myofascial structures, and their related vascular, lymphatic, and neural elements” (American Osteopathic Association, 2010). Somatic dysfunction is identified clinically by osteopathic practitioners using palpation, based on the presence of altered tissue texture, asymmetry, decreased range of motion, and tenderness (DiGiovanna, Schiowitz, & Dowling, 2005). Although the methodologies vary between manual therapies and practitioners, the core approach of the of altered tissue texture, asymmetry, decreased range of motion, and tenderness model are known by most osteopaths (DiGiovanna et al., 2005; Snider, Johnson, Snider, & Degenhardt, 2008).

#### *4.3.3 Ventilatory Parameters*

Ventilatory parameters provide objective measures of respiratory function. Parameters include forced expiratory volume (FEV), peak expiratory flow, FVC, peak oxygen uptake ( $\text{VO}_2$ ), tidal volume and dead space. These parameters are considered to be reliable and valid measures of respiratory function, however, correlations between ventilatory parameters and DB should not be based on isolated ventilatory parameters alone (Van Dixhoorn & Folgering, 2015). Furthermore, the equipment required to measure ventilatory parameters are often cumbersome, expensive and therefore have low utility for routine clinical use.



## 5.1 Conscious Control of Ventilation

A vast amount of literature exists surrounding conscious control of ventilation (see Figure 2.5). The literature may be divided by the type of conscious control (breathing manipulation or BRT), the associated medical condition/s present, and the outcome variables measured.

### *5.1.1 Types of Conscious Control of Ventilation*

There are two types of studies evident within conscious control of ventilation, namely acute manipulation of breathing and longer-duration breathing retraining (BRT). The purpose of both breathing manipulation and BRT is to alter the functional modifiable aspects of breathing. Functional aspects include the engagement of muscles, and orifice utilised during breathing (nose and/or mouth), as well as breath length, duration, depth, pause, rhythm and inhalation: exhalation duration ratio. Altering the functional aspects of breath can influence multiple health systems (Section 3), including cardiac autonomic activity (Kulur et al., 2009; Tharion, Samuel, Rajalakshmi, Gnanasenthil, & Subramanian, 2012).

#### 5.1.1.1 Breathing Manipulation

Breathing manipulation is predominantly implemented to standardise breathing during a given task or recording. For example, a breath frequency of 6 breaths per minute has been prescribed during HRV recordings, which regulates the effect of breath on HRV indices (Quintana & Heathers, 2014; Song & Lehrer, 2003). Alternatively non-standardised breathing manipulation is required when short-term alterations of breathing frequency and depth are required for the manipulation of bodily functions, or to examine the underlying physiological response of changes in breath elements. For example, respiratory biofeedback provided from observations or recordings of aspects of breath frequency and depth are shown to alter pain levels (Kapitza, Passie, Bernateck, & Karst, 2010), and venous flow (Kimura et al., 2011). Short term breathing manipulation is reported to have effects of up 24 hours (Prinsloo, Derman, Lambert, & Laurie Rauch, 2013). However, in the majority of the literature, breathing manipulation is implemented for same day goals, without consideration of longer term follow up.

#### 5.1.1.2 Breathing Retraining

The general aim of BRT is to alter the functional aspects of breathing over a period of time, so that altered aspects carry over into spontaneous breathing. In commonly applied BRT consists of individuals becoming aware and or altering their breath to become a nasal, abdominal breath

(Chaitow et al., 2014). BRT methods have been utilized in various forms by manual therapists for many years, with accounts originating in the 1900s, with pioneers A.T Still and D.D Palmer practicing and teaching BRT (Soley & Shock, 1938). Today, manual therapists commonly practice their own individually or clinically designed BRT methods, for example Breathing Works™ (formerly Better Breathing Clinic), Auckland, New Zealand (Nicholls, Walton, & Price, 2009). Several structured breathing retraining methods exist, for example, the Butekyo methods (S. Thomas, 2004) and pranyamic yoga breathing (Prem, Sahoo, & Adhikari, 2012). The Butekyo method and pranyamic yoga methods are applied within a structured framework, with set guidelines and prompts. Structured BRT methods are reproducible, allowing for methodical implementation and teaching, but may compromise individual tailored application to the individual. Alternatively, individualised methods provide freedom for practitioners to tailor BRT based on the individual patient's presentation, and may therefore be more acceptable in typical clinical settings where patient-centred factors are valued. Core similarities of BRT methods do allow the outcomes of BRT studies to be compared and contrasted. The general consensus surrounding BRT implementation is that BRT is an effective method to improve parasympathetic activity (Woolfolk et al., 2007), improve respiratory function measures (González-Álvarez, Valenza, Cabrera-Martos, Torres-Sánchez, & Valenza-Demet, 2015) and improve breathing symptoms (M. Jones et al., 2015).

### *5.1.2 Medical conditions*

Multiple studies have investigated conscious control of ventilation, in over 23 conditions (Figure 2.5). The majority of research has been conducted in relation to the respiratory disease asthma. Predominantly, studies report positive health results following BRT interventions, however, no definitive conclusions could be drawn from a literature review due to methodological differences (Freitas et al., 2014; Ram, Holloway, & Jones, 2003). Furthermore, conscious control of ventilation is believed to improve breathing symptoms and increase a better sense of asthma control, but does not directly influence the underlying disease (Ram et al., 2003). The effect of BRT on breathing symptoms and cardiac autonomic measures, for non-pathological conditions, such as DB, have not been extensively investigated.

### 5.1.3 Outcome variables

Another complication in synthesis of DB research is the existence of many outcome variables. Outcomes are predominantly associated with the health condition under investigation. For instance, in studies investigating conscious control of ventilation in people with diabetes, outcome measures include levels of glycated haemoglobin and blood glucose (Kulur et al., 2009). Outcome measures specific to cardio-respiratory health include respiratory physiological measures, breathing symptom questionnaire scores and participants' cardiac autonomic measures. Relatively few studies have investigated the longer lasting effects of conscious control of ventilation on cardio-respiratory and breathing symptom measures, in high quality randomised controlled trials (Table 2.4).

### 5.1.4 Effect of Conscious Control of Breathing on Cardio-Respiratory Autonomic Indices and Breathing Symptoms in Healthy Individuals

#### 5.14.1 Breathing Manipulation

Whilst, little to no literature measuring respiratory measures or breathing symptoms during and following breathing manipulation exists in healthy individuals, breathing manipulation has been implemented whilst measuring the cardiac autonomic variable HRV. Subsequently, a very large body of literature exists in relation to HRV, and detailed review is beyond the scope of this review. A brief overview is given below.

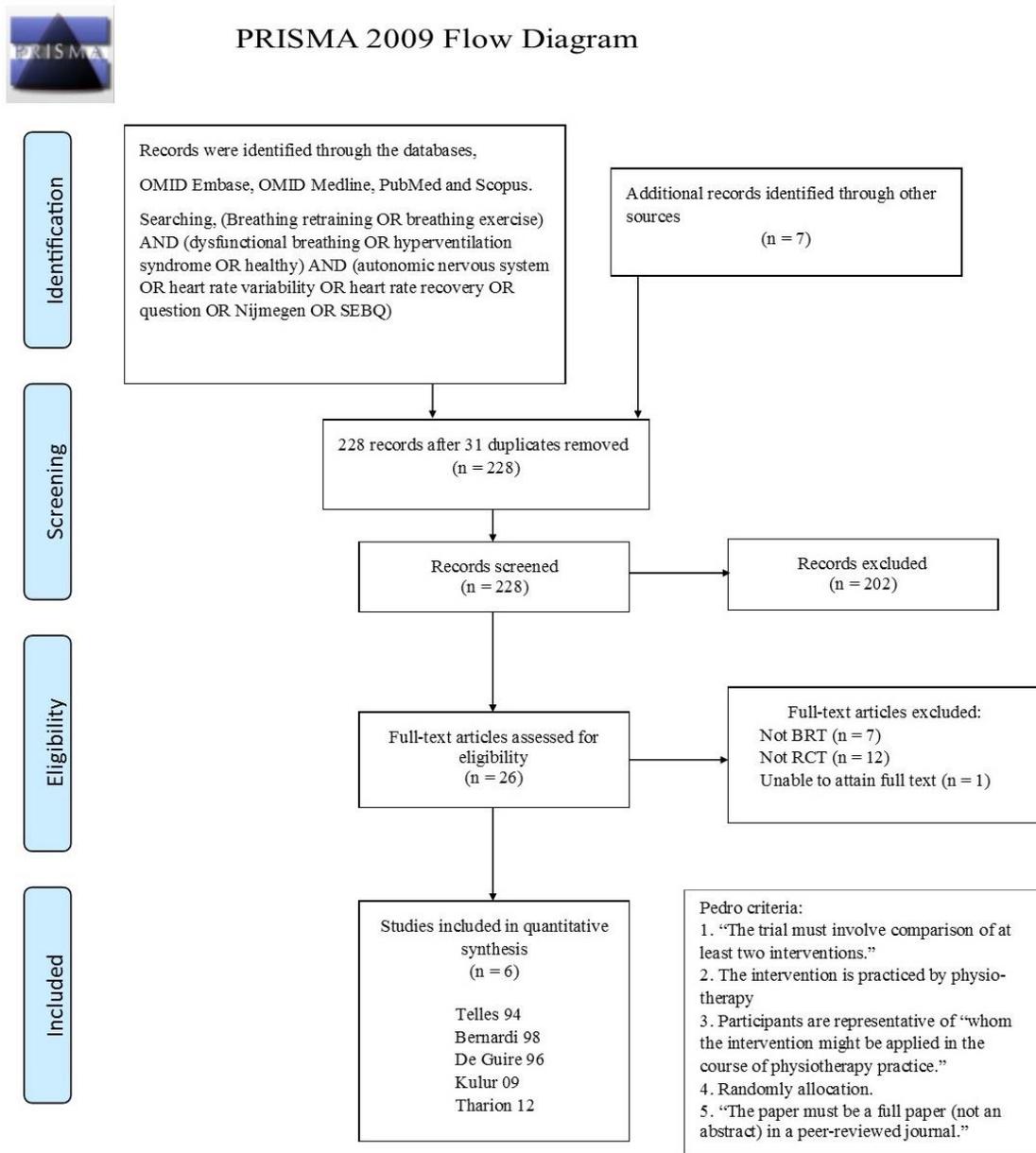
#### *Breathing Manipulation and Heart Rate Variability in Healthy Individuals*

Functional changes to breathing alter HRV recording, with improvements (increases) in HRV observed during breathing manipulation. Increased HRV is observed in healthy patients during diaphragmatic breathing (Kulur et al., 2009; S.-Z. Wang et al., 2010), a slower breathing frequency (Courtney, Van Dixhoorn, Greenwood, & Anthonissen, 2011; Lin, Tai, & Fan, 2014; Scolnick, Mostofsky, & Keane, 2014; Song & Lehrer, 2003), an equal inhalation: exhalation ratio (Lin et al., 2014), and during a deep breathing (Wheeler & Watkins, 1973). Conversely, decreases in HRV occur during shallow rapid chest breathing (Telles, Singh, & Balkrishna, 2011). Nonetheless, although alterations to breathing function have been investigated, optimum breath frequency and ratio to alter HRV are yet undetermined. For example, Lin et al. (2014) report that a breath frequency of 5.5 breaths per minute combined with an equal inhalation: exhalation ratio of 5:5 has a greater positive parasympathetic impact than breathing at 6 breaths per minute at a ratio of 4:6. However did not investigate slower breath rates of 5 and 4 breaths

per minute, reported by Song and Lehrer (2003) to further improve HRV. The benefits of an even inhalation: exhalation duration ratio (5:5) reported by Lin et al. (2014), is contested by Strauss-Blasche et al. (2000), who reported that a longer exhalation phase during a breath frequency of 10 breaths per minute increased HRV activity compared to a shorter exhalation phase. It appears different breathing rates, such as 5.5 breaths per minute (Lin et al., 2014) or 10 breaths per minute (Strauss-Blasche et al., 2000) provide differing optimal breathing ratios, inferring that an optimal exhalation duration might exist at rest. It is possible that once the minimal exhalation length for diaphragm relaxation and dead air expulsion is met, the breathing ratio becomes less important.

#### 5.1.4.2 Breathing retraining effects on Cardiorespiratory and Breathing Symptoms in Healthy Individuals

A literature review was performed to determine the effects of BRT on people with dysfunctional breathing and healthy individuals. BRT was defined as an intervention that retrains an aspect of breathing for longer term goals; greater than 1 week. Multiple databases were searched for the key terms (Figure 2.6). The PEDro criteria for assessment of methodological quality were implemented to include relevant papers (Anon, 1999). Six studies met the selection criteria, the study characteristics are summarized in Table 2.4. The 6 studies comprise two studies that investigate the effects of BRT on cardiac autonomic measures (HRV), (Kulur et al., 2009; Telles et al., 2011; Tharion et al., 2012) none on HRR. One study investigated the effects of BRT on the breathing symptom questionnaire NQ (M. Jones et al., 2015). Three randomised controlled trials have reported respiratory physiological measures following an intervention of BRT (Bernardi et al., 1998; DeGuire, Gevirtz, Hawkinson, & Dixon, 1996; M. Jones et al., 2015).



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For more information, visit [www.prisma-statement.org](http://www.prisma-statement.org)

Figure 2.6: PRISMA Flow Diagram.

Author	Objective	Target population	Outcome Measures	Intervention	Performed by	Results
Telles 94	That breathing exclusively through one nostril may alter the autonomic functions.	Healthy (48)	Galvanic skin resistance, oxygen consumption and heart rate.	Customary yoga plus, either left, right or both nasal breathing exercises. Practiced 27 respiratory cycles through the nostril, repeated 4 times a day for one month.	Yoga instructors	Breathing exclusively through the right nostril significantly increased the baseline oxygen consumption by 37%, breathing through the left nostril produced a significant increase in the baseline level of volar galvanic skin resistance (GSR).
De Guire 1996	To evaluate the long-term effects of paced diaphragmatic breathing on subjects who demonstrated associated signs of hyperventilation syndrome.	Hyperventilation syndrome (66)	Respiratory rate, end-tidal CO <sub>2</sub> , number of days that symptoms occurred, and frequency of cardiac symptoms.	Participants underwent either, guided breathing retraining and physiologic monitoring of diaphragmatic breathing and end-tidal CO <sub>2</sub> , or guided breathing retraining and physiologic monitoring of diaphragmatic breathing, or guided breathing retraining, or no treatment (control).	Undisclosed	Following intervention higher end-tidal carbon dioxide levels and lower respiratory rates when compared to pretreatment levels measured three years earlier. Subjects also continued to report a decrease in the frequency of functional cardiac symptoms when compared to pretreatment levels.
Bemardi 98	To establish the optimum breathing rate on SaO <sub>2</sub> in patients with CHF and healthy controls.	Chronic heart failure (50) and healthy volunteers (11).	SaO <sub>2</sub> , SaO <sub>2</sub> variability, minute ventilation, respiratory instability, respiratory instability, dead space ventilation.	The aims of training was to reduce breathing rate and learn how to mobilise in sequence, ie. within the same breath- the diaphragm, the lower chest, and then the upper chest during inspiration, and the same sequence during expiration.	Physiotherapist	There were no substantial changes in mean respiratory rate interval, systolic and diastolic blood pressures in CHF patients and controls during the study. In both groups, controlled breathing significantly increased mean SaO <sub>2</sub> at all breathing rates, even at three breaths per min.
Kulur 09	To evaluate the effect of diaphragmatic breathing on heart rate variability (HRV) in ischemic heart disease patients with diabetes.	Ischemic heart disease (45), Diabetics with ischemic heart disease (52), Ischemic heart disease and diabetic autonomic neuropathy (48) and healthy (60)	HbA <sub>1c</sub> , blood glucose and the average interval between normal heart beats (AVNN).	Abdominal breathing while lying in the supine position. Slow deep nasal inhalation into the bottom of the lungs. After taking a full breath, the patients were asked to hold it for a moment and then to exhale slowly.	Physiotherapist	A significant decrease in heart rate variability in ischemic heart disease patients and IHD patients who had diabetes. At 3 months and 12 months. The control group also improved at 12 months.
Tharion 12	To investigate the effects of non-yogic breathing exercise on respiratory rate and heart rate variability.	Healthy (36)	Baseline respiratory rate and short-term heart rate variability indices RMSSD and HF, amongst others.	Deep slow breathing exercise at 6 breaths per min, daily, for half an hour, for the duration of one month of the study. Inhalation: exhalation ratio = 4:6. Half hour breathing exercise either as one sitting or as two equally divided sittings, at any convenient time of the day, either in the sitting or in the supine position adopting any convenient posture.	Undisclosed	A significant difference in the spontaneous respiratory rate, mean arterial pressure, high frequency power and sum of low and high frequency powers was reported when compared against the control.
Jones 2015	To Investigate whether manual therapy produces additional benefit when compared with breathing retraining, in DB sufferers.	Dysfunctional breathers (60)	Quantitative evaluation of respiration, via the Nijmegen Questionnaire, forced expiratory volume in one second (FEV <sub>1</sub> ), forced vital capacity (FVC), breath hold time, Hospital Anxiety and Depression Scale (HADS) and musculoskeletal measurements.	Participants were allocated into the Breathing retraining group (standard treatment; n = 30) or breathing retraining plus manual therapy (intervention; n = 30) group. Both the groups received standardized respiratory physiotherapy, which included: DB education, breathing retraining, home regimen, and audio disc. Manual therapy consisted of an individualized selection of Maitland mobilisations, muscle energy techniques, trigger point therapy, myofascial and positional release techniques, diaphragm doming and rib raising.	Physiotherapist	No significant difference was found between the groups for primary outcome Nijmegen score, or any secondary outcomes (Hospital Anxiety & Depression Score, spirometry or exercise tolerance). However significant reductions in Nijmegen scores were reported for both groups.

Table 2.8: Review of Breathing Retraining, Randomised Controlled Trials on Healthy Individuals.

*The Effects of Breathing Retraining on Cardiac Autonomic Measures in Healthy Individuals*

Improved autonomic activity has been observed following BRT in healthy individuals. Often BRT incorporates slower breath rate (S.-Z. Wang et al., 2010), longer exhalation, diaphragmatic breathing, and deep breathing (S.-Z. Wang et al., 2010). Improved PSNS activity following BRT has been reported during conscious and non-conscious control of breathing (Hirsch & Bishop, 1981).

To date, there appears to be no peer reviewed literature investigating the effects of BRT on the cardiac autonomic measure HRR. Deep slow nasal/abdomen breathing initiate's higher PSNS activity (S.-Z. Wang et al., 2010), hence it may be assumed that this style of breathing would be associated with faster HRR. However, it is possible that such a breathing style, would not provide sufficient oxygen during recovery, leading to an unwanted metabolically driven increase in SNS activity (Buchheit, Al Haddad, Mendez-Villanueva, Quod, & Bourdon, 2011) and therefore a decreased HRR.

Three randomly controlled trials have investigated the effects of BRT on cardiac autonomic measures in healthy participants, all finding improved autonomic activity following BRT of at least one month (Kulur et al., 2009; Telles, Nagarathna, & Nagendra, 1994; Tharion et al., 2012). One of these studies, Telles et al. (2011) manipulated breathing through left and right nasal passages, and the other two Kulur et al. (2009) and Tharion et al. (2012) applied more comprehensive instructions which emphasized diaphragmatic breathing. Telles et al. (2011) comprised a BRT intervention consisting of 27 respiratory cycles, 4 times daily for a month. Their cohort was divided into four groups of 12 participants, two groups of alternate nostril breathing and a left and a right nostril only breathing group. The authors reported that nasal BRT through alternate nostrils and right nostril breathing alone, increased sympathetic activity as measured by HR Telles et al. (1994). In contrast, left nostril breathing only resulted in a reduction in sympathetic activity. ANS activity was measured by galvanic skin resistance, suggestive of reduced gland secretion. Galvanic skin response was measured once for each data set, even though other authors have suggested it should be recorded at multiple times on one day to address reliability issues (T. W. Smith & Blascovich, 1992). Further studies should investigate such a BRT method on the validated cardiac autonomic measures RMSSD and HF, as galvanic skin resistance lacks validity as an autonomic measure, and is more suited for measuring arousal

states (Y. J. Wang & Minor, 2008). Telles et al. (2011) findings are novel and therefore require confirmation by further replication studies.

Kulur et al. (2009) study consisted of diabetics, with and without ischemic heart disease, and 60 age matched healthy participants. The BRT comprised, slow breathing frequency, deep breathing, pausing at the end of inhalation, while avoiding undue upper chest movement. Thirty-two healthy participants were asked to practice the BRT intervention for 10 – 15 minutes, twice daily. HRV recordings were undertaken at 3 and 12 months following intervention during a breath consisting of 6 breaths per minute, with a period of 5 seconds for inhalation and exhalation. Kulur et al. (2009) reported that the BRT intervention groups improved HRV activity at 12 months, compared with the non-intervention healthy participants. However, Kulur et al. (2009) recorded HRV during a controlled breath, which literature previously suggested breathing be monitored during HRV recordings (undertaken by Kulur) thereby avoiding unwanted respiratory sinus arrhythmic fluctuations (Denver, Reed, & Porges, 2007).

The frequency domain HRV measures are highly altered by breath frequency and depth, however recently developed quantification methods allow valid recording of HRV during spontaneous breathing (Quintana & Heathers, 2014). For instance, the time domain measure RMSSD is less affected by breath (Penttilä et al., 2001; Saboul, Pialoux, & Hautier, 2013), with matching day-to-day variations observed for RMSSD during controlled and spontaneous breathing (Saboul, Pialoux, & Hautier, 2013).

The results of Tharion et al. (2012) study supports Telles et al. (2011) and Kulur's Kulur et al. (2009). However the methodology of data recording requires mentioning. Morning HRV was recorded in the laboratory, at least 2-4 hours after waking and following 20 minutes of supine rest. (Tharion et al., 2012) controlled for the HRV affecters: diet, exercise, alcohol and caffeine intake, however they did not control for gastric distention and bladder filling. Recent literature suggests HRV be recorded on waking, before urination, to reduce the involvement of HRV affecters (Stanley, Peake, & Buchheit, 2013). Furthermore, all three studies (Telles, Kulur & Tharion) did not effectively account for the day to day fluctuations of HRV (Heathers, 2014), by averaging as suggested previously (Heathers, 2014; Pinna et al., 2007; Plews et al., 2013).

*The Effects of Breathing Retraining on Respiratory Measures in Healthy Individuals*

Several studies have reported changes in respiratory measures following BRT in healthy participants and those with DB. Measures have included resting ventilation levels (O<sub>2</sub> and CO<sub>2</sub>) (Bernardi et al., 1998; DeGuire, Gevirtz, Kawahara, & Maguire, 1992) and respiratory rates (Bernardi et al., 2003; DeGuire et al., 1992; M. Jones et al., 2015). Studies include two pilot studies (Kraft & Hoogduin, 1984; S. E. Maclellan, G. A Silvestri, J. Ward, & D. A Mahler, 1994), a non-controlled study (Han et al., 1996), and three randomised control trials (Bernardi et al., 1998; DeGuire et al., 1992; M. Jones et al., 2015). DeGuire et al. (1996) investigated BRT in a cohort of 24 people with cardiac/breathing symptoms, M. Jones et al. (2015) on participants with initial NQ breathing symptom scores above 23 (n= 60), whereas Bernardi et al. (1998) included a sub cohort of 11 healthy individuals amongst patients who had previous cardiac failure (n = 50). All three reported positive changes in respiratory measures, including a decreased respiratory rate, following BRT, comprising deep diaphragmatic breathing. Furthermore, DeGuire et al. (1992) reported an increase in end-tidal carbon dioxide levels, Bernardi et al. (1998) an increased oxygen saturation and in a more recent study in a larger sample, Jones et al. found an improved forced expiratory volume.

*The Effects of Breathing Retraining on Breathing Symptom Questionnaire scores in Healthy Individuals*

A recent systematic review applying a PEDro analysis (Barker, M, O'Connell, & Everald, 2013) identified one study which met its inclusion criteria (Lindeboom & Vlaender-van derGiessen, 1980). Subsequently another randomised control trial has been published, investigating the effects of BRT on breathing symptoms and respiratory measures (M. Jones et al., 2015). Outcomes measures included NQ, spirometry measures, breath hold time, Hospital Anxiety and Depression Scale, as well as musculoskeletal measurements. The 60 participants in M. Jones et al. (2015) study were split into either the BRT only or BRT with MT groups (n=30), 27 BRT only and 28 BRT with MT completed at least 3 follow-up recordings. Improvements for both cohorts, in NQ, Hospital Anxiety and Depression Scale, 6-minute walk, breath hold test and some musculoskeletal measurements were reported. However, no additional benefits were observed for the BRT and MT group compared with the BRT group, suggesting no additional benefits were gained with the addition of MT. The M. Jones et al. (2015) study appears to be a well designed and implemented study, however, higher initial baseline NQ scores for BRT group may have allowed greater room for change, than the BRT and MT group. Had allocation been

more even, with respect to initial level of breathing symptoms, it is possible that the effects observed in the BRT plus MT group might have been greater than for those receiving BRT alone. Furthermore, the time of year in which the study was completed was not reported – an important omission, since seasonal variation is well known to effect respiratory health (Kaisina, Sizova, Tsirkin, & Trukhina, 2005).

#### *5.1.5 Breathing Retraining Directed at Breathing Structure*

BRT may affect the structural components of breath (e.g. related soft tissue), although this topic is largely under-investigated and seldom discussed in the literature. One study reported improvements in neck range of motion following BRT (M. Jones et al., 2015), although greater increases were observed for bilateral neck flexion when BRT was combined with MT (14.9 degrees) than BRT alone (6.9 degrees). BRT potentially improves structural components of breathing by altering vascular circulation, soft tissue compliance and resulting length tension relationships and by enhancing the neuromuscular system. Alternatively to BRT, the application of MT to initiate neurophysiological based breathing structural changes (Bialosky, Bishop, Price, Robinson, & George, 2009) has been moderately investigated.

#### *5.1.6 Conclusion:*

Conscious control of ventilation has been shown to improve HRV and respiratory measures including NQ scores. Breathing modulation has been reported to acutely alter HRV activity. A limited number of randomised control trials have also reported the long term effects of BRT on HRV. Respiratory measures, including breathing questionnaires have been reported to improve following interventions of BRT, supported by a moderate level of evidence. To date, no studies have been identified which assess the effects of BRT on HRV or other cardio-respiratory autonomic variables.

## 5.2 Manual Therapy and Breathing Structures

The second group of breathing management therapies (in addition to conscious control of ventilation) are manual and/or manipulative therapy techniques intended to influence breathing. In the context of breathing, the purpose of manual and manipulative therapy (MT) application may vary depending on the patient and their presentation. Several purposes for application of MT to improve breathing include: influencing the functions of structural components of

breathing (e.g. muscle length-tension relationships), improving respiratory measures (peak flow etc.), and improving cardiac autonomic modulation.

Manual therapists utilise the patient's movement ('active') and practitioner-facilitated ('passive') techniques to affect breathing. Manual therapeutic techniques can be categorized by the nature of application and putative interaction with body tissues (Ward, 2003). Osteopathic manual therapy (OMT) categories include soft tissue treatment (e.g. massage, inhibition, myofascial release, muscle energy technique), manipulation (high-velocity low-amplitude thrusts), mobilisation (articulation, mobilisation with movement) and 'Functional techniques' (strain-counter strain, positional release, and balanced ligamentous tension) (Ward, 2003). Osteopaths apply OMT to alter soft tissue length-tension relationships (muscle, fascia, ligaments and tendons), neural function (peripheral, autonomic and central nervous systems), fluid circulation (arterial, venous and lymphatic), modulate pain and improve biomechanical movement patterns (Bockenbauer, Julliard, Lo, Huang, & Sheth, 2002). The nature of application of OMT differs between practitioners, and between different technique categories (Farrell & Jensen, 1992), with similar health outcomes often produced independent of the techniques applied (Bialosky et al., 2009).

It has been argued that an optimal musculoskeletal system is required for optimal adaptation in response to BRT (Chaitow et al., 2014). The application of MT, through soft tissue, mobilisation, manipulation and other techniques is reported to decrease pain (Hurwitz et al., 2002; Millan, Leboeuf-Yde, Budgell, Descarreaux, & Amorim, 2012; Wong, Abraham, Karimi, & Ow-Wing, 2014), decrease muscle tension (Delaney, Leong, Watkins, & Brodie, 2002) fascial tension (Chaudhry et al., 2007), as well as improve range of joint motion (Hurwitz et al., 2002; MacLennan et al., 1994; Millan et al., 2012; Yu, Hou, Wu, & He, 2011). A decrease in breathing-related pain, muscle and fascial tension, and an increase thoracic cage range of motion should permit the optimum adaptation of a retrained breath.

### *5.2.1 Manual Therapy and Respiratory Measures*

Manual therapy induced changes to joint range of motion and muscle tension improve respiratory measures such as FEV (Rupp, 2006), FVC (Gibellini, 2002; Rupp, 2006) and peak expiratory flow (Rupp, 2006). Selected MT techniques, including soft tissue, manipulation, mobilisation and 'Functional techniques' (Chaitow et al., 2014), when applied alone are also reported to have varying effects on respiratory measures. Soft tissue techniques been reported to

decrease muscle (Delaney et al., 2002) and fascial tension (Chaudhry et al., 2007), which in theory enhances respiratory mechanics. Soft tissue techniques have been found to improve breathing parameters such as FVC (González-Álvarez et al., 2015), vital capacity (Schröder, 2003) (González-Álvarez et al., 2015), maximal respiratory pressures (González-Álvarez et al., 2015) and forced expiratory volume (Engel & Vemulpad, 2007). Strong evidence supports the application of manipulation to decrease pain and increase spinal joint mobility (Millan et al., 2012); increasing thoracic mobility should increase lung capacity. Mobilisation and Functional techniques, are implemented to decrease pain and increase mobility (Hurwitz et al., 2002; Wong et al., 2014; Yu et al., 2011), which may, if applied to painful musculoskeletal tissues involved in breathing, improve measures of respiratory function. However, several isolated OMT treatment methods alone, such as treating the pericardial ligaments, sternal recoil, cervical spine manipulations and inhalation muscle training, have shown no effect on vital capacity, forced expiratory volume at 1min, FVC and maximal expiratory flow at 25% (Weiler, 2008; Wieser, 2006), therefore further research is suggestable to distinguish the effects of OMT on respiratory measures.

### *5.2.2 Manual Therapy and Breathing Symptom Questionnaires*

Practitioners employing MT, claim to alter the function of structures involved in breathing, breathing pattern (Chaitow et al., 2014) and therefore breathing symptoms. However, to date no studies have investigated the application of MT alone on breathing symptoms questionnaires such as the NQ or SEBQ. Further investigation is required to assess MT's impact on breathing symptoms questionnaires.

### *5.2.3 Osteopathic Manual Therapy and its Effects on the Autonomic Nervous System*

In addition to the proposed effects on respiratory structures, MT, specifically OMT, is claimed to affect the ANS (Ward, 2003). In general terms, modification of the ANS can be initiated through four basic mechanisms: central command, sensory receptors, neural transmitter/receptor disruption and changes to neural pathways (e.g. compression, irritation and laceration/denervation). For MT to alter autonomic activity, it must affect the ANS through at least one of these mechanisms. The autonomic peripheral receptors of muscle and fascia are the therapeutic targets for OMT (Barnes, 1997; Schleip, 2003) to decrease pain and therefore reduce sympathetic vasoconstriction (Lewit, 2009). Central command may be influenced through the

use of education, empathy and a non-threatening consulting environment (Woolfolk et al., 2007). Several studies support claims that MT affects the ANS (Cerritelli et al., 2011; Henley, Ivins, Mills, Wen, & Benjamin, 2008; Perry, Green, Singh, & Watson, 2011). However, such studies usually comprise a single or limited range of techniques, e.g. soft tissue, manipulation, mobilisation and Functional techniques, these studies are discussed below.

A modest volume of evidence, including one randomised controlled trial (Arroyo-Morales et al., 2008), supports the use of soft tissue therapy for improving parasympathetic, autonomic activity (Arroyo-Morales et al., 2008; Borm, Schins, & Albrecht, 2004; Giles, Hensel, Pacchia, & Smith, 2012; Morgan, 2010). Manipulation has been demonstrated to alter autonomic modulation (Borm et al., 2004; Budgell & Hirano, 2001; Budgell & Polus, 2006; Roy, Boucher, & Comtois, 2009; Welch & Boone, 2008), yet it is still unclear if this change is predominantly sympathetic or parasympathetic in nature. A Theory has emerged suggesting specific manipulations of intervertebral joint (cervical, thoracic or lumbar) are associated with a specific PSNS or SNS change (Welch & Boone, 2008), although this theory is not supported by others in the field (Budgell & Polus, 2006; Reis et al., 2014). There is a reasonable body of evidence to show that spinal mobilisation results in elevated sympathetic excitatory state (Kingston, Claydon, & Tumilty, 2014). Furthermore, side specific mobilisation may initiate greater same side autonomic changes (Tsirakis & Perry, 2015). The support for Functional techniques lie in one study (Giles et al., 2012), which reports an intervention of balanced ligamentous technique, increasing the PNS measure HF.

#### *5.2.4 Conclusion*

In conclusion, few studies have investigated the effect of MT on autonomic activity. Studies that have are generally of poor methodological quality, for example, published studies do not often effectively control for daily HRV fluctuations, a well-known occurrence in HRV recording (Heathers, 2014). Additionally, conclusions are founded on low frequency and low frequency/HF indices, which do not solely measure cardiac sympatho-vagal balance, as once believed (Billman, 2013) (see Section 2). Not surprisingly, when conclusions are based on non-validated measures, conclusions drawn surrounding the effect of MT on the ANS are highly varied and often equivocal. Furthermore, results from studies often report sympathetic activity at rest (Kingston et al., 2014), an undesirable state given that chronic sympathetic excitation is

associated with negative health states, for example renal and cardiac failure (Joles & Koomans, 2004; Rajendra, Paul, Kannathal, Lim, & Suri, 2006).

### 5.3 Breathing Management: The Combined Therapy of Breathing Retraining and Manual Therapy

Breathing management for long-term health benefits could involve combinations of both BRT and MT within the same intervention. The alteration of breathing structures following MT, if not utilized, will likely return to their pre-MT states. BRT may possess the ability to utilise and maintain breathing structures altered by MT. Therefore, if BRT is applied following MT, it is possible that MT based structural changes may be maintained for longer durations.

MT is reported to directly alter respiratory measures (Engel & Vemulpad, 2007) and cardiac autonomic measures (Ke-mi et al., 2006). Furthermore, MT may ameliorate the effect of BRT on respiratory and cardiac autonomic measures, as MT can improve mobility of breathing structures (Bockenbauer et al., 2002), which may mitigate biomechanical or somatic dysfunction that interferes with motor skill training in the form of BRT. Similarly, BRT could plausibly facilitate the effect of MT on respiratory and cardiac autonomic measures by maintaining MT induced breathing alterations.

Two studies have investigated the combined effects of BRT and MT, on respiratory measures and breathing symptom-based questionnaires, McLaughlin et al. (2011) and M. Jones et al. (2015). The first, McLaughlin et al. (2011) primarily investigated the additional effect of BRT following MT on back pain and function, whereas the second M. Jones et al. (2015) primarily investigates the adjunctive effect of MT combined with BRT for dysfunctional breathers. In the McLaughlin et al. (2011) study, participants were admitted into the study after a plateau was observed in their pain and disability scores after a course of MT. Hence changes following the addition of BRT to MT suggested that BRT may be a useful adjunct to standard MT management. However, McLaughlin et al. (2011) does not report the duration of treatment, nor give a thorough description of the BRT or MT intervention implemented. The omission of this data makes the study difficult to effectively critique.

In the more recent study by (M. Jones et al. (2015)), the combined intervention of BRT and MT was reported to have no additional benefit on breathing symptoms scores (measured by NQ) respiratory measures, compared to BRT alone. However, the BRT alone group, although

randomly allocated, initially had higher NQ scores than the combined treatment group, and therefore greater opportunity for improvement. In conclusion, the study by M. Jones et al. (2015) reported that BRT is recommended as the mainstay of treatment for patients with low NQ.

No previous studies have investigated breathing management comprising both BRT and MT on cardiac autonomic measures. Moreover, no previous studies have averaged waking cardiac autonomic indices following an intervention of BRT, MT, or BRT combined with MT.

Furthermore, no studies have compared the effects of the cardiac autonomic measures HRV and HRR following an intervention of BRT, MT, or BRT combined with MT.

## SUMMARY

Several key points were identified in this literature review. Firstly, that LnRMSSD and LnHF are effective measures of cardiac autonomic activity. Secondly, that optimal breathing at rest encompasses nasal breathing into the abdomen, utilises the diaphragm, occurs with a longer exhalation phase, and each breath is followed by a pause. Thirdly, BRT, which utilises the above forms of breathing, appears to improve breathing symptoms and is likely to improve cardiac autonomic activity. Furthermore, cardiac autonomic activity and the function of anatomical structures associated with breathing are altered by MT. Finally, a combined intervention consisting of BRT and MT, is reported to improve respiratory measures including self-reported breathing symptom scores. To date no studies have investigated the combined effects of BRT and OMT on cardiac autonomic activity.

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## CHAPTER 3: DEVELOPMENT OF A COMPLEX BREATHING RETRAINING AND MANUAL THERAPY INTERVENTION

This manuscript is prepared in accordance with the International Journal of Osteopathic Medicine's guide for authors; <http://www.journalofosteopathicmedicine.com/content/authorinfo#idp1421984>.



## **Preliminary Development of a Complex Intervention for Osteopathic Management of Dysfunctional Breathing**

Short Title: **Osteopathic Management of Dysfunctional Breathing**

### **ABSTRACT**

*Background:* Breathing retraining (BRT) is commonly used during osteopathic consultations as an adjunct to osteopathic manual therapy (OMT) for assessment and treatment of breathing-related dysfunction. Although BRT and OMT are widely recognised within osteopathy and other allied health disciplines, there are few descriptions of clinically applicable protocols in the literature.

*Objective:* To describe the development of a dual-protocol framework (BRT and OMT) for assessment and treatment of dysfunctional breathing.

*Design:* Development and evaluation of a complex intervention.

*Methods:* Cyclical, iterative processes of development, feasibility and piloting, evaluation and subsequent redevelopment were applied in the design of two conceptual protocols for BRT and OMT.

*Results:* The resulting BRT protocol consists of progressive steps of breathing practice in three body positions (neutral, flexion, extension), followed by a guide for more advanced breathing challenges that can be tailored towards the individual. The OMT protocol provides a semi-standardised assessment and treatment plan, which details body regions for assessment of somatic dysfunction and a list of techniques that can be selected according to practitioner clinical judgement, based on patient presentation and preferences, and clinical context.

*Conclusions:* Here we present a clinically applicable guide for a complex intervention entailing assessment and management of dysfunctional or abnormal breathing. Implementation of this protocol within the clinical setting is now recommended, along with ongoing development, and further randomised clinical trials assessing its efficacy, effectiveness, and acceptability.

*Key Words:* Abnormal Breathing Pattern Disorders, Breathing Dysfunction, Breathing Exercises, Physical Therapy Modalities, Osteopathic Manipulation

## INTRODUCTION

Dysfunctional breathing (DB) is an alteration in the normal patterns of breathing and results in intermittent or chronic symptoms mediated through biomechanical, biochemical and psychological mechanisms.<sup>1</sup> Multifactorial, diffuse, but cumulative pathological and pathophysiological changes make DB difficult to diagnose, and a diagnosis of DB is often arrived at by exclusion.<sup>2</sup> DB may present with diverse symptoms and signs including respiratory, cardiac, neurological, metabolic and gastrointestinal presentations.<sup>3</sup> Many of these symptoms arise from respiratory alkalosis brought about by chronic or transient bouts of hyperventilation and the term ‘hyperventilation syndrome’ has often been used to describe this state.<sup>4</sup> However, it is now accepted that the clinical picture of DB encompasses more than traditionally recognised hyperventilation syndrome, since experimentally provoked hyperventilation will not consistently elicit symptoms, and symptoms may appear in the absence of decreased end-tidal pCO<sub>2</sub>.<sup>5,6</sup> Furthermore, distinctions have recently been made between thoracic (involving ventilatory alterations), and extra-thoracic (e.g. vocal cord dysfunction) forms of DB.<sup>3,7</sup>

Symptoms arising from DB may occur independently of other medical conditions or secondary to them.<sup>4,8,9</sup> DB is also strongly associated with anxiety and affective states.<sup>10</sup> To date there has been no attempt to establish a consensus on diagnostic criteria for DB. For this reason, and because most evidence associating DB with other medical conditions is cross-sectional, it is difficult to establish whether conditions may cause or exacerbate DB or, conversely, when symptoms arising from DB exacerbate the existing condition.

Various interventions have been developed to address DB.<sup>11,12</sup> These can be broadly classified into two groups: (i) those that focus on improving conscious neuromuscular control of ventilation, commonly referred to as ‘breathing retraining’ (BRT); and (ii) those that apply manual therapy to improve the mechanical function of body structures involved in breathing.

Courtney and Greenwood,<sup>13</sup> and more recently Chaitow,<sup>14,15</sup> have outlined principles of osteopathic assessment and management of DB. Despite this, there are few clear descriptions of a practical osteopathic approach to DB in the literature. We propose that a comprehensive approach should encompass both BRT and osteopathic manual therapy (OMT). The aims of BRT are to aid neuromuscular reacquisition of normal breathing patterns and to utilise and reinforce alterations in respiratory function facilitated by OMT. OMT can improve mobility of breathing structures,<sup>16</sup> which may mitigate biomechanical or somatic dysfunction that interferes with motor skill training in the form of BRT. Thus, BRT and OMT may be co-dependent within an intervention.

Complex interventions comprise multiple interacting features, those arising from the intervention itself, as well as from a diversity of behavioural characteristics of both patients and practitioners.<sup>17</sup> Craig *et al.*, 2008 have described a process of development, feasibility and piloting, evaluation, and implementation as key elements in the design of complex interventions. Here we describe iterative cycles of these elements employed in the initial development and preliminary evaluation of two protocols for an individualised approach to osteopathic management of DB, incorporating both BRT and OMT.<sup>17</sup>

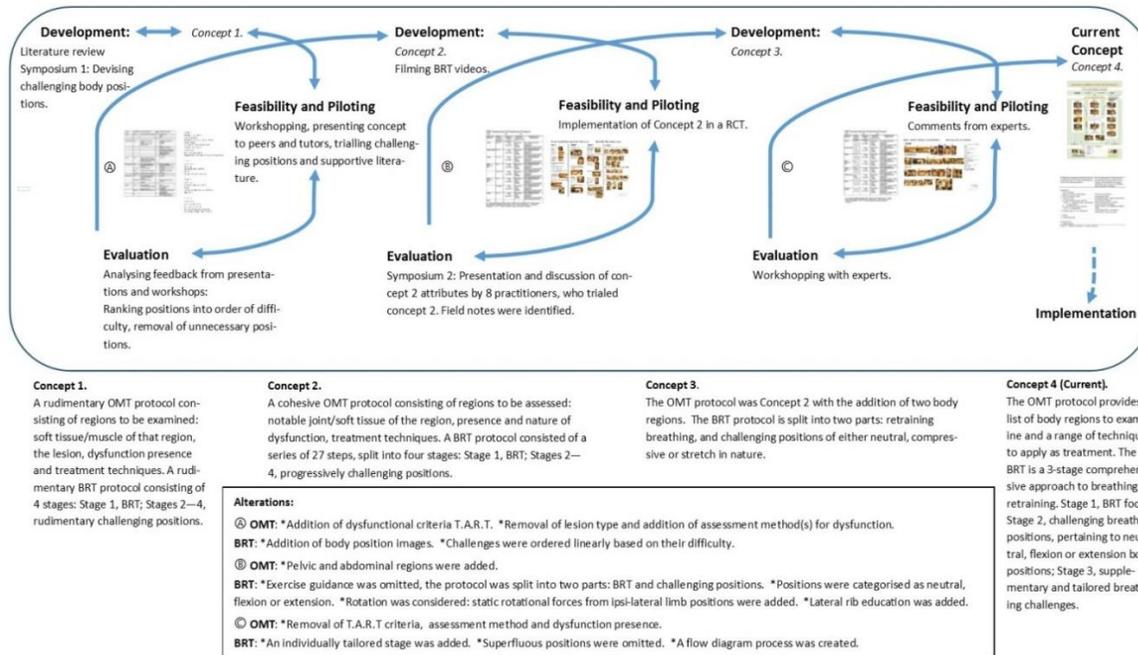
## METHODS

### Development Phases

The initial purpose was to develop an intervention for delivery in a randomised-controlled clinical trial investigating the effect of BRT and OMT on dysfunctional breathing symptoms, cardiac autonomic measures and exercise economy ([*clinical trial registration number and institutional name redacted for review*] Research Ethics Approval 2013-1080). Key processes for development of BRT and OMT protocols were adapted from Craig *et al.*'s 2008 model for developing and evaluating complex interventions.<sup>17</sup> Craig *et al.* argue that in practice, the four elements of their model (Development, Feasibility and Piloting, Evaluation, and Implementation) may not occur in a linear or even cyclical sequence. Here, we describe three iterative cycles of Development, Feasibility and Piloting, and Evaluation leading to the development of a complex intervention ready for Implementation (Figure 3.1).

### First Iteration: Concept 1

The first stage of development entailed identification of the evidence base for breathing interventions, underlying theory surrounding mechanisms and current clinical ideas and practice. A literature review was undertaken and a 3-hour symposium was organised by the authors (Symposium I), at which clinicians with a special interest and expertise in DB discussed recognition, assessment and management of the condition within their practice. Symposium I concluded with a discussion about the common features of optimal management of the patient experiencing DB symptoms. Included in this discussion were suggestions about the likely clinical aims of OMT as well as of body positions that would provide sufficient challenge for practice of diaphragmatic breathing. Thus, protocols for an integrated intervention which included plans for OMT plus specific BRT were developed from this symposium (Concept 1). The BRT protocol was designed to allow home-based practice for the purpose of improving habitual breathing patterns. It was supported by online and printed materials and guidelines for regular assessment of progress. The OMT protocol was a goal-oriented semi-standardised osteopathic assessment and treatment plan for somatic dysfunction associated with breathing dysfunction. Feasibility testing and piloting of Concept 1 involved a series of informal workshops at which the concepts were presented to osteopathic students and tutor/clinicians, who applied the suggested approaches on each other and provided feedback.



*Figure 3.1. Development and Evaluation of a Complex Intervention for Dysfunctional Breathing.* Abbreviations are as follows: BRT, breathing retraining; OMT, osteopathic manual therapy; RCT, randomised controlled trial; TART, texture, asymmetry, range of motion, and tenderness.

## Second Iteration: Concept 2 (Clinical Trial) Protocols (Appendix 3 & 4).

Feedback from the informal workshops was analysed, both protocols were revised to develop Concept 2 which was then utilised as the intervention in the clinical trial. For the BRT, the order of exercises was adapted to facilitate gradual progression; and for OMT, simplified guidelines for assessment of somatic dysfunction were added. Eight osteopath clinicians evaluated the intervention while treating participants recruited as part of the clinical trial. Clinicians included both clinical teaching staff ( $n = 3$ ), and private practitioners ( $n = 5$ ). All except one of these clinicians attended Symposium I, and all were invited to participate in a second 3-hour symposium (Symposium II), scheduled 9 months after the first. Five of the clinicians prepared a presentation for Symposium II, and the remaining three provided written or verbal feedback to authors, for which they were instructed to critique the Concept 2 protocols used within the study and during their routine clinical practice. They were asked to consider practical utility, and aspects requiring development and modification. In order to synthesise information coming from these critiques, two investigators ([*author initials redacted*]) independently produced field notes from the feedback provided.

### Third Iteration: Concept 3

On the basis of the evaluative process of Concept 2 protocols following Symposium II, the development team made modifications to the protocols which were then documented as Concept 3. Several key changes were made to the BRT and OMT protocols. The BRT protocol was rearranged into 3 stages from the original 27 steps, several body positions were removed and more emphasis was placed on 'lateral rib breathing'. The OMT protocol received two further specifically-identified regions for assessment of somatic dysfunction: the pelvis and abdominal wall regions. Concept 3 protocols were distributed to practitioners and amongst the research group for comment. Further minor refinement took place following informal presentation and discussion with field experts at a national osteopathic conference, resulting in the development of Concept 4 protocols presented here. The OMT protocol was completely unaltered between Concepts 3 and 4. Most of the changes within the second and third iterations therefore occurred in the adaptation of the BRT protocol. The key changes made at each stage of concept development are summarised at the foot of Figure 3.1.

## RESULTS

### Concept 4: Breathing Retraining (BRT) Protocol

The resulting Concept 4 BRT protocol is arranged in three stages. Stage 1 focusses on developing components of supposed optimal breathing (Figure 3.2). Stage 2 consists of three routines which each progressively introduce a series of challenging breathing positions emphasising neutral, flexed or extended body positions. The order of the flexion and extension routines is interchangeable, with the position providing the lesser challenge to the performer implemented first (Figure 3.3). Stage 3 comprises supplementary breathing challenges and a guide to tailored breathing challenges (Figure 3.3).

## STAGE 1 BREATHING RETRAINING PROGRESSION

### Step 1: Nasal passage awareness

Feel the air flow in through the nose into the back of the throat.  
Feel the warm air coming out through the nasal passage and over the lip. This can be aided by pursing the upper lip or placing a finger on top of the lip.  
Maintain awareness of breathing; keep thinking about the air flowing through your nose.



### Step 2: Hi-Lo awareness

Place one hand on your chest the other over your abdomen (in between your ribs and navel), feel which hand rises the most.



### Step 3: Nasal breathing with abdominal movement

The aim here is to breathe through your nose, ensure your abdomen rises during your in breath and falls during your out breath. Your upper chest should stay motionless. Feel your lower ribs move out to the side as well as to the front and back. Awareness of motion can be aided by using a towel or belt wrapped around the abdomen, or by placing a weight upon the abdomen or chest. Blow a balloon up 1/4 full, keep it in your mouth as seen on the video. If you use your mouth to breathe, the balloon will change in size, ensure this does not occur.

### Step 4: 'Out' breath longer than in breath.

The length of your out breath should be equal to, or longer than your in breath. Comfortably alter your breath, stay relaxed, changing it when ready.

### Step 5: Pause after out breath

After the end of each out breath pause for a second, then inhale.

### Step 6: Evenness of breath

Ensure that each breath is as deep/shallow as all the previous and the duration of breaths are the same.

### Step 7: Steps C, D, E and F. (Nasal, Out, Pause, Evenness = "N.O.P.E")

Note that the Steps 3 to 6 are the characteristics features of a 'good breath'. To help make this memorable we use the acronym "N.O.P.E", where N = Nasal breathing, O = Out longer than in, P = pause at end of our breath, E = evenness of breath. Give it a go.



### Step 8: N.O.P.E while lying on your back with your knees bent.



### Step 9: N.O.P.E while lying on your back, legs straight.



### Step 10: N.O.P.E while seated.



Nasal  
Out  
Pause  
Evenness

Stage 1 Breathing Retraining Quick Reference  
Version 2.1

Figure 3.2. Concept 4 breathing retraining protocol, Stage 1.

*Stage 1 of breathing retraining (BRT) protocol, consisting of 10 challenge steps. Breathing awareness is developed (Steps 1 & 2); a series of independent breathing patterns are taught (Steps 3 – 6); then combined (Step 7). Finally, the combined breathing pattern is maintained during seated and supine body positions (Steps 8 – 10).*

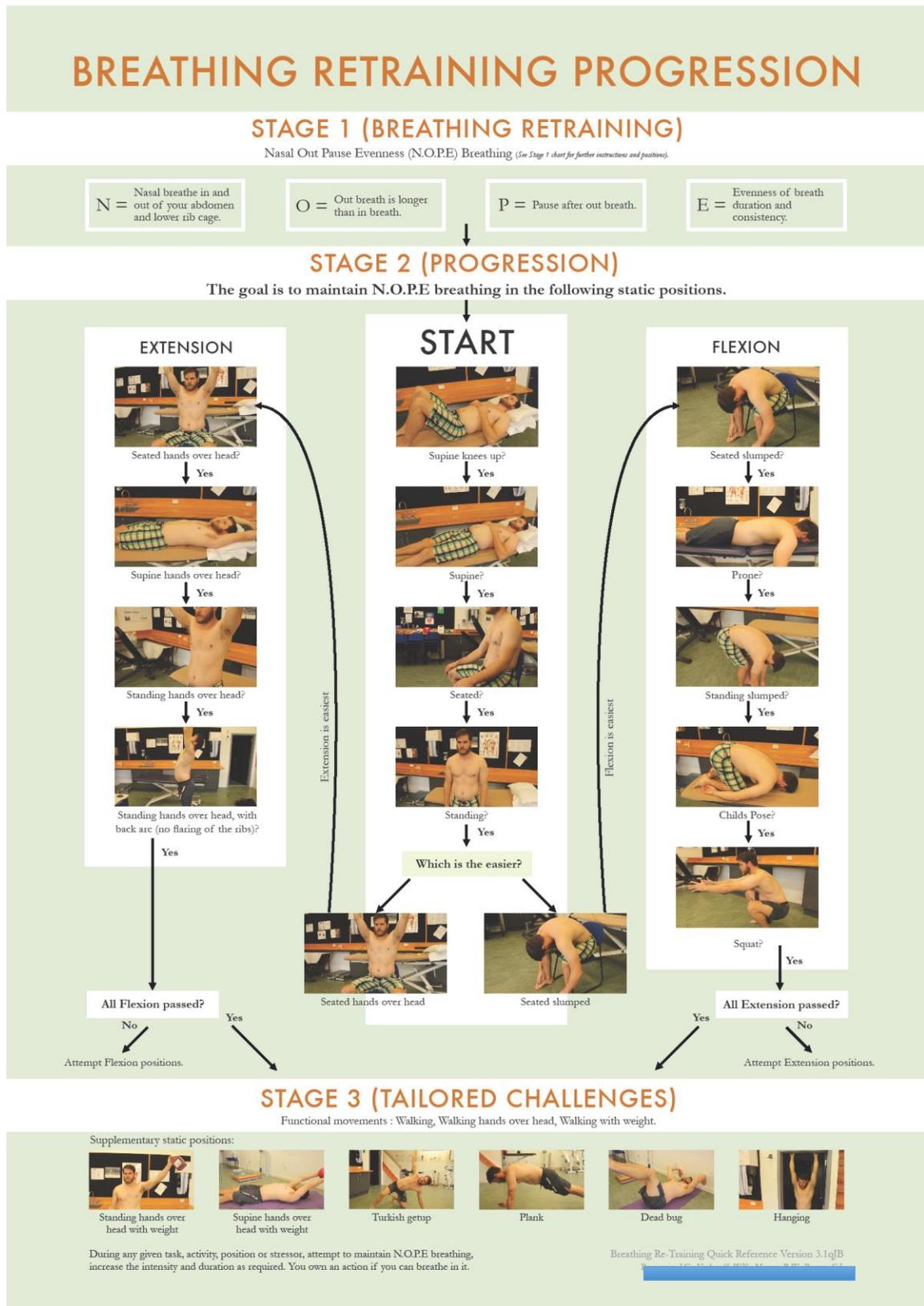


Figure 3.3. Concept 4 breathing retraining protocol, all stages.

The entire progression in the Concept 4 breathing retraining (BRT) protocol. Stage 1: Breathing retraining focus; Stage 2: Challenging breathing positions, pertaining to neutral, flexion or extension body positions; Stage 3: Supplementary and tailored breathing challenges.

Nasal, Out, Pause, Evenness (N.O.P.E.) acronym cues nasal breathing, exhalation longer than inhalation, pause following inhalation, and evenness of breath.

#### Concept 4: Osteopathic Manual Therapy (OMT) Protocol

The resulting Concept 4 OMT protocol was designed as a semi-standardised assessment and treatment approach, which provided a list of body regions most relevant to addressing somatic components of breathing dysfunction and a range of techniques selected according to practitioners' clinical judgements, based on patient presentation and preferences and clinical context (Table 3.1). An accompanying treatment worksheet and notation system was also developed (Appendix 5).

Table 3.1. Osteopathic manual therapy assessment and treatment approach.

A. Physical examination and assessment to identify presence of somatic dysfunction in each of the following regions:	B. For each region in which somatic dysfunction is present, address by selecting, at the practitioner's clinical judgement, one or more of the following osteopathic techniques:		
<table> <tr> <td data-bbox="181 1071 698 1543"> <p><i>Mandatory</i></p> <ol style="list-style-type: none"> <li>1. Cervical spine (upper, and mid-cervical spine)</li> <li>2. Cervico-thoracic spine</li> <li>3. Thoracic spine               <ol style="list-style-type: none"> <li>3a. Upper thoracic spine and ribs (ribs 1–2 ; sternoclavicular, acromioclavicular joints)</li> <li>3b. Lower thoracic spine and ribs (ribs 3–12; diaphragm)</li> </ol> </li> <li>4. Lumbar spine (L1–S1; iliopsoas)</li> <li>5. Pelvis</li> <li>6. Abdominal wall</li> </ol> <p><i>Discretionary</i></p> <ol style="list-style-type: none"> <li>1. Other region/s</li> </ol> </td> <td data-bbox="727 1134 1172 1470"> <p>Soft-tissue techniques</p> <p>Myofascial release</p> <p>Positional release / strain-counterstrain</p> <p>Functional technique</p> <p>Balanced ligamentous tension</p> <p>Muscle-energy techniques</p> <p>High-velocity, low-amplitude manipulation</p> <p>Joint mobilisation / articulation</p> <p>Mobilisation with movement</p> </td> </tr> </table>		<p><i>Mandatory</i></p> <ol style="list-style-type: none"> <li>1. Cervical spine (upper, and mid-cervical spine)</li> <li>2. Cervico-thoracic spine</li> <li>3. Thoracic spine               <ol style="list-style-type: none"> <li>3a. Upper thoracic spine and ribs (ribs 1–2 ; sternoclavicular, acromioclavicular joints)</li> <li>3b. Lower thoracic spine and ribs (ribs 3–12; diaphragm)</li> </ol> </li> <li>4. Lumbar spine (L1–S1; iliopsoas)</li> <li>5. Pelvis</li> <li>6. Abdominal wall</li> </ol> <p><i>Discretionary</i></p> <ol style="list-style-type: none"> <li>1. Other region/s</li> </ol>	<p>Soft-tissue techniques</p> <p>Myofascial release</p> <p>Positional release / strain-counterstrain</p> <p>Functional technique</p> <p>Balanced ligamentous tension</p> <p>Muscle-energy techniques</p> <p>High-velocity, low-amplitude manipulation</p> <p>Joint mobilisation / articulation</p> <p>Mobilisation with movement</p>
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Notes: L = lumbar vertebrae; S = sacral vertebrae.

## DISCUSSION

This report describes the preliminary development of a treatment and management approach that can be practically implemented with individuals who exhibit signs or symptoms of DB. The development of this integrated BRT and OMT approach was undertaken with the collaboration of practicing osteopaths and other clinicians with expertise in breathing dysfunction. Clinician feedback on the concept protocols was reflective and diverse and occurred at several stages, enabling consideration of their use in a wide range of contexts and adaptations which are likely to improve general clinical applicability.

Several ideas that arose from evaluation within the three development cycles warrant discussion. Firstly, various collateral benefits of following the BRT protocol have been identified. Secondly, though feedback from practitioners indicated acceptable face validity and applicability within routine clinical settings, the applicability of the BRT to the clinical situation was the focus of critical reflection at all stages of the evaluation process. Reflection occurred particularly in relation to session duration and number of challenge steps in the protocol. The possibility that some steps in the BRT protocol might be redundant in most clinical situations was considered.

For the OMT protocol, evaluation has centred on its purpose and use amongst practitioners with a diverse range of practice styles. Further discussion pertaining to both protocols and their integration includes the implications of assessing the Concept 2 (Clinical Trial) protocols on healthy active individuals and the length of time required to complete the protocols in relation to a typical osteopathic consultation.

### Collateral Benefits of the BRT Protocol

An unanticipated outcome for use of the protocol was that previously unidentified dysfunction became apparent in the course of delivering the BRT. Using the tool sometimes uncovered musculoskeletal dysfunctions such as impaired range of movement or motor patterns, for example an inability to lateral rib breathe was highlighted in flexion positions. Sometimes dysfunctions unrelated to the main purpose of the test were highlighted when individuals failed to assume positions, for example poor ankle dorsiflexion was identified during a failed squat attempt in Stage 2 (Figure 3.3). Alternatively, musculoskeletal dysfunction noted in body regions indirectly associated with breathing function were reported to affect breathing in certain body positions, such as a propensity to upper rib breathe during ipsilateral glenohumeral external rotation, with shoulder joint dysfunction.

Another indirect benefit of BRT implementation noted in the evaluation process was an observed improvement of breathing and whole body awareness that sometimes developed simultaneously with its use. Improved awareness of body functions, termed 'interoception',<sup>18</sup> could possibly increase the ease of acquisition of the BRT challenges, however this needs to be further investigated.

### Applicability of the Protocols to the Clinical Situation

The length of time required to integrate these protocols may exceed the duration of routine osteopathic consultations in usual practice. In relation to the BRT protocol, evaluators made a number of suggestions about how the process could be adapted to address this. For example, some clinicians reported ‘cherry-picking’ challenging positions applicable to their patient and testing-retesting the patients only on these challenges. Furthermore, some clinicians, particularly in the evaluation of the Concept 2 protocol, considered some steps to be redundant in most clinical situations, and that shortening and simplifying the protocol might improve its applicability.

We have attempted to incorporate modern principles of motor skill development within the BRT protocol. A current model for developing a well-learned, resilient motor pattern is to allow exploration and practice of the best movement solution within a range of practice conditions. Schöllhorn *et al.* argue for the importance of applying variations in kinematic and kinetic properties of movement in its acquisition to stabilise learning.<sup>19</sup> These authors demonstrate 94% greater improvement in a group of young hurdlers randomly assigned to a 6-week training programme incorporating variability in body positions and practice tasks. The programme was oriented towards developing individual and situational movement solutions in hurdling, compared to a more traditional practice towards mastery of traditional drills displaying increasing similarity to the supposed optimal movement technique.<sup>19</sup> Therefore in the final BRT protocol, additional scope for adapting breathing in a greater variety of situations has been provided. The range of possible movements allowed in these situations would also have been increased by the OMT. We recommend application of as many steps as can reasonably be achieved within the available time-frame without compromising quality.

A final issue of applicability was that practitioners found it difficult to observe breathing in positions where movement or body parts obstructed observation. For example, during seated slumped when the anterior chest view is obstructed. The suggestion was made to assess such cases with the aid of palpation.

### Purpose and Application of OMT Protocol

The differing practicing methodologies inherent amongst osteopaths make it difficult to develop a semi-standardised assessment and treatment that is universally applicable. The evaluation process revealed that different clinicians used the OMT protocol in slightly different ways. Some found that using the protocol markedly changed their normal practice routine.

The OMT protocol was designed as a semi-standardised guide for treatment and also as a systematic generalised recording tool for the purposes of describing a research intervention. It was intentionally designed to accommodate a range of practitioner preferences in administering examination and treatment

techniques, whilst setting some constraints. It was not intended to provide a prescription, nor to represent or replace clinical reasoning and practitioner judgement in determining the most appropriate form of OMT applied. An effort was made to create a balance between very detailed explanations of assessment or treatment and abbreviated reporting, which may have incurred a degree of generalisation not suiting all practitioners' approaches to practice.

During evaluation of the Concept 2 protocol it was highlighted that the OMT protocol lacked hierarchy surrounding the severity of the dysfunction(s) presented. This meant some practitioners were sometimes unsure on an order of treatment priority for an individual's dysfunctions. An attempt was made for subsequent protocols to provide greater clarity of the intention that the semi-standardised plan was not to specifically direct treatment but to provide a framework that encouraged individual practitioner judgement on the basis of the practitioner's own clinical reasoning in each case.

Some practitioners also noted that following the protocol resulted in a substantial change from their usual treatment approach. A sense of compromising 'flow' during consultation was sometimes noticed, and this may reflect the need for further development around operationalising the protocol in practice. Whilst some practitioners tended to deviate from their regular treatment in order to accommodate the semi-standardised approach, others maintained their regular approach, which they attempted to retrospectively fit into the model presented. Practitioners reported that they found this tool effective as an assessment chart and acknowledged that the protocol be useful as a 'checklist' for novice and trainee osteopaths.

#### Limitations of Testing the Protocol on Healthy Active Individuals

During evaluation of the Concept 2 (Clinical Trial) protocols, clinicians noted that involving relatively healthy patients as models may have influenced the degree to which the protocol could be applied in a clinical setting. Practitioners pointed out that patients' pre-existing conditions or health status could affect the management and the progress of using the intervention. Sometimes these specific conditions tended to direct treatment planning, rather than the breathing itself. At other times, breathing issues would not resolve until a dysfunction located outside of respiratory structures was addressed, suggesting that even peripheral dysfunctions, such as a sprained ankle, may affect breathing function and health.

Secondly, the patterns of dysfunction highlighted in the evaluation of the Concept 2 BRT protocols may have been specific to the activities undertaken by patients. The active healthy adults here were mainly endurance athletes or regularly participated in high-intensity strength and conditioning exercise. Levels of strength, fitness, flexibility and motor pattern adaptability were likely to have positively influenced their ability achieve the challenges and also their ability to perform the positions, for example squatting. Implementation of this protocol on a more typical clinical population might present different challenges

arising from greater difficulty in performing positions and achieving the more advanced breathing challenges.

Thirdly, practitioners pointed out that compliance would be an important success factor for the BRT intervention. The physically active patients who volunteered to take part in the trial for which this intervention was developed were mainly sportspeople, and were possibly more highly motivated to improve their breathing than many clinically-treated DB patients. Therefore, we consider that this group may have displayed a greater level of compliance than is typical within the clinical setting. Attaining motivation and compliance might have been difficult for steps that provided greater challenge to the individual. It was felt that these could be addressed through practitioner-designed, patient-specific interim challenges.

Exercise is associated with relative protection from a range of psychological health disorders.<sup>20</sup> It is therefore also possible that active participants might respond more favourably to the intervention. We recognise that neither protocol may be effective without consideration of psychosocial elements that often underpin or are closely linked to disturbed breathing patterns.<sup>21,22</sup> BRT may also have a role in the treatment of anxiety and depression.<sup>23</sup> There are multiple components of DB, and the protocols developed here focus primarily on two: motor skills and somatic dysfunctions likely to be associated with breathing, but at present the described protocols do not specifically address psychosocial issues that may be important determinants of dysfunctional breathing.

### Suggestions for Further Research

This investigation does not extend to a true implementation phase of a complex intervention, and implementation in a broader clinical setting would be the next logical step. Implementation is envisaged by Craig *et al.* as including dissemination, ongoing surveillance and monitoring and longer-term follow-up.<sup>17</sup> We argue that evaluation of a complex intervention should be a continuous process, maintained through feedback following implementation, as well as from subsequent research. Further, we suggest that clinical and physiological outcomes of using this protocol should next be assessed in rigorously-designed clinical trials.

There is also potential for further exploration of specific instructions or non-verbal cues, for example using a rolled towel under the thorax to mobilise breathing structures, or promotion of breathing awareness through inflating a balloon. Although such cues were applied in the BRT protocols during development, they could be extended. Further work might alternatively focus on development of BRT in stressful situations or as an intervention for breathing-mediated anxiety.

## CONCLUSIONS

Past literature on DB provides a comprehensive theoretical basis for understanding an approach to treatment but perhaps does not go far enough in providing practical guidelines. Here we report the development and evaluation of a novel dual-protocol framework for clinical assessment, diagnosis and management of DB in the clinical setting. Implementation of this protocol within the clinical setting is now recommended, along with ongoing development, and further randomised clinical trials assessing its efficacy, effectiveness, and acceptability.

## WHAT THIS PAPER ADDS

- A novel, clinically-applicable osteopathic approach to dysfunctional breathing is described
- The cyclical process of development, piloting and evaluation of the complex intervention presents a valuable model for designing osteopathic interventions
- The intervention was developed in consultation with a large number of clinicians and experts in the field
- Implementation and further randomised clinical trials assessing its efficacy, effectiveness and acceptability are recommended

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## CHAPTER 4: RANDOMISED CONTROL TRIAL

This manuscript is prepared in accordance with the European Journal of Applied Physiology guide for authors; <http://www.springer.com/biomed/human+physiology/journal/421>.

The Effect of Osteopathic Manual Therapy with Breathing Retraining on Cardiac Autonomic Measures and Breathing Symptoms Scores: A Randomised Controlled Trial.

**ABSTRACT:****Purpose:**

Breathing retraining (BRT) and manual therapy (MT), delivered independently or together influence autonomic activity and improve symptoms thought to be associated with dysfunctional breathing. This study evaluated the effects of BRT and osteopathic MT on cardiac autonomic measures and dysfunctional breathing symptoms during spontaneous breathing in healthy active adults.

**Methods:**

Participants received BRT and four, weekly MT sessions, randomised to start immediately or after a 6-week delay. Heart rate variability (HRV) was assessed as a 7-day average of waking 6-min electrocardiogram recordings, using time (logarithm of the root mean square of the successive differences; LnRMSSD) and frequency domain (logarithm of high frequency; LnHF) measures. Recordings were taken before, 1 week following intervention or delay, and then following the later intervention for those with a delayed start. Changes were compared between those who received or had yet to receive the intervention, and for the whole cohort before and after treatment.

**Results:**

Following the intervention, LnRMSSD and LnHF increased 3–4% compared with control: immediate start group, LnRMSSD 0.27 (0.09–0.45 95%CI) Ln ms, and LnHF 0.30 (-0.04–0.70) Ln ms<sup>2</sup>; delayed start (pre-intervention) group, LnRMSSD -0.09 (-0.26 – 0.08) Ln ms, and LnHF -0.19 (-0.52–0.15) Ln ms<sup>2</sup> ( $P=0.02$ – $0.03$  for interaction). For dysfunctional breathers, breathing questionnaire scores decreased after intervention, but not compared with control: Cohort change NQ 10.0 (4–16,  $P=0.007$ ); SEBQ 20.1 (13–27,  $P=0.001$ ).

**Conclusion:**

A 6-week osteopathic treatment consisting of BRT and MT improved HRV compared to no treatment, and breathing symptom questionnaire scores of dysfunctional breathers.

Keywords:

Breathing Exercises, Heart rate recovery, Heart rate variability, Osteopathic Manipulation, Physical Therapy Modalities.

## INTRODUCTION

The relationship between the autonomic nervous system (ANS) and breathing is complex (Eckberg 2003), with conscious and subconscious control of respiratory rate and depth affecting, and being affected by, the ANS (Kirkman 2014; Ley 1994; Wang et al. 2010). Conscious alterations of breathing allow a direct cognitive influence on subconscious neurophysiological autonomic mechanisms (Eckberg 2003). Breathing interventions may therefore be useful for modulating autonomic activity.

Research and clinical interest in the ANS arises from its relationship with various morbidities, for example cardiac failure (Ershler and Keller 2000; Gorman and Sloan 2000), diabetes (Kiecolt-Glaser et al. 2002) and psychological disorders (Bär 2015). Autonomic state can be quantified with cardiovascular and respiratory measures (Brierly-Bowers et al. 2011). Cardiac autonomic measures include resting heart rate (HR), heart rate variability (HRV) and heart rate recovery (HRR), and reflect cardiac autonomic innervation (Buchheit 2014). These cardiac autonomic measures are non-invasive, inexpensive (Hall 2010), validated (Al Haddad, Laursen, Chollet, Ahmaidi, & Buchheit, 2011) and therefore attract considerable research interest (Al Haddad et al. 2011).

Heart rate variability is the durational variance between heart beats, with greater variance indicative of greater parasympathetic (PSNS) activity (Guidelines 1996). HRR represents the strength of parasympathetic re-innervations following exercise: a higher HRR represents faster PSNS reactivation (Peçanha et al. 2013). Although HR, HRV and HRR measure autonomic activity, their measurements recorded simultaneously are not always highly correlated (Dupuy et al. 2012; Esco et al. 2010b). Several variables, for example age, diet, excretion, medication, comorbidities, and breathing (Heathers 2014) influence cardiac autonomic activity, hindering interpretation of cardiac indices as indicators of overall ANS state. HRV, in particular, is highly variable and susceptible to external influences (Plews et al. 2013). Acute and chronic breathing alterations impact cardiac autonomic measures in those with established breathing disorders (Handa et al. 2012), and in healthy individuals (Telles et al. 2011a), in whom shallow rapid breathing has been shown to reduce HRV.

The term dysfunctional breathing (DB) has been defined in several different studies (Barker and Everard 2015; Courtney 2009), but no consensus-based definition exists. Various interventions

and philosophical perspectives for breathing management have been developed and described (Chaitow et al. 2014). We categorise breathing management interventions into two groups: (1) those that focus on conscious control of ventilation (including acutely-implemented breathing manipulation, and longer-duration breathing retraining); and (2) those that apply manual therapy (MT) to influence breathing.

Breathing manipulation, often applied in a single session, has well-reported short-term effects on various physiological functions such as forced vital capacity, forced expiratory volume and heart and respiratory rate (Bruton et al. 2007; Yamaguti et al. 2012). Breathing retraining (BRT), in contrast, is taught over a period of time and promotes active nasal and abdomen spontaneous breathing whilst avoiding undue upper chest movement (Chaitow et al. 2002). It has been shown to reduce respiratory symptoms (Thomas et al. 2009), improve respiratory measures (Saboul et al. 2015) and increase PSNS activity (Kulur et al. 2009). These beneficial effects of BRT have been served in cohorts with conditions such as asthma (Thomas et al. 2009), chronic obstructive pulmonary disorder (Gosselink 2003), constipation (Silva & Motta, 2013) and reflux (Eherer et al. 2012; Gosselink 2003; Silva and Motta 2013; Thomas et al. 2009). The second category of intervention, MT, may influence breathing by targeting the nervous and the musculoskeletal system, to produce neurophysiological changes responsible for clinical outcomes, such as altered tissue texture, improved range of motion and decreased pain (Bialosky et al. 2009).

Breathing management may comprise both BRT, for respiratory motor skill re-acquisition and reinforcement, and MT to mitigate biomechanical dysfunction that could interfere with motor skills gained from BRT. Indeed, spinal pain and disability scores have been observed to improve following the addition of BRT to ongoing MT (McLaughlin et al. 2011). The combined effects of MT with BRT have also been reported in another study to reduce breathing symptoms, although no additional benefit was observed with the application of MT to BRT (Jones et al. 2015). Along with these demonstrated improvements in pain, disability and breathing symptoms, the combined intervention of BRT and MT potentially alters the ANS. The literature reporting the effects of BRT on HRV during controlled and spontaneous is sparse, with only two RCT studies reporting such effects during controlled breathing (Kulur et al. 2009; Telles et al. 2011b) and one during spontaneous breathing (Tharion et al. 2012). These studies were conducted in people with diabetes and ischaemic heart disease (Kulur et al. 2009) or healthy

participants (Telles et al. 2011b; Tharion et al. 2012??), and there is no evidence that observed effects can be extrapolated to dysfunctional breathers. To our knowledge, no study has reported the combined effects of MT and BRT on autonomic measures, during either spontaneous or controlled breathing, in healthy individuals or dysfunctional breathers. The primary aim of this study was to investigate the effects of a combined BRT with MT intervention on cardiac autonomic measures during spontaneous breathing on healthy individuals who have concern about their breathing. Secondary aims were to investigate the effect of this intervention on breathing symptoms questionnaire scores, and to determine relationships between breathing questionnaire scores, the extent of progression during a BRT protocol, and cardiac autonomic measures.

## METHODS

### Design

A randomised controlled trial was performed during which participants were randomly assigned to begin a simultaneous BRT and MT intervention immediately (immediate start group) or after 6 weeks delay (delayed start group) (Table 4.1). This design allowed for three levels of analysis. Firstly, it was possible to compare the effect of the 6-week intervention with a control condition. Secondly, because all participants eventually received the intervention, an additional single-cohort analysis of change from pre-intervention to 6-week post-intervention was conducted. Thirdly, an explorative analysis of the correlations between breathing outcome variables and their changes was undertaken. Cardiac autonomic and breathing symptom questionnaire measures were collected pre- and post-intervention, during two visits to the laboratory for the immediate start and three for the delayed start group (Table 4.1).

This was a sub-study embedded within a primary study also investigating exercise economy and spirometry measures (unpublished data). All participants gave written informed consent and the study was approved by Unitec Ethics committee (UREC Approval 2011-1196) and registered with the Australian New Zealand Clinical Trials Registry (ACTRN12613001267741).

Table 4.1: Participant Procedure Schedule.

Week	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	
IS	Pre-intervention 1	Intervention					Post-Intervention									
DS	Pre-intervention 1	Delay					Pre-intervention 2			Intervention					Post-Intervention	

Both the immediate start and delayed start groups received intervention, although the delayed start group after a delay (D). This design allowed two analysis. For the RCT analysis, the immediate start pre-intervention and post-intervention recordings were compared with the first second pre-intervention recordings (pre-intervention 1 and 2) of the delayed start group. For the cohort analysis, the immediate start group's pre-intervention measurement was combined with the average of pre-intervention 1 and 2 for the delayed start group recordings, against which post-intervention recordings for both groups were compared.

## Participants

### *Sample Size:*

Calculations prior to the study determined that a sample size of 15 for the entire cohort would be required to detect change effect sizes of 0.8, assuming a level of significance of 0.05 and statistical power of 0.8. An effect size of 0.8 equates to a change of approximately 0.24 ln ms (Pinna et al. 2007), or 9 points in SEBQ (Mitchell et al. 2015).

### *Recruitment*

Between December 2014 and May 2015, participants were recruited from the general public within the Auckland, New Zealand area using online advertising [Appendix 6]. Respondents undertook an online inclusion criteria screening [Appendix 7]. Inclusion criteria required participants to be aged 19 – 45 years, have no known cardiac or autonomic disorders, have no history of smoking or respiratory-related hospital admission in the previous 6 months, participate in at least 4 hours of exercise per -week, perceive that breathing may be limiting their sporting performance, and have no diagnosed affective disorders. Following informed consent, participants were allocated to immediate or delayed intervention groups, in the order recruited, using a single block randomisation schedule generated prior to the start of enrolment, using <http://www.randomization.org>. The allocation was concealed to study personnel involved in recruitment. Participants were not made aware that a deliberate delay in treatment was part of the design.

### *Outcomes Measures*

Primary outcome measures were natural logarithm of the root mean square of successive differences (LnRMSSD) and high frequency (LnHF) for HRV assessment. Secondary measures were online-administered dysfunctional breathing symptoms questionnaire scores: Nijmegen

(NQ) and Self-Evaluation of Breathing Questionnaire (SEBQ) (Chaitow et al. 2014); HRR measures at 30s (HRR<sub>30</sub>), 60s (HRR<sub>60</sub>) and 120s (HRR<sub>120</sub>); and BRT progression step achieved (range 1 to 27).

Heart rate (HR) and HRV was measured upon waking, as per verbal and written instructions provided to participants [Appendix 8] for 7 consecutive days prior to laboratory visits, at which HRR was recorded. Cardiac autonomic recordings were transferred from the heart rate monitors to the Polar ProTrainer 5® software, saved, exported and imported into Kubios HRV software (version 2.2, 2014). For HRV indices, ectopic beats were corrected within Kubios to a custom level of 0.3. Approximately 5 min durations of HF & RMMSD were calculated using Kubios. LnHF and LnRMMSD were calculated using Microsoft Excel® spreadsheet software (Microsoft Inc. Redmond, WA, USA). HRR<sub>30</sub>, HRR<sub>60</sub> and HRR<sub>120</sub> were calculated using Polar ProTrainer 5®. Scores for NQ (out of 60) (Dixhoorn and Folgering 2015) [Appendix 1] and SEBQ (Courtney and Greenwood 2009) [Appendix 2] were completed at pre- and post-intervention time points.

### *Experimental Procedure*

#### Familiarisation and Incremental Maximal Oxygen Consumption Test:

At the first laboratory session, all participants were familiarised with questionnaires, heart rate recording devices and procedures, and completed a cycle exercise test to exhaustion. The incremental cycle test was performed on an electromagnetically-controlled ergometer (Velotron, RacerMate®, Seattle, U.S.A), during which participants wore a face-mask (Oro-Nasal 7450 V2 Mask, Hans Rudolph, Shawnee, U.S.A) connected to a metabolic cart (Moxus modular V O<sub>2</sub> system AEI Technologies, Pittsburgh, U.S.A) and a heart rate monitor (S810i or RS800x, Polar, Kempele, Finland). Following a 5 min warm-up, the starting power was set at 50 W or 100 W, after which it was incrementally increased by 10, 15, or 20 W/min depending on the age, gender and estimated current level of fitness of each participant. The peak oxygen consumption was calculated as the average of the highest three breath-by-breath values, using three of the six criteria of the British Association for Sports and Exercise Sciences (Bird and Davison 1997) to determine whether a maximal test had been achieved: respiratory exchange ratio (>1.15), final heart rate within 10 beats/min of age-predicted maximum = 220 – age (years) beats/min, and the attainment of volitional exhaustion.

#### Pre- and Post-Intervention Visits:

During subsequent visits, participants underwent a 20 min stationary cycle, consisting of four, 5 min bouts at fixed work rates at 30, 40, 55 and 70% of their oxygen volume ( $V O_2$ ) maximum, whilst maintaining a cadence of 75 revolutions per min, during which HR was recorded. The work rate for each individual at these intensities was determined from the maximal  $V O_2$  and  $V O_2$  to HR linear relationship obtained during the  $V O_2$  max test. Once completed, the ventilation mask was removed, participants rested, sitting on the cycle seat for 5 min, without moving or talking, whilst HRR was recorded.

#### Intervention

At the laboratory session immediately prior to the start of the intervention, the BRT protocol was introduced. This included an assessment of conscious breathing [as per the BRT protocol assessment; Appendix 3 & 4], which was repeated at the post-intervention sessions approximately one week following their final osteopathic consultation. Four, weekly osteopathic consultations occurred, at which participants received MT, and BRT tutorage. All participants underwent six BRT sessions at which they followed a protocol consisting of a series of progressive steps. BRT included breathing education, awareness, instruction, practice (two 10 min daily sessions), positions to challenge breathing, assessment and tips on BRT [Appendix 9]. The initial and final sessions were delivered by JB during pre-intervention visits (the second pre-intervention visit for the delayed start group) and post-intervention visits, the 2<sup>nd</sup> – 5<sup>th</sup> by an osteopath within the first 10 min of the osteopathic consultation. Supplementary online and paper-based information were provided at the initial BRT session [Appendix 8 & 9]. The remainder of the 40 min consultation consisted of MT as prescribed by a MT breathing protocol [Appendix 4]. This protocol guided the practitioner through a semi-standardised assessment, diagnosis and treatment. The assessment incorporated observation, active movements, palpation and passive movements. The diagnosis was based on the presence of either a change in texture, asymmetry, range of motion or tenderness. The treatment prescription gave the practitioner the choice of either soft tissue massage, mobilisation, manipulation or functional osteopathic techniques.

#### *Practitioners*

Participants visited one of eight osteopaths approximately 1 week following their initial BRT session, assigned by geographical convenience and availability. Practitioners were located in the

Auckland region, actively treated breathing, held a current New Zealand annual practicing certificate and attended symposium where a semi-standardised treatment was discussed and agreed [refer Chapter 3 of thesis]. Practitioners each treated between one and three patients. Following each osteopathic visit, practitioners emailed the investigators the BRT progression progress.

### Statistical Analysis

#### *Data Analysis:*

Changes in outcome variables from pre- to post-intervention were checked for violations of a normal distribution. Change in outcome measures following recruitment were compared between those randomised to the immediate or delayed start groups, using 2-way ANOVA. Data from both groups comparing pre- and post-intervention were then combined to allow analysis of a change in outcomes at variables for the cohort, using two-tailed paired t-tests. Exploration of descriptive factors that might have influenced the effect of the intervention was also completed using Pearson's correlation coefficient.

The treatment effect sizes (Cohen's *d*) were calculated by dividing the difference in the pre- to post-intervention means by the standard deviation of the change scores, thus the size of the effects were scaled according to the variability in within-participant response (Wu et al. 2011). Magnitudes of these effects and of correlations were described according to Hopkins' scale (Hopkins 2006). Statistical analyses were performed using SPSS® version 18.0 (SPSS Inc. Chicago, IL, USA). Level of statistical significance was set at  $\alpha < 0.05$ .

## RESULTS

### Participant Analysed Data

Of 34 screened individuals, 19 completed this study with sufficient data for analysis (Figure 4.1). Characteristics are shown in Table 4.2.

All 19 participants completed at least 6 weeks of BRT, receiving six BRT sessions over an average 7-week period (SD = 1.2). Over an average period of 4 weeks (SD = 0.8) all participants also underwent four 40 min MT treatments, except two who received only three treatments. Compliance for participants providing morning heart rate measures was 6.1 (1.6) out of 7 days.

Inspection of data revealed differences at pre-intervention that occurred by chance despite random allocation. Pre-intervention HRV measures for the immediate start group were substantially lower than for the delayed start group scores for LnRMSSD: IS = 3.77 Ln ms (0.67), DS = 4.63 Ln ms (0.15); and LnHF: IS = 6.44 Ln ms<sup>2</sup> (1.20), DS = 7.99 Ln ms<sup>2</sup> (0.37). The immediate start group's breathing questionnaire scores were also higher (NQ = 21(8), SEBQ = 30(17)) than delayed start group's (NQ = 16(7), SEBQ = 26(14)). A subset of 7 participants recorded breathing symptoms scores above 23, proposed as a threshold for DB (Dixhoorn and Folgering 2015).

Table 4.2. Pre-Intervention Characteristics.

	Immediate start, <i>n</i> = 10	Delayed start, <i>n</i> = 9	Cohort, <i>n</i> = 19
Age (years)	29.7 (7.5)	26.0 (5.3)	27.9 (6.7)
Height (cm)	173 (8.4)	170.0 (10.9)	171.9 (9.6)
Weight (kg)	74.8 (8.7)	68.4 (14.5)	71.8 (11.9)
NQ cohort scores	21.0 (7.9)	15.8 (7.4)	18.4 (7.9)
SEBQ cohort scores	29.8 (17.2)	26.0 (13.9)	26.6 (16.3)
BRT progression	6.4 (4.5)	6.6 (3.4)	6.5 (3.9)
NQ (>23) (n)	3	4	7
SEBQ (NQ>23) (n)	3	3	6

Data are mean (SD) except Nijmegen Questionnaire (NQ) and Self-Evaluation of Breathing Questionnaire (SEBQ) above 23, which are participant numbers, n. Breathing retraining (BRT) progression denotes the step in the protocol that participants achieved at study onset.

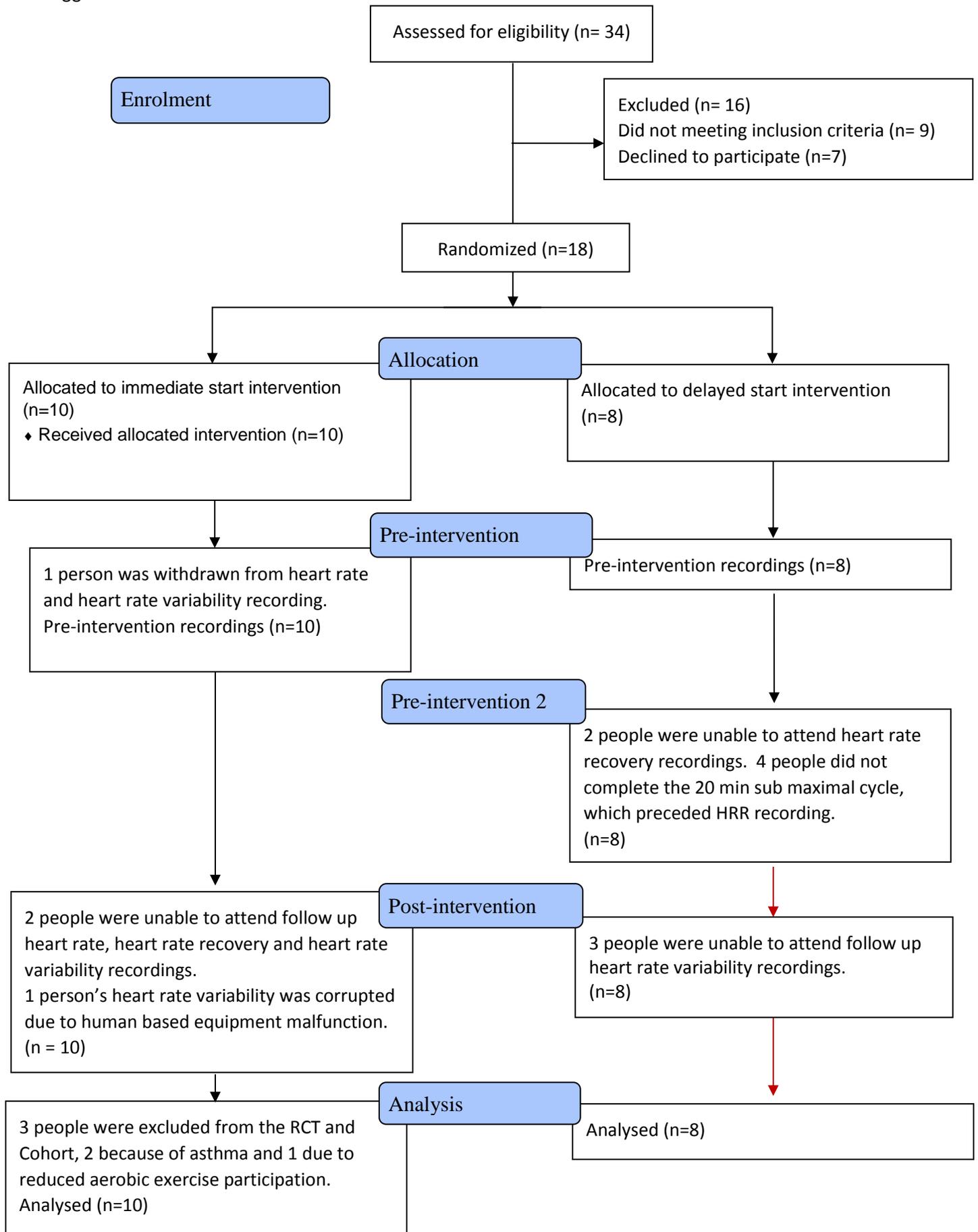


Figure 4.1. Consort Diagram.

### Change in Primary Outcomes: LnRMSSD and LnHF:

No primary outcome (HRV) change variables violated Shapiro-Wilk or Kolmogorov-Smirnov tests of normality, although the change in secondary outcome SEBQ variables violated Shapiro-Wilk test. Parametric statistical analyses have been applied throughout. When changes in HRV variables were compared between the immediate start group, who received the intervention, and the delayed start group, who did not, LnRMSSD and LnHF both increased more from pre- to post-intervention in the immediate start group: 0.27 (95% CI: 0.09 to 0.45) Ln ms, and 0.30 (-0.04 to 0.70) Ln ms<sup>2</sup>; compared to changes in the delayed start group prior to receiving the intervention: 0.09 (-0.26 to 0.08) Ln ms; and -0.19 (-0.52 to 0.15) Ln ms<sup>2</sup>; LnRMSSD ( $P = 0.01$  for ANOVA time x group interaction); LnHF, ( $P = 0.03$ ); Figure 4.2a and b; Table 4.3.

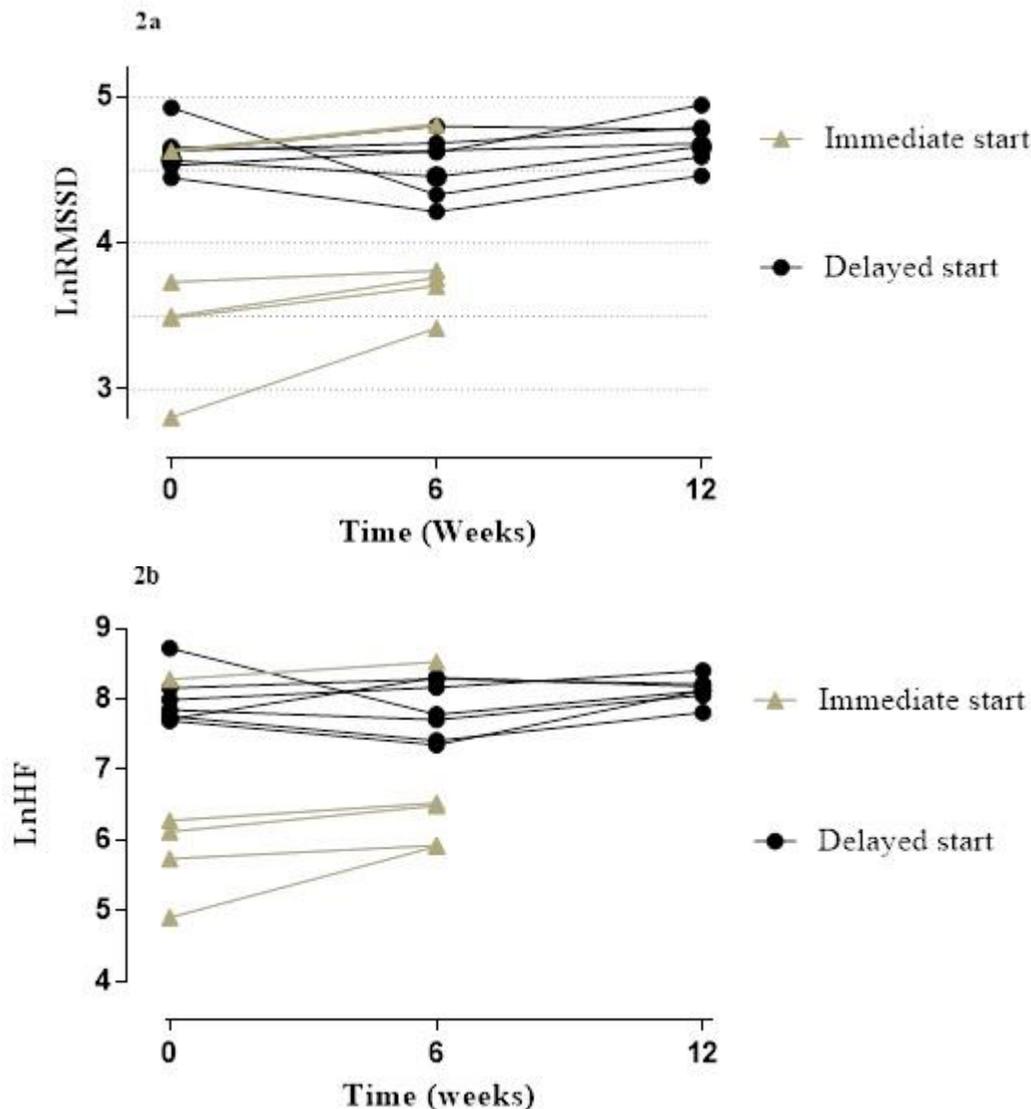


Figure 4.2: Plot of Heart Rate Variability data. 2a. Natural logarithm of, the root mean square of successive differences (LnRMSSD). 2b. Natural logarithm of high frequency (LnHF). For immediate start pre-intervention was at 0-weeks and final 6-weeks. For delayed start, pre-intervention was at 0-weeks, 2<sup>nd</sup> pre-intervention 6-weeks and final at 12-weeks.

Table 4.3: Analysis of Randomised Control Primary and Secondary Outcomes.

<b>Randomised Control</b>		<b>Immediate Start</b>		<b>Delayed Start</b>		<b>P (RCT)</b>
<b>Trial</b>	<b>Measures</b>	<b>Pre</b>	<b>Post</b>	<b>Pre</b>	<b>Pre 2</b>	
Primary:	LnRMSSD	3.77 (3.2–4.3)	3.90 (3.4–4.3)	4.63(4.5–4.7)	4.54 (4.4–4.7)	0.02
	LnHF	6.44 (5.5–7.4)	6.68 (5.7–7.6)	7.99(7.7–8.3)	7.86 (7.6–8.2)	0.03
Secondary:	HR	61.6 (52–70 )	56.6 (49–64)	53.6 (51–56)	54.0 (48–60)	0.09
	HRR 30	24.9 (17–33)	30.3 (22–39)	21.1 (18–24)	21.8 (19–24)	0.1
	HRR60	44.1 (32–56)	48.3 (37–60)	41.4 (37–46)	43.5 (34–53)	0.8
	HRR 120	60.9 (41–80)	62.8 (45–81)	62.6 (54–71)	62.0 (49–75)	0.8
	NQ	21 (16–26)	17 (11–23)	16 (11–21)	13 (11–15)	0.2
	SEBQ	30 (19–41)	23 (14–31)	26 (17–35)	15 (13–18)	0.5

Heart rate (HR). Natural logarithm of, the root mean square of successive differences (LnRMSSD). Natural logarithm of high frequency (LnHF). Heart rate recovery at 30, 60 & 120 seconds (HRR<sub>30</sub>, HRR<sub>60</sub> and HRR<sub>120</sub>). Nijmegen questionnaire (NQ). Self-evaluating breathing questionnaire (SEBQ). Breathing retraining (BRT) progression. Immediate start (IS). Delayed start (DS).

For the cohort analysis, using the average of the two pre-intervention values for the delayed start group, HRV variables reduced from pre- to post-intervention (Table 4.4;  $P = 0.003 - 0.005$ ).

More conservative analysis applying non-parametric equivalents did not alter the statistical significance of any of the between-group or cohort effects for these variables.

Table 4.4: Analysis of Cohort Primary and Secondary Outcomes.

Cohort						
Outcomes	Measures	Pre	Post	Change	<i>P</i> (Cohort)	Effect Size
Primary:	LnRMSSD	4.21 (3.9–4.5)	4.37 (4.1–4.8)	0.18 (0.1–0.3)	0.003	1.1
	LnHF	7.24 (6.6–7.8)	7.53 (7.0–8.1)	0.29 (0.1–0.5)	0.005	1.0
Secondary:	HR	57.4 (53–62)	54.8 (51–59)	-3.0 (-5–1)	0.02	-0.9
	HRR 30	23.6 (19–28)	29.2 (23–35)	7.5 (1–14)	0.08	0.7
	HRR60	43.2 (38–51)	47.9 (40–56)	7 (-2–17)	0.3	0.4
	HRR 120	62.4 (52–73)	64.8 (51–79)	7 (-7–21)	0.9	0.3
	NQ	18 (15–22)	15 (12–19)	-4 (-9–0)	1.0	-0.5
	SEBQ	28 (21–34)	19 (14 – 24)	-9 (-15–3)	0.017	-0.7
	BRT	6 (4–8)	21.9 (5.6)	14 (11–18)	<0.001	2.5
	NQ>23	27 (24 – 30)	17 (11 – 23)	10 (5 – 15)	0.01	1.5
	SEBQ (NQ>23)	43 (34 – 53)	20 (7 – 34)	23 (17 – 29)	<0.001	3.3

Heart rate (HR). Natural logarithm of, the root mean square of successive differences (LnRMSSD). Natural logarithm of high frequency (LnHF). Heart rate recovery at 30, 60 & 120 seconds (HRR<sub>30</sub>, HRR<sub>60</sub> and HRR<sub>120</sub>). Nijmegen questionnaire (NQ). Self-evaluating breathing questionnaire (SEBQ). Breathing retraining (BRT) progression. Immediate start (IS). Delayed start (DS).

### Change in Secondary Outcomes

No differences between the changes from pre- to post-intervention for any secondary outcomes (breathing symptom questionnaire scores, resting HR, and HRR measures) attained statistical significance when analysed against the control.

When results from the immediate start and delayed start groups were pooled, pre- to post-intervention reductions were observed for SEBQ (non-significant with non-parametric analysis) ( $P = 0.04$ ) and resting HR ( $P = 0.02$ ) Table 4.4. In order to determine whether those who had dysfunctional breathing had improved their symptoms, as assessed by NQ and SEBQ, we repeated the analysis for those who scored above 23 at pre-intervention for NQ. A reduction in breathing questionnaires scores was observed for dysfunctional breathers: NQ, 10.0 (5 to 15) ( $P < 0.007$ ), SEBQ, 23 (17 to 29) ( $P < 0.001$ ). All participants improved their BRT progression

scores following the intervention, by a minimum of 9 steps, with an average change of 14.33 (11 to 18) ( $P < 0.001$ ). No correlation was observed between low scores breathing questionnaire scores and cardiac autonomic measures at pre-intervention and post-intervention.

### Correlation

At baseline, correlations were observed between LnRMSSD and the cardiac autonomic measures LnHF and HF,  $r = 0.9$  ( $P < 0.001$ ),  $r = 0.6$ , ( $P = 0.008$ ), respectively, however not with the HRR measures. Intercorrelations between cardiac autonomic variables and their change are reported in Table 4.5. A change in LnRMSSD correlated to a change in the autonomic measures LnHF, HRR<sub>30</sub>, HRR<sub>60</sub> and resting HR (Table 4.5.  $P < 0.01$ ). In order to determine whether a relationship was present between breathing questionnaires and the BRT protocol, the change in values were also analysed. Correlations were observed between baseline NQ and SEBQ scores ( $r = 0.7$   $P = 0.001$ ,  $N = 18$ ), and change in NQ and SEBQ scores ( $r = 0.7$ ,  $P = 0.001$ ,  $N = 18$ ). The relationship between cardiac autonomic measures and breathing parameters was also analysed. No correlations were observed between any of the cardiac autonomic LnRMSSD, LnHF, HR, HRR<sub>30</sub>, HRR<sub>60</sub> and HRR<sub>120</sub> and the breathing measures NQ, SEBQ and BRT progression.

Table 4.5: Correlation of Cardiac-Autonomic Measures.

		LnRMSSD $\Delta$	LnHF $\Delta$	HR $\Delta$	HRR30 $\Delta$
LnHF $\Delta$	Correlation				
	Coefficient	.80**			
	Sig.(2-tailed)	<0.001			
	N	15			
HR $\Delta$	Correlation				
	Coefficient	.70**	.79**		
	Sig.(2-tailed)	0.004	0.005		
	N	15	15		
HRR30 $\Delta$	Correlation				
	Coefficient	.73**	.46	.26	
	Sig.(2-tailed)	.007	.1	.4	
	N	12	12	13	
HRR60 $\Delta$	Correlation				
	Coefficient	.61*	.41	-.49	.22
	Sig.(2-tailed)	0.04	0.2	0.1	0.5
	N	12	12	13	14

A comparison of change ( $\Delta$ ) in final compared with averaged pre-intervention 1 and 2 indicia, heart rate (HR), log of the root mean square of the standard deviation (LnRMSSD, log of high frequency (LnHF) and heart rate recovery at 30, 60 & 120 seconds (HRR<sub>30</sub>, HRR<sub>60</sub> and HRR<sub>120</sub>).

## DISCUSSION

To our knowledge, this is the first controlled study to examine the effects of a combined intervention of BRT progression and MT on measures of cardiac autonomic function or on any outcomes during spontaneous breathing. Following this intervention, we report a 3 to 4% improvement, effect size of 1.1 to 1.2, in LnRMSSD and LnHF compared to a no-intervention control condition, in healthy active individuals, during spontaneous breathing. Secondly, our findings show some evidence of improvements in resting HR and self-reported breathing symptoms following the intervention, though not compared to control. In contrast to some previous work, we have also noted correlations between changes in measures of cardiac autonomic function: LnRMSSD, LnHF, HRR<sub>30</sub>, HRR<sub>60</sub> and HR.

### Effects of the Intervention on Cardiac Autonomic Variables

Several previous studies have recorded HRV indices during acute breathing manipulation (controlled breathing), reporting that a slow breathing frequency and deep breathing enhance vagal activity (Kulur et al. 2009; Lin et al. 2014; Scolnick et al. 2014; Wang et al. 2010). It appears only one RCT study investigates the long term effect of BRT on HRV during spontaneous breathing (Tharion et al. 2012). The present study supports these results, showing that BRT combined with MT is capable of increasing HRV in healthy individuals. The BRT intervention implemented by Tharion et al. (2012) comprised 1 month of 30 min daily deep slow breathing. Tharion et al. (2012) reported an improvement in frequency domain (HF) but not in the time domain measure RMSSD, compared to a no-intervention control. In contrast, here we show improvements in natural logarithms of both HF and RMSSD against control.

The improvements seen in both time and frequency domain measures are likely due to the method of data recording employed by this current study. Assessing improvements in HRV following changes in habitual breathing are difficult, since both breathing rate and depth are known determinants of HRV measures, through the effects of sinus arrhythmia. Some literature in the area suggests that breathing should therefore be controlled during HRV recordings (Heathers 2014; Quintana and Heathers 2014). Although frequency domain HRV measures are strongly altered by breath frequency and depth, recently developed quantification methods allow valid recording of HRV during spontaneous breathing. The time domain measure RMSSD (Penttilä et al. 2001; Saboul et al. 2013), reported here and in the Tharion et al. (2012) study are

less affected by breath (Penttilä et al. 2001; Saboul et al. 2013), with matching day-to-day variations observed for RMSSD during controlled and spontaneous breathing (Saboul et al. 2013). In order to further reduce RMSSD variation in the current study we applied two further steps. The first was determining an average of multiple waking HRV recordings, which has been previously shown to smooth out day-to-day RMSSD variations (Heathers 2014; Pinna et al. 2007; Plews et al. 2013). The second was in calculation of log transformations of RMSSD. Correspondingly, multiple-day averages of LnRMSSD recordings are the preferred cardiac PSNS measure during spontaneous breathing (Buchheit 2014; Esco and Flatt 2014; Heathers 2014; Pinna et al. 2007).

Whilst the specific cause(s) of HRV improvements reported in this study are unclear, they possibly arise from two mechanisms. Firstly, participants may have adopted elements of the retrained breath during spontaneous breathing, such as a slower breathing frequency, a deeper breath or a longer inhalation: exhalation ratio, all known to increase HRV (Ben-Tal et al. 2014). However, cardiac autonomic indices have been observed to improve following BRT, during controlled breathing (Kulur et al. 2009), suggesting that autonomic adaptations are not exclusively caused by a change in breathing frequency, depth or inhalation: exhalation ratio (Tharion et al. 2012). Nonetheless, controlling breathing does not eliminate the possible neuroplastic alterations in respiratory function caused following BRT, which may facilitate alterations in autonomic activity (Cramer et al. 2011). Secondly, MT and BRT may have initiated peripheral stimuli that instigated a central command modification towards an increased HRV (Bialosky et al. 2009), where future stressors generate a reduced sympathetic, or improved parasympathetic, response.

The effect of manual therapy on autonomic activity has been poorly investigated in the past. Often studies measuring HRV do not effectively control for all effectors of the ANS, for example digestion and excretion are infrequently controlled (Heathers 2014). Not surprisingly the literature surrounding MT's effects on the ANS is highly varied, and furthermore, often produce changes in the favour of sympathetic excitation (Kingston et al. 2014), rather than the more desirable parasympathetic alterations. Whilst acute SNS excitation may be unproblematic, chronic sympathetic excitation is associated with negative health states, for example renal and cardiac failure (Joles and Koomans 2004; Rajendra Acharya et al. 2006). Furthermore, the

patient's tissue response to MT varies between individuals (Bronfort et al. 2010). In the present study, MT was semi-standardised, which allowed for a balance between the pragmatic aspects of typical MT treatments that were individually tailored to the participant, and a more strictly controlled intervention protocol. Hence, it is highly possible that differences in response to the intervention may have been affected by differences between individuals. We speculate that autonomic changes predominantly arose from the effects of BRT, with BRT utilising and benefiting from neurophysiological changes generated through MT.

Changes in HRR indices, unlike HRV indices, did not significantly improve against control or amongst the entire cohort. This lack of response could arise from differing experience of cycling between participants and the inconsistent physiological fatigue occurring at pre-set percentage of maximum  $\text{VO}_2$  during cycling. Higher exercise intensities are shown to lengthen HRR times (Buchheit et al. 2007; Haddad et al. 2011; Mann et al. 2014). Notably, some participants here struggled to maintain 70% of  $\text{VO}_2$  max for 5 min. Several participants did not complete 5 min at 70% and instead inadvertently performed a maximum exertion test. Additionally, recording only single HRR measures following each exercise test, may have subjected HRR to greater ANS variation. Averaging of same day successive HRR recordings does not sufficiently improve validity of these measures (Dupuy et al. 2012). In future, averaging HRR recordings measured over a period of days, may increase the validity of HRR data sets.

#### Inter-Relationships between Cardiac Autonomic Measures

The autonomic measures  $\text{HRR}_{30}$ ,  $\text{HRR}_{60}$  and  $\text{HRR}_{120}$  have previously been reported to correlate poorly with  $\text{LnRMSSD}$  and  $\text{LnHF}$  (Esco et al. 2010a), even though they are all assumed to be measures of PSNS activity. Daily autonomic fluctuations are believed to prevent accurate representation of their inter-relationship (Dupuy et al. 2012). We analysed logarithms of waking HRV recordings averaged over several days (Heathers 2014; Pinna et al. 2007; Plews et al. 2013), which may explain that here we report higher correlations (of range) than previously. Previous studies such as Esco et al. (2010b), reported no cross-sectional correlations between another HRV measure, standard deviations of N-N intervals ( $\text{SDNN}$ ), and  $\text{HRR}_{60}$  and  $\text{HRR}_{120}$  measures from a single time-point. This study also observed no cross-sectional correlation between HRV and HRR measures at baseline. The observed correlation of the change in cardiac

autonomic measures, here, tend to confirm the assertion that HR, LnRMSSD, LnHF, HRR<sub>30</sub> and HRR<sub>60</sub> are measuring components of the same system, potentially PSNS (vagal) activity.

#### Effect on Breathing Symptoms Questionnaire Scores

The effect of a combined treatment comprised of MT and BRT has recently been reported to improve NQ (Jones et al. 2015) in adults with breathing abnormalities. Jones et al. (2015) revealed an average reduction of NQ score of 12.6 (9.0) following interventions of BRT combined with MT groups. In the current study decreases in NQ were similar, 10 (6.6) for all participants who scored above 23 on the NQ; where 23 is a suggested threshold for DB (Dixhoorn and Folgering 2015). Although this change was not shown to be significant when compared against control, this is likely to be due to inadequate statistical power due to the smaller sample size of the subgroup of dysfunctional breathers.

Seasonal changes are known to affect respiratory health (Braga et al. 2002; De Sario et al. 2013) and exercise participation (Alahmari et al. 2015; Belanger et al. 2009). Autonomic changes are also known to be affected by seasonal variations and alterations to exercise regimes (Heathers 2014), with a reduction of HF observed in autumn and winter, compared to summer and spring (Kaisina et al. 2005). The current study was performed across three seasons (summer to winter), which might have influenced respiratory health and subsequent breathing symptoms questionnaires and cardiac autonomic indices. In fact, delayed start group participants showed deterioration in HRV in the 6-week control period, prior to intervention, suggesting that the external environment factors might have influenced participants' autonomic activity during this period. On the contrary, for the same time period, the immediate start group who received intervention at this time, saw an improvement in cardiac autonomic indices, despite the environmental conditions.

The changes in NQ scores reported by Jones et al. (2015) are believed to predominantly originate from BRT, as no significant difference in changes in breathing symptoms were reported between BRT alone compared with BRT combined with MT. However, in Jones et al. (2015) study, the possibility that MT altered cardiac autonomic activity, cannot be ruled out. It is therefore problematic to conclude that the change seen in this present study occurred specifically due to BRT, or MT, as likely it was the combination of the two. A similar study to Jones et al. (2015) is

required to investigate if BRT alone is as effective as the combined treatment of BRT and MT, at improving the cardiac autonomic measures LnRMSSD and LnHF.

#### LIMITATIONS:

Several limitations impact upon conclusions drawn from this study. Firstly, number of participants recruited may have been a little small to detect the between group differences for the secondary outcomes: breathing symptoms questionnaire scores and HRR measures. Secondly, HRR measures were recorded in the laboratory, and were subject to more missing data than HR and HRV data points, which were recorded at participants' own homes.

## CONCLUSION

In summary this study revealed that an intervention comprising a BRT protocol, combined with semi-standardised MT sessions increased HRV indices, LnRMSSD and LnHF. Breathing symptom questionnaire scores improved in those who scored greater than 23 in the NQ, however because they did not differ significant from the control group it is difficult to positively attribute this change to the intervention. Our results imply that an improvement in autonomic activity, characterised by increased parasympathetic drive was brought about by this combined breathing retraining and manual therapy intervention.

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## APPENDICES:

### Appendix 1: The Nijmegen Questionnaire.

#### The Nijmegen Questionnaire

The Nijmegen questionnaire gives a broad view of symptoms associated with dysfunctional breathing patterns. It is only a preliminary guide to breathing training.

Please ring the score that best describes the frequency with which you experienced the symptoms listed

Symptom	Never	Seldom	Some-times	Often	Very often
Chest pain	0	1	2	3	4
Blurred vision	0	1	2	3	4
Dizziness	0	1	2	3	4
Confusion or loss of touch with reality	0	1	2	3	4
Fast or deep breathing	0	1	2	3	4
Shortness of breath	0	1	2	3	4
Tightness across chest	0	1	2	3	4
Bloated sensation in stomach	0	1	2	3	4
Tingling in fingers and hands	0	1	2	3	4
Difficulty breathing or taking deep breaths	0	1	2	3	4
Stiffness or cramps in fingers and hands	0	1	2	3	4
Tightness around the mouth	0	1	2	3	4
Cold hands or feet	0	1	2	3	4
Palpitations in the chest	0	1	2	3	4
Anxiety	0	1	2	3	4
<b>Totals</b>					

**Grand Total Score**

A grand total score of over 20 indicates significant hyperventilation.  
 A grand total score of between 10 and 20 suggests mild hyperventilation.  
 If your score is under 10 your breathing may not be causing you any serious health problems. However with any score over zero you should do the other checks on your breathing.

## Appendix 2:

<u>Self Evaluation of Breathing Questionnaire</u>				
	0 Never or not true at all	1 occasionally a bit true	2 frequently mostly true	3 very frequently – very true
I get easily breathless on physical exertion out of proportion to my fitness	0	1	2	3
I get breathless even when resting	0	1	2	3
I get breathless when I am anxious	0	1	2	3
I get short of breath or very tired when reading out loud or talking a lot	0	1	2	3
I feel breathlessness in association with other physical symptoms	0	1	2	3
I feel that the air is stuffy, as if there is not enough air in the room.	0	1	2	3
I feel I cannot get a deep or satisfying breath	0	1	2	3
I can't catch my breath	0	1	2	3
My breathing feels stuck, restricted	0	1	2	3
I Feel that my ribcage is tight and can't expand.	0	1	2	3
My clothing often feels too tight or uncomfortable around my chest.	0	1	2	3
I sigh, yawn or gasp.	0	1	2	3
I find myself holding my breath at various times	0	1	2	3
I notice myself breathing shallowly using my upper chest and shoulders.	0	1	2	3
I notice myself breathing quickly.	0	1	2	3
I notice myself mouth breathing	0	1	2	3
I have trouble co-ordinating my breathing when I am speaking	0	1	2	3
I notice myself breathing irregularly.	0	1	2	3

Appendix 3:

# Breathing Re-training Protocol

Thank you for your interest in our breathing study.

We have been busy developing the breathing re-training protocol, making a poster and videos to go with the breathing guide for patients.

I am keen to arrange a time and date to explain the breathing guide to you in person and answer any questions you might have.

I have attached the information that will be given to the patient (see attached). Note - They will receive the initial training, when I first meet them for their first data collection (before they see you).

Please read the following and familiarize yourself with the breathing guide attached.

Prior to arrival at your clinic, your patients will undergo a breathing assessment, followed by hands-on breathing re-training and will take home a video-directed breathing guide.

Your role is to re-assess your patient at the beginning of every clinic session, to determine their progression through the breathing guide, then relay this information back to us.

The breathing re-training is divided into 4 stages, from Stage 1: relearning to breathe using the diaphragm, and through the nose, before the introduction of various body positions to increase work load (Stages 2 & 3), to functional movements (Stage 4). Note -Each of the stages is further considered using a series of step wise progressions.

Participants will commence at Stage 1's first step (nasal breathing awareness), or one step prior to their best step achieved during their initial assessment.

Once a patient has satisfied all the steps within a stage they may progress to the next stage and receive the YouTube tutorial link for the following stage - once a step is achieved it does not need to be re-tested. Therefore, could you please email us after each session stating the stage and steps the patient achieved.

Progress through the breathing re-training will vary between participants. One patient may progress through all stages of the protocol, yet another may only reach Stage 2, which is fine by us. When assessing the patient's breath, a 'pass' is determined by performing the steps for 6 consecutive breathing cycles (1 inspiration + 1 expiration = 1 breathing cycle) – See relevant video for the expected step performance. If a patient 'fails' a step, can you please give them feedback and coaching for them to take home.

For step 8 and onwards, patients are required to maintain the skills shown at step 7 throughout the positions listed.

Patients would not achieve the step if they failed to pass the above in any way, for example by breathing via the upper rib cage or mouth.

## Breathing Re-Training Protocol:

Developed by Jordan Benjamin, Rob Moran and Wesley Verhoeff.

This guide is to be used concurrently with the YouTube videos (which will be emailed to you and found below). Please practice each step for at least 10 minutes, twice daily. Once you are comfortable achieving a step, you can move onto the next step. The first stage retrains normal breathing, stages 2 onwards challenge your normal breath (learnt in stage one), placing you in positions, which are progressively difficult to maintain a normal breath.

### Stage 1

#### Step 1: Nasal passage awareness

- Feel the air flow in through the nose into the back of the throat.
- Feel the warm air coming out through the nasal passage and over the lip.
- This can be aided by pursing the upper lip or placing a finger on top of the lip.
- Maintain awareness of breathing; keep thinking about the air flowing through your nose.

#### Step 2: Hi-lo awareness

- Place one hand on your chest the other over your abdomen (in between your ribs and navel), and feel which hand rises the most.

#### Step 3: Nasal breathing with abdominal movement

- The aim here is to breathe through your nose and ensure your abdomen rises during your inspiratory (“in”) breath and falls during your expiratory (“out”) breath. Your upper chest should stay motionless during this procedure.
- Awareness of motion can be aided by using a towel wrapped around the abdomen, belt wrapped around the abdomen, or weight upon the abdomen to give abdominal breathing feedback.
- Blow a balloon up 1/4 full. Keep it in your mouth as seen on the video. If you use your mouth to breathe, the balloon will deflate or inflate – ensure the balloon size does not change.

#### Step 4: Breath length

- The length of your out breath should be longer than your in breath
- Comfortably alter your breath to match this ratio, stay relaxed and change it when ready.

#### Step 5: Breath pause

- After the end of each out breath pause for a second or two, then inhale.

#### Step 6: Evenness of breath

Ensure that each breath is as deep/shallow as all the previous and that each breath’s duration is the same.

Note that the Steps 3 to 6 are the characteristics features of a ‘good breath’. To help make this memorable we use the acronym “**N.O.P.E**”, where **N** = Nasal breathing, **O** = Out longer than in, **P** = pause at end of our breath, **E** = evenness of duration.

## Stage 1

### Steps:

Step 1 – Step A = Nasal passage awareness

Step 2 – Step B = Hi-Lo awareness

Step 3 – Step C = Nasal breathing with abdominal movement

Step 4 – Step D = ‘Out’ breath is longer than inspiration

Step 5 – Step E = **P**ause

Step 6 – Step F = **E**venness

Step 7 – Steps C, D, E and F. (Nasal, Out, Pause, Evenness = “**N.O.P.E**”)

Breathing in through the nose, into the abdomen, then out through the nose. With an out breath longer than the in breath and a pause after the out breath. Each breath is then repeated for the same duration and consistency, without the use of mouth breathing and upper chest movement.

Step 8 – N.O.P.E while lying on your back with your knees bent.

Step 9 – N.O.P.E while lying on your back, legs straight.

Step 10 – N.O.P.E while seated.

## Stage 2

### Steps:

Step 11 – N.O.P.E while seated with hands on head.

Step 12 – N.O.P.E while standing.

Step 13 – N.O.P.E while lying on your back with your hands over head.

Step 14 – N.O.P.E while seated and slumped forward.

Step 15 – N.O.P.E while lying on your front.

## Stage 3

### Steps:

Step 16 – N.O.P.E while standing and bending down to touch your toes.

Step 17 – N.O.P.E while standing with hands overhead.

Step 18 – N.O.P.E while walking.

Step 19 – N.O.P.E while in child pose.

Step 20 – N.O.P.E while standing, hands over head and leaning back.

Step 21 – N.O.P.E while in a squat (Progression).

Step 22 – N.O.P.E while walking with arms above head.

## Stage 4

### Steps:

Step 23 – Standing with weight progression.

Step 24 – Standing up progression.

Step 25 – Supine hands over head progression.

Step 26 – Plank Progression.

Step 27 – Walking with weight progression.

### When Exercising – adapted from Rachel Vickery’s thesis

- Try to keep breathing into your stomach through your nose for as long as possible. At some point you may revert to breathing through your mouth, when you do try to keep breathing into your stomach. At some point you may revert to breathing through to your upper chest but try to delay this as long as possible.
- When you breathe in, think of “sipping” the air in over your bottom lip. As you breathe out, blow the air out over your bottom lip as if blowing out a straw. Allow your out breath to become more forceful as exercise intensity increases.
- Try to keep your mouth opening as small as possible, for as long as possible.

#### Note:

*The ability to perform the above in order will be determined by:*

1. *Soft tissue apposition – positions that restrict diaphragm movement, such as with abdominal flexion positions.*
2. *Anterior fascial chain tightness – such as during thoracic extension positions.*

*Due to the variability of soft tissue apposition and anterior fascial chain tension found from person to person, not all consecutive Steps may seem as if they are increasing in respiratory demand; as intended.*

#### YouTube Links:

Intro <http://youtu.be/Lx-DjJCt-bU>

1. <http://youtu.be/Lx-DjJCt-bU>
2. <http://youtu.be/Sn41al0qCZg>
3. <http://youtu.be/ykKsyz4W6Gg>
4. <http://youtu.be/nyj2bINwfBk>
5. <http://youtu.be/nyj2bINwfBk>
6. <http://youtu.be/0ZOcF2RaIpE>
7. <http://youtu.be/c0RnGFcE2Ps>

Explanation <http://youtu.be/HKH6jcbEPrC>

8. <http://youtu.be/005BYr04-mw>
9. <http://youtu.be/8Hd73qlpiGM>
10. <http://youtu.be/lH68lbgDNM>

For questions about the breathing protocol, please contact Jayden Beginheim:

For any further questions about the study, please contact the research supervisor Dr. Catherine Bacon

## STAGE 1 Breathing Retraining

### Step 1: Nasal passage awareness

Feel the air flow in through the nose into the back of the throat.  
 Feel the warm air coming out through the nasal passage and over the lip.  
 This can be aided by pursing the upper lip or placing a finger on top of the lip.  
 Maintain awareness of breathing; keep thinking about the air flowing through your nose.



### Step 2: Hi-Lo awareness

Place one hand on your chest the other over your abdomen (in between your ribs and navel), and feel which hand rises the most.



### Step 3: Nasal breathing with abdominal movement

The aim here is to breathe through your nose and ensure your abdomen rises during your inspiratory ("in") breath and falls during your expiratory ("out") breath. Your upper chest should stay motionless during this procedure.  
 Awareness of motion can be aided by using a towel wrapped around the abdomen, belt wrapped around the abdomen, or weight upon the abdomen to give abdominal breathing feedback. Blow a balloon up 1/4 full. Keep it in your mouth as seen on the video. If you use your mouth to breathe, the balloon will deflate or inflate – ensure the balloon size does not change.

### Step 4: 'Out' breath longer than in breath.

The length of your out breath should be longer than your breath in breath. Comfortably alter your breath, stay relaxed, change it when ready.

### Step 5: Pause after out breath

After the end of each out breath pause for a second or two, then inhale.

### Step 6: Evenness of breath

Ensure that each breath is as deep/shallow as all the previous and that each breath's duration is the same.

### Step 7: Steps C, D, E and F. (Nasal, Out, Pause, Evenness = "N.O.P.E")

Note that the Steps 3 to 6 are the characteristics features of a 'good breath'. To help make this memorable we use the acronym "N.O.P.E", where **N** = Nasal breathing, **O** = Out longer than in, **P** = pause at end of our breath, **E** = evenness of duration.



### Step 8: N.O.P.E while lying on your back with your knees bent.



### Step 9: N.O.P.E while lying on your back, legs straight.



### Step 10: N.O.P.E while seated.



Nasal  
 Out  
 Pause  
 Evenness

Step 1: Breathing Re-Training Quick Reference, Version 1.1  
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## Breathing Re-Training Quick Reference

### STAGE 1

Step 1: Nasal passage awareness



Step 2: Hi-Lo awareness



Step 3: Nasal breathing with abdominal movement



Step 4: 'Out' breath longer than 'In' breath.

Step 5: Pause after out breath

Step 6: Evenness of breath

Step 7: Steps C, D, E and F. (Nasal, Out, Pause, Evenness = "N.O.P.E")

Step 8: N.O.P.E while lying on your back with your knees bent.



Step 9: N.O.P.E while lying on your back, legs straight.



Step 10: N.O.P.E while seated.



### STAGE 2

Step 11: N.O.P.E while seated with hands on head.



Step 12: N.O.P.E while standing.



Step 13: N.O.P.E while lying on your back with your hands over head.



Step 14: N.O.P.E while seated and slumped forward.



Step 15: N.O.P.E while lying on your front.



### STAGE 3

Step 16: N.O.P.E while standing and bending down to touch your toes.



Step 17: N.O.P.E while standing with hands overhead.



Step 18: N.O.P.E while walking.



Step 19: N.O.P.E while in child pose.



Step 20: N.O.P.E while standing, hands over head and leaning back.



Step 21: N.O.P.E while in a squat



Step 22: N.O.P.E while walking with arms above head.



## Nasal Out Pause Evenness

### STAGE 4

Step 23: N.O.P.E during standing with weight progression.



Step 24: N.O.P.E during standing up progression.



Step 25: N.O.P.E during supine hands over head progression.



Step 26: N.O.P.E during plank progression.



Step 27: N.O.P.E during walking with weight progression



Breathing Re-Training Quick Reference  
 Version 1.1  
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## Appendix 4:

## OMT Assessment & Treatment Protocol

Patient:

DOB:

Date:

Region to be assessed	Notable Joint/soft tissue of the region	Presence of Dysfunction Y/N? (please circle)	Dysfunction present in? (please circle)	Technique(s) used? (please circle)
Cspine	OA-AA	Y/N T.A.R.T	Observation, Active, Passive, Palpation.	S/T, Inhibition, Myofascial release, M.E.T Manipulation. Mobilisation, Articulation, MWM SCS, Functional; BLT.
Cspine/Tspine	C3-T5	Y/N T.A.R.T	Observation, Active, Passive, Palpation.	S/T, Inhibition, Myofascial release, M.E.T Manipulation. Mobilisation, Articulation, MWM SCS, Functional; BLT.
Tspine	T6-T12	Y/N T.A.R.T	Observation, Active, Passive, Palpation.	S/T, Inhibition, Myofascial release, M.E.T Manipulation. Mobilisation, Articulation, MWM SCS, Functional; BLT.
Upper thoracic & ribs	R1-2	Y/N T.A.R.T	Observation, Active, Passive, Palpation.	S/T, Inhibition, Myofascial release, M.E.T Manipulation. Mobilisation, Articulation, MWM SCS, Functional; BLT.
	AC-CC-SC	Y/N T.A.R.T	Observation, Active, Passive, Palpation.	S/T, Inhibition, Myofascial release, M.E.T Manipulation. Mobilisation, Articulation, MWM SCS, Functional; BLT.
Lower thoracic & ribs	3-12	Y/N T.A.R.T	Observation, Active, Passive, Palpation.	S/T, Inhibition, Myofascial release, M.E.T Manipulation. Mobilisation, Articulation, MWM SCS, Functional; BLT.
	Diaphragm	Y/N T.A.R.T	Observation, Active, Passive, Palpation.	S/T, Inhibition, Myofascial release, M.E.T Manipulation. Mobilisation, Articulation, MWM SCS, Functional; BLT.
Lspine	L1-L5	Y/N T.A.R.T	Observation, Active, Passive, Palpation.	S/T, Inhibition, Myofascial release, M.E.T Manipulation. Mobilisation, Articulation, MWM SCS, Functional; BLT.
Other				

BLT = Balance ligamentous technique, BMT = Balance muscle technique, M.E.T = Muscle energy technique, MWM = Mobilisation with movement, SCS = Strain counter strain, S/T = Soft tissue manipulation, T.A.R.T = Texture, Asymmetry, Range of movement & Tenderness

## Appendix 5:

**Breathing Assessment Protocol:**

First, please circle which stage your patient has attained.

## Stage 1

1. *Nasal Awareness*
2. *Hi-Lo awareness*
3. *Nasal with Abdominal movement*
4. *Longer out breath*
5. *Pause*
6. *Evenness*
7. *N.O.P.E*      *Supine,*
8. *knees bent*
9. *Supine legs straight*
10. *Seated*

## Stage 2

11. *Seated HOH*
12. *Standing*
13. *Supine HOH*
14. *Seated slumped*
15. *Prone*

## Stage 3

16. *Standing forward flexion*
17. *Standing HOH*
18. *Walking*
19. *Child Pose*
20. *Standing HOH + thoracic extension*
21. *Squat*
22. *Walking HOH*

## Stage 4

23. *Standing with weight*
24. *Standing up progression*
25. *Supine HOH*
26. *Plank progression*
27. *Walking weight progression.*

*HOH = hands above head N.O.P.E = Nasal breathing, Out longer than in, Pause at end of out breath, Evenness of duration*

	Y/N	Notes.
1. Nose breathing?		

2. Abdomen breathing?		
3. Longer exhalation		
4. Pause after exhalation?		
5. Consistent breath rhythm?		
6. Normal breath rate?		
7. Presence of cough, sigh or wheeze?		

*Primarily focus on the following 6 aspects; mouth/nose, abdomen/thorax, length of breath, pause, consistency of breath rhythm, breathing rate and abnormalities.*

*Achieved looks like: 1 Nasal, 2 Abdomen, 3 Abdomen, 4 Exhalation or even, 5 Exhalation, 6 Consistent, 7 Normal, 8 No abnormalities detected.*

Appendix 6:

## Appendix 7:

8/23/2015

General Information Questionnaire

[Edit this form](#)

## General Information Questionnaire

\* Required

Name (Last, First) \*

Date of Birth \*

Are you between the age of 19 and 45 \*

- Yes  
 No

Are you willing and able to give informed written consent (Understand written and verbal English language). \*

- Yes  
 No

Do you have well controlled asthma if asthmatic \*

- Yes  
 No  
 I don't have Asthma

Do you participate in aerobic exercise for at least four hours per week. \*

- Yes  
 No

Do you perceive that breathing may be limiting you sporting performance \*

- Yes  
 No

Do you currently smoke or have a history of smoking in the last 6 months \*

- Yes  
 No

Have you had any respiratory-related hospitalisations in the last 6 months. \*

- Yes  
 No

Do you have any diagnosed emotional disorders or cardiac dysfunctions \*

- Yes  
 No

Do you have any autonomic nervous system pathologies. \*

- Yes  
 No

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Appendix 8

Appendix 9:

## Breathing Retraining Information sheet:

Thank you for participating in our study.

This document contains information regarding the breathing re-training phase of this study and is to be used alongside the YouTube breathing tutorial videos, found on YouTube.

Begin at Stage 1 - Step 1, or at the stage allocated to you during your baseline data collection (the first day of the study). Practice the steps you are working on as often as you can throughout your day, for example: when you wake up, while you are waiting for the kettle to boil, at the traffic lights etc.

This should be done for at least 10 minutes, twice a day or equivalent. We'll ask you to keep a brief record of the number and length of practice sessions that you've been able to maintain.

Your breathing progress will be assessed at each osteopathic consultation and we'll provide you with links to subsequent breathing retraining videos once you have mastered each step.

This information will continue to be available to you once the study finishes, if you wish to continue using them.

## Breathing Re-Training Protocol:

Developed by Jordan Benjamin, Rob Moran and Wesley Verhoeff.

This guide is to be used concurrently with the YouTube videos (which will be emailed to you and found below). Please practice each step for at least 10 minutes, twice daily. Once you are comfortable achieving a step, you can move onto the next step. The first stage retrains normal breathing, stages 2 onwards challenge your normal breath (learnt in stage one), placing you in positions, which are progressively difficult to maintain a normal breath.

### Stage 1

#### Step 1: Nasal passage awareness

- Feel the air flow in through the nose into the back of the throat.
- Feel the warm air coming out through the nasal passage and over the lip.
- This can be aided by pursing the upper lip or placing a finger on top of the lip.
- Maintain awareness of breathing; keep thinking about the air flowing through your nose.

#### Step 2: Hi-lo awareness

- Place one hand on your chest the other over your abdomen (in between your ribs and navel), and feel which hand rises the most.

#### Step 3: Nasal breathing with abdominal movement

- The aim here is to breathe through your nose and ensure your abdomen rises during your inspiratory (“in”) breath and falls during your expiratory (“out”) breath. Your upper chest should stay motionless during this procedure.
- Awareness of motion can be aided by using a towel wrapped around the abdomen, belt wrapped around the abdomen, or weight upon the abdomen to give abdominal breathing feedback.
- Blow a balloon up 1/4 full. Keep it in your mouth as seen on the video. If you use your mouth to breathe, the balloon will deflate or inflate – ensure the balloon size does not change.

#### Step 4: Breath length

- The length of your out breath should be longer than your in breath
- Comfortably alter your breath to match this ratio, stay relaxed and change it when ready.

#### Step 5: Breath pause

- After the end of each out breath pause for a second or two, then inhale.

#### Step 6: Evenness of breath

Ensure that each breath is as deep/shallow as all the previous and that each breath’s duration is the same.

Note that the Steps 3 to 6 are the characteristics features of a ‘good breath’. To help make this memorable we use the acronym “**N.O.P.E**”, where **N** = Nasal breathing, **O** = Out longer than in, **P** = pause at end of our breath, **E** = evenness of duration.

### Stage 1

#### Steps:

Step 1 – Step A = Nasal passage awareness

Step 2 – Step B = Hi-Lo awareness

Step 3 – Step C = Nasal breathing with abdominal movement

Step 4 – Step D = ‘**O**ut’ breath is longer than inspiration

Step 5 – Step E = **P**ause

Step 6 – Step F = **E**venness

Step 7 – Steps C, D, E and F. (Nasal, Out, Pause, Evenness = “**N.O.P.E**”)

Breathing in through the nose, into the abdomen, then out through the nose. With an out breath longer than the in breath and a pause after the out breath. Each breath is then repeated for the same duration and consistency, without the use of mouth breathing and upper chest movement.

Step 8 – N.O.P.E while lying on your back with your knees bent.

Step 9 – N.O.P.E while lying on your back, legs straight.

Step 10 – N.O.P.E while seated.

### Stage 2

#### Steps:

Step 11 – N.O.P.E while seated with hands on head.

Step 12 – N.O.P.E while standing.

Step 13 – N.O.P.E while lying on your back with your hands over head.

Step 14 – N.O.P.E while seated and slumped forward.

Step 15 – N.O.P.E while lying on your front.

### Stage 3

#### Steps:

Step 16 – N.O.P.E while standing and bending down to touch your toes.

Step 17 – N.O.P.E while standing with hands overhead.

Step 18 – N.O.P.E while walking.

Step 19 – N.O.P.E while in child pose.

Step 20 – N.O.P.E while standing, hands over head and leaning back.

Step 21 – N.O.P.E while in a squat (Progression).

Step 22 – N.O.P.E while walking with arms above head.

### Stage 4

#### Steps:

Step 23 – Standing with weight progression.

Step 24 – Standing up progression.

Step 25 – Supine hands over head progression.

Step 26 – Plank Progression.

Step 27 – Walking with weight progression.

### When Exercising – adapted from Rachel Vickery’s thesis

- Try to keep breathing into your stomach through your nose for as long as possible. At some point you may revert to breathing through your mouth, when you do try to keep breathing into your stomach. At some point you may revert to breathing through to your upper chest but try to delay this as long as possible.
- When you breathe in, think of “sipping” the air in over your bottom lip. As you breathe out, blow the air out over your bottom lip as if blowing out a straw. Allow your out breath to become more forceful as exercise intensity increases.
- Try to keep your mouth opening as small as possible, for as long as possible.

#### Note:

*The ability to perform the above in order will be determined by:*

3. *Soft tissue apposition – positions that restrict diaphragm movement, such as with abdominal flexion positions.*
4. *Anterior fascial chain tightness – such as during thoracic extension positions.*

*Due to the variability of soft tissue apposition and anterior fascial chain tension found from person to person, not all consecutive Steps may seem as if they are increasing in respiratory demand; as intended.*

#### YouTube Links:

Intro <http://youtu.be/Lx-DjJCt-bU>

11. <http://youtu.be/Lx-DjJCt-bU>

12. <http://youtu.be/Sn41al0qCZg>

13. <http://youtu.be/ykKsy4W6Gg>

14. <http://youtu.be/nyj2bINwFBk>

15. <http://youtu.be/nyj2bINwFBk>

16. <http://youtu.be/0ZOcF2RaIpE>

17. <http://youtu.be/c0RnGFcE2Ps>

Explanation <http://youtu.be/HKH6jcbEPrc>

18. <http://youtu.be/005BYr04-mw>

19. <http://youtu.be/8Hd73qlpiGM>

20. <http://youtu.be/lH68lbgDNM>

For questions about the breathing protocol, please contact Jayden Beginheim:

For any further questions about the study, please contact the research supervisor Dr. Catherine Bacon.

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**Self Evaluation of Breathing Questionnaire**

	0 Never or not true at all	1 occasionally a bit true	2 frequently mostly true	3 very frequently – very true
I get easily breathless on physical exertion out of proportion to my fitness	0	1	2	3
I get breathless even when resting	0	1	2	3
I get breathless when I am anxious	0	1	2	3
I get short of breath or very tired when reading out loud or talking a lot	0	1	2	3
I feel breathlessness in association with other physical symptoms	0	1	2	3
I feel that the air is stuffy, as if there is not enough air in the room.	0	1	2	3
I feel I cannot get a deep or satisfying breath	0	1	2	3
I can't catch my breath	0	1	2	3
My breathing feels stuck, restricted	0	1	2	3
I Feel that my ribcage is tight and can't expand.	0	1	2	3
My clothing often feels too tight or uncomfortable around my chest.	0	1	2	3
I sigh, yawn or gasp.	0	1	2	3
I find myself holding my breath at various times	0	1	2	3
I notice myself breathing shallowly using my upper chest and shoulders.	0	1	2	3
I notice myself breathing quickly.	0	1	2	3
I notice myself mouth breathing	0	1	2	3
I have trouble co-ordinating my breathing when I am speaking	0	1	2	3
I notice myself breathing irregularly.	0	1	2	3



**Declaration**

Name of candidate: Jordan Benjamin

This Thesis/Dissertation/Research Project entitled: A Novel Breathing Retraining & Osteopathic Manual Therapy Intervention, and its effect on cardiac Autonomic activity is submitted in partial fulfillment for the requirements for the Unitec degree of & Breathing symptoms

Principal Supervisor: Dr Catherine Bacon

Associate Supervisor/s: Robert Moran

**CANDIDATE'S DECLARATION**

I confirm that:

- This Thesis/Dissertation/Research Project represents my own work;
- The contribution of supervisors and others to this work was consistent with the Unitec Regulations and Policies.
- Research for this work has been conducted in accordance with the Unitec Research Ethics Committee Policy and Procedures, and has fulfilled any requirements set for this project by the Unitec Research Ethics Committee.

Research Ethics Committee Approval Number: .....

Candidate Signature: [Signature] Date: 25/09/2019

Student number: 1232407

Full name of author: Jordan Guy Benjamin

ORCID number (Optional): .....

Full title of thesis/dissertation/research project ('the work'):

A Novel breathing retraining & osteopathic manual therapy intervention, and its effect on cardiac autonomic activity & Breathing symptoms.

School: osteopathy

Degree: MOST

Year of presentation: 2016

Principal Supervisor: Dr Catherine Bacon

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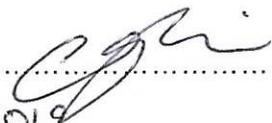
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