

**The Effect of Osteopathic Manual Therapy with Breathing Retraining on  
Dysfunctional Breathing and Exercise Economy: A Randomised Controlled  
Trial**

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## List of Abbreviations

BHT	breath-hold time
BR	breathing rate
BRT	breathing retraining
CO <sub>2</sub>	carbon dioxide
DH	dynamic hyperinflation
DS	delayed start
EFL	expiratory flow limitation
FEV <sub>1</sub>	forced expiratory volume in one second
FVC	forced vital capacity
HR	heart rate
IPFV	isovolume pressure/flow curve
IS	immediate start
MEF	maximal expiratory flow
NEP	negative expiratory pressure
NQ	Nijmegen Questionnaire
O <sub>2</sub>	oxygen
OMT	osteopathic manual therapy
PEF	peak expiratory flow
ROM	range of motion
RPE	rating of perceived exertion
SEBQ	Self-Evaluation of Breathing Questionnaire
VE	minute volume
VO <sub>2</sub>	oxygen uptake
VO <sub>2</sub> max	maximal oxygen uptake
VT	tidal volume
W	watts
WR <sub>max</sub>	work rate max

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## Abstract

**Background:** Dysfunctional breathing patterns during exercise can result in a tachypnoeic breathing pattern, expiratory flow limitation and dynamic hyperinflation. These factors can increase the work and metabolic cost of breathing, decreasing exercise economy and potentially performance. Breathing pattern retraining and osteopathic manual therapy aim to optimize breathing mechanics by decreasing the work of breathing. To date no studies have investigated the effects of breathing pattern retraining and osteopathic manual therapy on exercise economy in trained individuals. **Purpose:** The aim of the present study was to investigate the effect of breathing retraining and osteopathic manual therapy on exercise economy during a 20-minute submaximal cycling bout. Secondary aims of this study included investigating the effect of the intervention on lung function and dysfunctional breathing symptoms and behaviours at rest, and on breathing rate, perceived exertion and heart rate during exercise. **Method:** Fourteen healthy trained individuals were randomly assigned to either an immediate start group (received intervention immediately, N = 8) or delayed start group (received intervention after a six-week delay, N = 6). Oxygen consumption ( $VO_2$ ), Borg rating of perceived exertion, breathing rate and heart rate response during 20 minutes incremental cycling, with steady state bouts performed at 30, 40, 55 and 70% of  $VO_2$  maximum, were assessed before and after the intervention. Both groups underwent six weeks of standardised breathing pattern retraining and semi-standardised osteopathic manual therapy aimed at optimising respiratory mechanics. **Results:** Analysis of the entire cohort (both groups) showed a significant reduction in  $VO_2$  from pre- to post-intervention at 30% ( $1370 \pm SD 414$  vs  $1271 \pm 307$ ), 40% ( $1762 \pm 503$  vs  $1681 \pm 393$ ), 55% ( $2505 \pm 637$  vs  $2322 \pm 508$ ) and 70%  $VO_2$  maximum ( $3053 \pm 1130$  vs  $2370 \pm 1397$ ). Additionally, breathing rate at each exercise intensity decreased following intervention (30%,  $22.1 \pm 3.97$  vs  $18.5 \pm 5.23$ ; 40%,  $21.9 \pm 3.35$  vs  $18.5 \pm 5.54$ ; 55%,  $25.4 \pm 3.53$  vs  $22.9 \pm 4.28$ ; 70%,  $30.5 \pm 10.59$  vs  $25.7 \pm 11.71$ ) and heart rate was also lower at each intensity, but failed to reach statistical significance. No significant changes were observed in Borg Ratings of Perceived Exertion at 9, 14 or 19 minutes into the protocol, nor in resting SEBQ, NQ, PFR, FEV1, FVC or BHT. **Conclusion:** These results provide evidence that optimising respiratory mechanics using breathing retraining and osteopathic manual therapy can decrease the metabolic demand of exercise at set submaximal intensities. Additionally, these results suggest that breathing rate can be optimised to exhibit greater efficiency.

**Key words:** Breathing dysfunction, breathing pattern disorders, metabolic demand, osteopathy, manipulation, respiratory mechanics, lung function.

## Chapter One: Introduction

Factors influencing endurance sporting performance have been studied extensively, investigating what sets the top athletes apart from the rest of the field. Research suggests superior exercise economy, or the ratio between oxygen uptake and power output, is associated with higher levels of performance in varying sporting codes (Coyle, 1994; López et al., 1993; Romer, Lovering, Haverkamp, Pegelow, & Dempsey, 2006). In elite endurance sport, where most athletes have a high maximal oxygen uptake (VO<sub>2</sub> max) and can sustain working at a high percentage of their VO<sub>2</sub> max for a prolonged period of time, small variations in economy can have significant effects on an athlete's performance (Costill, 2001; Stickford, Johnston, & Stager, 2015).

Due to the relationship between oxygen uptake, power output and aerobic performance, the effects of varying levels of atmospheric oxygen levels (Bonetti & Hopkins, 2009; Engelen et al., 1996; Haseler, Hogan, & Richardson, 1999; Hermand, Pichon, Lhuissier, & Richalet, 2015; Hermand et al., 2015) and loading and unloading the work of breathing (Babb, 2001; Babcock, Pegelow, Harms, & Dempsey, 2002; Borghi-Silva et al., 2008; Carra et al., 2003; Harms et al., 1997; Häussermann et al., 2015; Louvaris et al., 2012; Mancini, Donchez, & Levine, 1997; Salvadego et al., 2015; Stark-Leyva, Beck, & Johnson, 2004; Wüthrich, Notter, & Spengler, 2013) on oxygen uptake and exercise performance have been studied extensively. The latter studies show promising results of respiratory muscle loading and unloading on aerobic performance in pathological conditions such as chronic obstructive pulmonary disorders (Häussermann et al., 2015; Louvaris et al., 2012), congestive heart failure (Borghi-Silva et al., 2008; Mancini et al., 1997), as well as in older (Babb, 2001) and obese participants (Salvadego et al., 2015). Despite these results achieved by unloading via proportional assist ventilation and low-density heliox gas, there is a paucity of studies exploring the effect of other methods to decrease the work of breathing and their effect on exercise economy and performance.

Adaptation to prolonged inappropriate breathing mechanics may bring about dysfunctional breathing patterns (Chaitow, Gilbert, & Bradley, 2014). Breathing

mechanics can become inappropriate due to:

- i) Psychological factors, such as anxiety, social or work stress;
- ii) Biochemical factors such as allergies, drugs, altitude or normal breathing frequency and tidal volume responses to exercise;
- iii) Biomechanical factors such as chronic mouth breathing, posture and abnormal breathing patterns;
- iv) Pathological factors such as asthma or diabetic ketoacidosis.

(Chaitow et al., 2014)

Development of a chronic breathing pattern disorder can arise from any aforementioned factor, or combination thereof. The impact of these inappropriate and inefficient breathing mechanics on movement economy in athletes during exercise, and whether this can be altered with a breathing retraining intervention is of particular interest to this study.

Dysfunctional breathing patterns can be detrimental to an athlete during exercise due to a number of factors, particularly those influencing components of the oxygen supply chain and overall oxygen availability (Hanon, Dorel, Delfour-Peyrethon, Lepêtre, Bishop, Perrey & Thomas, 2013). Inappropriate breathing mechanics during exercise may lead to tachypnoea, an increase in breathing rate to maintain minute ventilation and a reduced inspiratory reserve; adding to the work of breathing (Chaitow et al., 2014; Rosalba Courtney, 2009). These changes can cause impaired oxygen perfusion at the alveolar capillaries, respiratory muscle fatigue, lowered pain threshold and diversion of blood from muscles of locomotion to respiratory muscles to meet elevated metabolic demands, a phenomenon called 'blood stealing' (Chaitow, 2007; Chaitow et al., 2014; Davies, 1995; Ritz, von Leupoldt, & Dahme, 2006); all of which may have a negative effect on exercise economy.

Traditionally, very little regard has been given to the possible limiting effects of breathing on aerobic exercise at sea level, due to the ventilatory system's apparent ability to cope with increased metabolic demand during activity (Fairbairn, Coutts, Pardy, & McKenzie, 1991). However, the notion that expiratory flow limitation and respiratory muscle fatigue are relatively common in endurance-trained athletes, this has more recently implicated breathing as a possible performance-limiting factor

(Guenette & Sheel, 2007; Plowman & Smith, 2014; Spengler, Roos, Laube, & Boutellier, 1999). Relative success of breathing manipulation methods such as intermittent hypoxic training (De Meersman, 1993) and various respiratory muscle-training techniques (Chatham, Baldwin, Griffiths, Summers, & Enright, 1999; Markov, Spengler, Knoèppli-Lenzin, Stuessi, & Boutellier, 2001; Spengler et al., 1999) in improving endurance performance indicates specific intervention strategies can be utilised to overcome limiting factors within the respiratory system.

Despite compelling evidence of a decrease in respiratory muscle fatigue and an increase in respiratory muscle endurance when employed (Guenette & Sheel, 2007), these techniques do not address the pattern of breathing.

Manipulating an athletes breathing pattern using manual therapy and breathing retraining is a relatively unexplored area of research. As there is little literature defining dysfunctional breathing patterns and the effectiveness of a breathing retraining intervention, there is currently little to no evidence supporting or refuting the application of breathing retraining as a method of improving economy in athletic populations. Vickery (2008) addressed this paucity of literature with a compelling study demonstrating a significant increase in 20km time trial performance and incremental power in competitive male cyclists following a 4-week breathing retraining intervention. Vickery's breathing retraining intervention specifically aimed to reduce dynamic hyperinflation and optimize breathing mechanics. This key paper highlighted the dramatic effect a relatively short breathing retraining protocol could have on the performance of a well-trained population, guiding the current research to determine if these effects would be enhanced with the addition of osteopathic manual therapy.

Osteopathic manual therapy (OMT) has commonly been used as a complementary therapy to standard medical protocols in cases of respiratory diseases such as whooping cough, asthma, chronic obstructive pulmonary disorders, respiratory infections and pneumonia, all with considerable benefit to patients (Chaitow et al., 2014; Gosling & Williams, 2004). In these cases OMT is used to encourage biomechanical improvements such as increased rib cage and thoracoabdominal diaphragm compliance to improve respiratory mechanics in addition to arterial, venous and lymphatic circulation (Chaitow et al., 2014). Despite OMT being used in the management of pathology, the concepts underlying its application to sub-clinical breathing dysfunction in athletes is similar and may also be beneficial to this

population.

Although osteopaths commonly address breathing pattern disorders in a clinical setting, there are no known published studies citing the possible benefit of these techniques in an active population. Furthermore, there are very few studies exploring the performance effect of optimizing respiratory mechanics by decreasing the metabolic demand of breathing, outside of breathing helium enriched oxygen and proportional assisted ventilation. This gap in literature has led to this investigation into the effect of OMT and breathing retraining on dysfunctional breathing and exercise economy.

The aim of this study is to determine the effect of six weeks of osteopathic manual therapy and breathing pattern retraining on exercise economy during a 20-minute sub-maximal cycling bout in trained individuals. The secondary aims of this study are to determine the effect of the intervention on resting variables of lung function and dysfunctional breathing symptoms and behaviours and on breathing rate, perceived exertion and heart rate during exercise.

The aim of Chapter Two (Review of Literature) is to introduce the concept and consequences of dysfunctional breathing patterns in athletes, to discuss how addressing breathing patterns in this population may improve the efficiency of breathing and therefore potentially improve sporting performance.

Chapter Three (Methods) outlines the design of the study investigating the effect of the intervention on a sample of active adults who perceive breathing to be limiting their performance. Chapter Four (Results) discusses the findings of the trial and Chapter Five (Discussion) addresses the possible interpretations, implications and applications of these results.

## Chapter Two: Review of Literature

The term respiration is used to describe the process of ventilation of the lungs (breathing), the exchange of gases between air and blood and between blood and tissue fluid, and the use of oxygen in cellular metabolism (Saladin, 2007). The primary role of respiration is to maintain homeostatic regulation of oxygen and carbon dioxide levels, according to changing demands of tissue metabolism, allowing normal function of the brain, organs and other tissues of the body (Chaitow et al., 2014; Mateika & Duffin, 1995). Regulation of gases and biomechanics of ventilation can influence widespread homeostatic functions including the autonomic nervous system, the circulatory system, chemical regulation, digestion and metabolism (Chaitow et al., 2014; Courtney, 2009; Saladin, 2007). The ventilation component of respiration also plays a key role in posture and spinal stabilization (Perri & Halford, 2004), as well as preserving spinal mobility through regular thoracic cage movement produced by breathing (Chaitow et al., 2014). During exercise, there is increased demand on the cardiovascular and respiratory system to meet the elevated metabolic demand and maintain homeostasis (Arena, Myers, & Guazzi, 2008; Costill, 2001; Olive, Slade, Dudley, & McCully, 2003; Saladin, 2007).

This review of literature will consist of three main sections; a discussion of breathing mechanics at rest and during exercise, how these can become dysfunctional and what effect this may have and a discussion of what strategies have been employed to influence abnormal breathing patterns to date; a discussion of exercise economy, followed by factors that can limit exercise and how this relates to abnormal breathing mechanics. The first section is largely narrative, giving insight into the physiology and pathophysiology of respiration, useful background knowledge for the subsequent sections involving more critical analysis of literature.

## **2.1. Breathing mechanics**

### **2.1.1. Normal breathing mechanics at rest**

Breathing relies on constant changes in pressure gradients within the abdomen and thorax in order for it to occur. This is achieved through mechanical expansion and contraction driven primarily by the diaphragm and intercostal muscles (Chaitow et al., 2014; Davies, 1995). As this is the principle role of the diaphragm and intercostals, these are considered the primary muscles of respiration, with the diaphragm responsible for around 75% of air movement during relaxed breathing (Caruana-Montaldo, Gleeson, & Zwillich, 2000).

During quiet inspiration (breathing at rest), the diaphragm contracts and flattens, causing lateral expansion of the lower ribs and consequently an increase in thoracic volume. This is inversely proportionate to reductions in pleural pressure (Perri & Halford, 2004). The resulting gradient of ambient pressure and intra-thoracic pressure causes air to pass from the nasal cavity or mouth through the airways to the alveoli of the lungs (Rosalba Courtney, 2009; Davies, 1995; Saladin, 2007). The sternocleidomastoid, levator scapulae, external intercostals and scalenes are also actively involved in the process of inhalation as secondary or 'accessory' muscles of respiration by increasing the anterior-posterior expansion of the upper ribs, an involvement that increases with respiratory demand (Chaitow et al., 2014; Davies, 1995) (Table 1). The internal and external intercostal muscles also aid breathing by stiffening the thoracic cage, preventing it from caving inward when the diaphragm descends and thoracic pressure is decreased (Saladin, 2007). Some literature argues that the scalenes are also primary muscles of respiration as slight activation occurs with light breathing (Perri & Halford, 2004); however, the scalenes only elevate the ribs during forced inspiration and are also actively involved in cervical movement (Saladin, 2007; Stone & Stone, 2011).

**Table 1. Muscles of Respiration (Chaitow et al., 2014)**

<b>Muscle of Inspiration</b>	<b>Muscles of Expiration</b>
<i>Primary</i>	<i>Primary</i>
Diaphragm	Elastic recoil of lungs, diaphragm, pleura and costal cartilages
Intercartilaginous Internal intercostals	
Upper and more lateral external intercostals	
Levatores costarum	
Scalenes	
 <i>Accessory</i>	 <i>Accessory</i>
Sternocleidomastoid	Interosseous internal intercostals
Upper trapezius	Abdominal muscles (rectus abdominus, internal and external obliques, transverse abdominus)
Serratus anterior (arms elevated)	Transversus thoracis
Latissimus dorsi (arms elevated)	Subcostales
Serratus posterior superior	Iliocostalis lumborum
Iliocostalis	Quadratus lumborum
Subclavius	Serratus posterior inferior
Omoxyoid	Latissimus dorsi

Contrary to inhalation, only relaxation of respiratory muscles is necessary for quiet expiration, allowing the elastic recoil of the costal cartilages, ligaments attaching ribs to the spine, the central tendon of the diaphragm and the elastic tissue in the bronchi and bronchioles to expel the contained air (Saladin, 2007). This is facilitated by the increase of thoracic pressure and compression of the lungs associated with cephalic diaphragmatic movement upon relaxation, allowing air to flow down its pressure gradient, out of the lungs (Davies, 1995; Saladin, 2007). During exhalation, neural stimulus of the diaphragm continues at a reduced level, its eccentric contraction preventing the lungs from recoiling too rapidly and allowing a smooth transition from inspiration to expiration (Saladin, 2007). At the end of relaxed expiration, a pause should occur as the diaphragm is released from negative and positive pressures acting on it during breathing (Chaitow et al., 2014).

For normal quiet breathing to occur the osseous and soft tissue structures of the thorax must be in a compliant, elastic, functional state; with any mechanical restrictions impairing the ability of the rib cage to adapt appropriately in response to muscular activity and altered pressure gradients during the breathing cycle. This loss in thoracic compliance can lead to compensatory mechanics occurring at the expense of optimal and efficient respiratory function, which would ultimately increase energy demands (Chaitow et al., 2014).

During quiet or relaxed breathing, between 10 and 14 breath cycles per minute, involving an inspiration to expiration ratio of 1:1.5 – 2, is considered 'optimal' (Chaitow et al., 2014). However, increased respiratory demand in response to physical or psychological stimulus can cause a fluctuation in the rate and depth of breathing (Chaitow et al., 2014; Costanzo, 2013). This change in breathing pattern should return to relaxed lower chest breathing, which requires the least amount of mechanical stress and work from respiratory musculature, upon cessation of the stimulus (Chaitow et al., 2014; Jones, Dean, & Chow, 2003). However, in some cases this 'optimal' breathing pattern can become disrupted and abnormal, and these inefficient breathing mechanics can potentially be adopted as the default pattern (Vickery, 2008).

### **2.1.2. Gas exchange**

At the cellular level, oxygen diffuses from the alveoli through the respiratory membrane into the surrounding capillaries whilst carbon dioxide, a gaseous metabolic by-product of respiration diffuses from the blood stream into the alveoli for exhalation (Davies, 1995). Here, oxygen can only diffuse into the blood stream and carbon dioxide into the alveoli, as each gas diffuses down its own pressure gradient (Saladin, 2007).

### **2.1.3. Regulation of breathing**

Breathing is controlled at two levels of the brain, subconscious and voluntary (Saladin, 2007). This allows inhalation and exhalation to occur at will, however, most of the time this occurs unconsciously. Breathing requires a central co-ordinating mechanism, as it is a complicated action requiring well-orchestrated actions of a number of muscles (Mateika & Duffin, 1995; Saladin, 2007).

### **2.1.4. Abnormal breathing at rest**

Signs of dysfunctional breathing have been noted as early as the beginning of the 19<sup>th</sup> century, with reports of unexplained symptoms akin to heart disease prevalent in American Civil War soldiers, most of whom were aged between 16 – 25 years

(Da Costa, 1871). Many years later it was proposed the unexplained dyspnea, chest pain, fatigue upon exertion, sweating and palpitations could be ascribed to hyperventilation, breathing in excess of metabolic demands, and the resultant hypocapnia and respiratory alkalosis (CliftonSmith & Rowley, 2011).

Hyperventilation has until recently been the focus of research into breathing pattern disorders, however, contention has arisen about whether hyperventilation causes all dysfunctional-breathing-associated symptoms, and it is more recently becoming accepted as a variation of breathing pattern disorder (Barker & Everard, 2015; Courtney, van Dixhoorn, Greenwood, & Anthonissen, 2011). Due to the vague and varying nature of the condition, dysfunctional breathing is difficult to define, however the currently accepted paradigm is 'an alteration in the normal biomechanical patterns of breathing that result in intermittent or chronic symptoms which may be respiratory and/or non-respiratory' (Barker & Everard, 2015, pg. 54). For diagnosis, clinicians rely on a suggestive group of symptoms, however the intermittent and variable intensity of these, in addition to patients failing to mention symptoms due to embarrassment or thinking they are unrelated, makes diagnosis incredibly difficult (Chaitow et al., 2014). Chaitow et al. (2002) suggest that abnormal respiratory patterns in a healthy human at rest include:

- i) Breathing to the upper chest (apical breathing)
- ii) Breathing frequency of greater than 16 breaths per minute
- iii) Inspiratory to expiratory ratio of greater than 1.5:1
- iv) Chronic mouth breathing
- v) Absence of an end-expiratory pause

Respiratory mechanics can become impaired and or distorted in various disease states such as kyphoscoliosis, neuromuscular disease, obesity, emphysema and asthma.

Sometimes, this change in function can be an appropriate response to increased ventilatory or metabolic needs of the disease state, and cannot thus be regarded as dysfunctional. However, inappropriate habits of muscle use may be retained after the psychological, physiological or environmental conditions that initiated their development have passed, leading to the emergence of a breathing pattern disorder (Chaitow et al., 2014; Courtney, 2009).

### **2.1.5. Diagnosis and prevalence of dysfunctional breathing**

It is near impossible to gauge the true prevalence of breathing dysfunction. Since diagnostic parameters remain undefined and ambiguity in the use of the term exists, controversial ideas remain among clinicians and specialists (Barker & Everard, 2015; Courtney & Greenwood, 2009). Difficulty with determining diagnostic criteria arises because the condition is often diagnosed on the basis of the presence of symptoms with no apparent pathological cause. There is also difficulty because these characteristic symptoms are common to many other diseases and can be intermittent in occurrence (Barker & Everard, 2015; Thomas, McKinley, Freeman, & Foy, 2001). Thomas et al. (2001) also suggest that the incidence of dysfunctional breathing may be underestimated when its symptoms are attributed to other causes, resulting in false diagnosis and inappropriate treatment. The same study suggests these symptoms of hyperventilation, without objective hypocapnia are likely mediated by an ulterior mechanism.

Perri and Halford (2004) define normal respiratory motor function to create their own simplified quantitative system of dysfunctional breathing diagnosis. In their system, diagnosis was made on lack of abdominal breathing, faulty upper rib expansion, faulty lower rib expansion and movement, presence of clavicular grooves and paradoxical breathing. These were then rated from one to three respectively, based on severity of findings, to give an indication of the degree of dysfunction, with the higher number indicating more severe symptoms (Perri & Halford, 2004). The broad, non-specific parameters used in this study resulted in a staggeringly high proportion of dysfunctional breathers. Of 94 participants who fully participated in the study, 56.4% were reported to display dysfunctional breathing mechanics with relaxed breathing, increasing to 75% with deep inhalation (Perri & Halford, 2004). This large proportion may likely be due to an extremely low threshold for participants to be considered faulty breathers, as diagnosis of dysfunctional breathing was made on the presence of any of the aforementioned factors. In a later study, Stanton et al, (2008) report a positive diagnosis of dysfunctional breathing in 29% of the sample who were all asthmatics receiving primary care. In this particular study, diagnosis was based on a positive scoring on the Nijmegen Questionnaire. Although this method of diagnosis has a relatively high specificity (94%) and sensitivity (89%) in detecting hyperventilation when validated against the Hyperventilation Provocation

Test (Van Dixhoorn & Duivenvoorden, 1985), it may have missed dysfunctional breathing patterns not resulting directly from hyperventilation. It has since been suggested that the Nijmegen Questionnaire is more appropriate to be used as a continuous variable rather than a definitive diagnosis of dysfunctional breathing (Thomas, Bruton, & Ainsworth, 2015). However, Courtney (2009) supports Stanton's (2008) findings, proposing that dysfunctional breathing is present in around 5 – 11% of the general population, 30% of asthmatics and up to 83% of anxiety sufferers (Courtney, 2009). This highlights the precipitating effect certain disorders can have on dysfunctional breathing, whilst also indicating the disparity in the estimation of prevalence with differing diagnostic criteria.

A strong theme that appears in each related study is the higher incidence of breathing dysfunction in women (Chaitow, 2007; Courtney et al., 2011; Perri & Halford, 2004; Thomas et al., 2001). Chaitow (2007) and Slatkovska et al (2006) attribute this prevalence to the menstrual cycle where following ovulation, increased progesterone causes an increased breathing rate, which can cause CO<sub>2</sub> levels to drop on average 25 percent resulting in hypocapnia.

### **2.1.6. Aetiology of dysfunctional breathing**

Literature proposes that biomechanical inadequacies, such as poor posture, rib head fixation, muscle imbalance and chronic mouth breathing; and biochemical factors involving disruption of the body's delicate pH homeostasis, may be key aetiological factors of breathing dysfunction (Table 2) (Chaitow et al., 2014; CliftonSmith & Rowley, 2011; Perri & Halford, 2004). Despite the biomechanical and physiological links being a relatively contemporary idea, early literature has long accepted the notion of psychological components of dysfunctional breathing connecting symptoms with anxiety and emotional distress (CliftonSmith & Rowley, 2011). This is further clarified by Chaitow, (2007) who describes a decreased diaphragm function and increase of upper rib breathing pattern evolving from a sympathetic nervous drive due to emotional stress. CliftonSmith and Rowley (2011) support this, alluding to a loss of thoracic cage compliance with the resultant overuse of accessory breathing muscles.

When relaxed diaphragmatic breathing is replaced by accessory muscles driving upper chest breathing, this is commonly associated with an irregular rate and volume of respiration, mild hyperinflation, frequent sighing and in some cases, an increase in breath rate sufficient to induce hypocapnea due to hyperventilation (Barker & Everard, 2015).

**Table 2. Aetiological breathing pattern disorder factors (from Chaitow et al., 2014)**

Psychological factors	Physiological & Biochemical factors	Biomechanical factors	Other factors
Anxiety	Allergies	Postural maladaptations	Undiagnosed organic causes, including mild/asymptomatic asthma, interstitial lung disease, pulmonary hypertension, diabetic ketoacidosis, left ventricular failure, post-nasal drip and rhinitis
Social or work stress	Diet	Chronic mouth breathing	
Panic disorders	Exaggerated hyperventilatory response to decreased CO <sub>2</sub>	Cultural, for example, 'belly in, chest out', tight-waisted clothing	
Personality traits, including perfectionism, high achiever, obsessive	Drugs, including recreational drugs, caffeine, aspirin, alcohol, opiates	Congenital deformities	
Suppressed emotions, for example anger	Hormonal, including progesterone	Overuse, misuse or abuse of musculoskeletal system	
Conditioning/learnt response	Exercise	Abnormal movement patterns	
Action projection/anticipation	Chronic low grade fever	Braced posture, for example, postoperative	
History of abuse	Heat	Occupational, for example, divers, singers, swimmers, dancers, musicians	
Mental tasks involving sustained concentration	Humidity		
Sustained boredom	Altitude		
Pain			
Phobic avoidance			
Misattribution of symptoms			

### 2.1.7. Strategies used to influence dysfunctional breathing

Breathing retraining is considered the first line treatment for adults with dysfunctional breathing (Thomas et al., 2003). Although there are several methods of breathing retraining used by many different modalities and practitioners, the aim is generally to enable patients to modify their breathing pattern with the ultimate goal of restoring and maintaining a normal diaphragmatic breathing pattern (Barker & Everard, 2015; Bott et al., 2009; Han, Stegen, De Valck, Clement, & Van de Woestijne, 1996; Kraft & Hoogduin, 1984; Ram, Holloway, & Jones, 2003). Most breathing retraining protocols also appear to have similar basic principles in common such as:

- Education and reassurance
  - Reattribution of symptoms
  - Self-observation of one's own breathing pattern with awareness of one's primary region of movement during breathing
  - Restoration to a basic physiological breathing pattern – relaxed, rhythmical, nasal, abdominal breathing
  - Appropriate depth and rate of breathing
- (Barker & Everard, 2015; Bruton & Thomas, 2011; CliftonSmith & Rowley, 2011; Courtney, 2009; Hazlett-Stevens & Craske, 2009; Thomas, McKinley, Freeman, & Foy, 2001)

Breathing retraining has been extensively studied in clinical settings, with investigations into the effect of breathing pattern exercises on subjects with tetraplegia (Per Bodin, Olsen, Bake, & Kreuter, 2005; Bodin, Kreuter, Bake, & Olsén, 2003), spinal cord injury (Brown, DiMarco, Hoit, & Garshick, 2006), allergic rhinitis (Corrêa & Bérzin, 2007; Resnick, Bielory, & Bielory, 2008), cystic fibrosis (McIlwaine, 2007), alecstasis (Ciesla, 1996), pneumonia (Graham, Bradley, Kleczek, & Bartlett, 1978), hypertension (Viskoper et al., 2003), post-operative following coronary artery bypass (Westerdahl et al., 2005), chronic obstructive pulmonary disease (Bradstreet & Parkman, 2016; Casciari et al., 1981; Dechman & Wilson, 2004; Nosedá, Carpioux, Vandeput, Prigogine, & Schmerber, 1986; Roberts, Schreuder, Watson, & Stern, 2016) and asthma (Bowler, Green, Mitchell, & others, 1998; Cooper et al., 2003; Cowie, Conley, Underwood, & Reader, 2008; Grammatopoulou et al., 2011; Holloway & West, 2007; Karam, Kaur, & Baptist, 2016; McHugh, Aitcheson, Duncan, &

Houghton, 2003; Opat, Cohen, Bailey, & Abramson, 2000; Slader et al., 2006; Thomas et al., 2003; Thomas et al., 2009). The effect of breathing retraining on asthma has been studied extensively. Breathing exercises studied include Buteyko, yoga techniques, respiratory muscle training and biofeedback training. Burgess (2011) sums up these studies in an elegant review of both randomized and non-randomized trials, therefore these won't be covered in detail in this review (Burgess et al., 2011). Cumulative results suggest that breathing retraining techniques all showed some benefit as an alternative treatment for asthma by increasing asthma related quality of life (Opat et al., 2000; Thomas et al., 2003), decreasing dosage of beta-2 agonist medication (Bowler et al., 1998; McHugh et al., 2003; Opat et al., 2000; Slader et al., 2006), decreasing symptom scores (Cooper et al., 2003; Holloway & West, 2007; Thomas et al., 2003) and a decreased respiratory rate (Ceugniet, Cauchefer, & Gallego, 1996; Grammatopoulou et al., 2011). However, a number of these studies were poorly designed, without adequate statistical power and length of follow-up to allow definitive conclusions to be drawn.

Despite the number of studies investigating breathing modifications in pathological conditions, there is a paucity of studies addressing the effect of breathing retraining on breathing pattern in normal healthy individuals. However, breathing modification and its effects have been extensively studied in relation to exercise performance, this will be discussed at length later in this review. Most of the studies investigating breathing pattern interventions in healthy individuals, with non-exercise outcomes, specifically address hyperventilation - one pattern of dysfunctional breathing. Han et al (1996) investigated the effect of 2 – 3 months of breathing pattern retraining by a physiotherapist on patients with hyperventilation. Retraining was largely focused on education and reducing hyperventilation by promoting an abdominal breathing pattern and slowing down expiration. Following the intervention Nijmegen Questionnaire scores were significantly decreased and mean values of inspiration and expiration time were increased, in addition to tidal volume (Han et al., 1996), indicating positive influences of breathing retraining on healthy individuals. These findings corroborate with other researchers who have found that breathing retraining in healthy subjects and those with hyperventilation can reduce respiratory symptoms, improve quality of life, decrease end-expiratory lung volume, alter breathing frequency and decrease levels of anxiety and depression (Dhungel, Malhotra, Sarkar, & Prajapati, 2008; Grossman, De Swart, & Defares, 1985; Holloway & West, 2007; Kraft & Hoogduin,

1984; Spahija & Grassino, 1996; Tharion, Samuel, Rajalakshmi, Gnanasenthil, & Subramanian, 2012). Due to the brief duration of these studies and lack of follow-up, it is unable to be ascertained whether or not the described changes are maintained subsequent to the study. However, follow-up breathing retraining studies in chronic obstructive pulmonary disease, asthma and hyperventilation patients, have demonstrated maintenance of improvements 6 – 24 months (Roberts et al., 2016), 3 years (Borge et al., 2015; DeGuire, Gevirtz, Hawkinson, & Dixon, 1996) and 5 years after the initial studies (Hagman, Janson, & Emtner, 2011; Thomas et al., 2003). This indicates the plausibility that changes brought about from a relatively brief intervention may be prolonged.

Osteopathic manual therapy (OMT) has commonly been used as a complementary therapy to standard medical protocols in cases of respiratory diseases such as whooping cough, asthma, chronic obstructive pulmonary disease, respiratory infections and pneumonia, largely with anecdotally-reported considerable benefit to patients (Chaitow et al., 2014). In these cases, OMT is used to encourage biomechanical improvements such as increased rib cage and thoracoabdominal diaphragm compliance to improve respiratory mechanics in addition to arterial, venous and lymphatic circulation (Chaitow et al., 2014; Noll, Johnson, Baer, & Snider, 2009). The use of OMT to influence breathing has been investigated in those with chronic obstructive pulmonary disease with varying results; Miller (1975) found no change in pulmonary function measures. Howell et al. (1975) noted improvements in a non-validated severity of illness score (calculated using blood gases and pulmonary function measures and Noll et al (2009) observed a decrease in forced vital capacity and increase in inspiratory capacity, residual volume and total lung capacity when compared to the control group.

The results of Noll et al (2009) suggest an overall worsening of air trapping and hyperinflation in the elderly COPD patients used for the study. As OMT generally involves a combination of techniques, it is not known what the contribution of each technique is to the final result, for example some may have a beneficial effect, but another may be detrimental. This is particularly relevant to the study by Noll et al. (2009), who used a semi-standardised OMT treatment involving a large number of relevant techniques to influence the mechanics of breathing. Jones et al. (2015) later performed an investigation to find if manual therapy had any additional benefit to

breathing retraining. Sixty participants were allocated to either standard breathing retraining, or breathing retraining with individualised manual therapy. Techniques outlined in the semi-standardised manual therapy protocol were very similar to OMT, however, they were applied by a physiotherapist rather than an osteopath and cannot therefore be referred to as OMT. No significant differences were noted in changes of Nijmegen Questionnaire scores, spirometry, breath hold time or 6-Minute Walk Test between those who received both breathing retraining and manual therapy or those who received breathing retraining alone. Analysis of the cohort (both groups combined) revealed a significant improvement in Nijmegen Questionnaire scores, with reduction in reported symptoms to healthy range in 65% of subjects and improvements in 6-minute walk and breath hold time (Jones et al., 2015). These results suggest that although breathing retraining can have significant beneficial effects on dysfunctional breathing, the contribution of manual therapy may be negligible.

Despite OMT being used in the management of pulmonary pathology, there appears to be only one published study investigating the effect of these techniques in healthy subjects. This particular article cited no immediate change to pulmonary function measures (Ortley et al., 1980), which is contrary to unreported anecdotal evidence. Results of Ortley et al. (1980) may be limited in generalisability as only manipulative techniques were used, which is not typical of more broadly focused OMT. Chaitow et al (2014) recommends that breathing retraining protocols should include relevant individualised strategies aimed at improving the biomechanical, as well as functional status of the structures that are involved in breathing. This recommendation is supported by the findings of Bradley and Esformes (2014), who observed a strong relationship between biomechanical (Hi-Lo assessment method) and biochemical (capnography measurements) patterns of dysfunctional breathing and breathing functionality measures (Functional Movement Screen) in 34 healthy individuals. This indicates that those who had breathing pattern disorders were more likely to score lower on the Functional Movement Screen and therefore have sub-optimal respiratory biomechanics, which could be inhibiting optimal breathing patterns.

### 2.1.8. Respiratory response to exercise

The primary function of the respiratory system during exercise is to alter pulmonary gas exchange to match metabolic requirements to maintain arterial-blood oxygen partial pressure and acid-base homeostasis (Mateika & Duffin, 1995; Romer & Polkey, 2008). This is achieved by keeping the respiratory exchange ratio (the ratio of CO<sub>2</sub> production to O<sub>2</sub> consumption), blood lactate, arterial partial pressure of carbon dioxide and partial pressure of oxygen at or near resting levels, even during strenuous exercise (Witt, McKenzie, Road, & Sheel, 2007; Mateika & Duffin, 1995).

During exercise, there is an immediate demand for additional energy; this demand varies with the type, intensity and duration of the exercise (Costill, 2001; Plowman & Smith, 2014). The body will respond by increasing metabolism (Plowman & Smith, 2014). With adequate circulation and oxygen delivery, all energy requirements during exercise may be supplied by adenosine triphosphates (which provides energy for muscle contraction) generated by aerobic metabolism (Wasserman, Whipp, Koyl, & Beaver, 1973). Therefore, the ability to respond to increased energy demands depends on the availability of oxygen (Arena et al., 2008; Bassett & Howley, 2000; Costill, 2001; Plowman & Smith, 2014; Saladin, 2007). Oxygen availability is addressed with an increase of minute ventilation (VE) (which is the volume of gas ventilated in one minute) to enhance alveolar ventilation, increased cardiac output and increased extraction of oxygen by the muscles (Costill, 2001; Olive et al., 2003).

#### 2.1.8.1. Minute Ventilation

*Minute ventilation = respiratory rate x tidal volume*

Minute ventilation (VE) is the product of respiratory rate and tidal volume (VT), both of which increase with metabolic demand. At rest, VE ranges from 6 – 8 L/min in healthy individuals and can reach up to 120 – 130 L/min during strenuous aerobic activity and above 200 L/min in highly trained athletes (Sheel, 2002). To achieve necessary increases in VE, the normal respiratory response is to increase the depth of breathing (increased VT) and as the workload progresses, by increasing respiratory rate, as VT normally plateaus at 50 – 60% of vital capacity (Pellegrino et al., 1999; Sheel, 2002;

Taguchi, Adachi, Hoshizaki, Oshima, & Kurabayashi, 2015; Vickery, 2008).

VT is increased with exercise as additional respiratory muscle recruitment and activity expands the diameter of the chest by as much as 20% with inspiration, causing a corresponding increase in end inspiratory lung volume (Sheel, 2002; Wüthrich et al., 2013). This expansion is due to upper thoracic extension and upper rib elevation produced by increased activity of the sternocleidomastoid (SCM), scalenes, pectoralis minor and major and serratus anterior (Courtney, 2009; Saladin, 2007). During forced expiration, the lower ribs and sternum are pulled inferiorly by the internal intercostals and rectus abdominis, respectively. This movement reduces chest volume and increases intrathoracic pressure more rapidly than elastic recoil alone, helping to expel air more rapidly and completely to reduce end expiratory lung volume (Plowman & Smith, 2014; Saladin, 2007; Sheel, 2002).

The increase in VT is a result of this increase in end-inspiratory lung volume and decreased end-expiratory lung volume. Accordingly, the increase in respiratory rate is a result of a decrease of inspiratory and expiratory time, although the decrease in inspiratory time is more marked (Sheel, 2002).

#### **2.1.8.2. Oxygen extraction**

At resting conditions, 75% of the oxygen in venous blood remains associated with red blood cells, thus allowing additional oxygen to be extracted in response to decreased pH, increased temperature, partial pressure of oxygen and partial pressure of carbon dioxide gradients, occurring during the commencement of exercise (Costill, 2001; Saladin, 2007). During maximal exercise however, almost all of the available oxygen is extracted from the blood to perfuse active muscles, with the oxygen content of arterial blood falling from 200 mL/O<sub>2</sub>/L<sup>-1</sup> to 20 – 30 mL/O<sub>2</sub>/L<sup>-1</sup> in venous blood draining the maximally working muscles (Cerretelli & Di Prampero, 2011).

### 2.1.8.3. Cardiac Output

*Cardiac output = stroke volume x heart rate*

In healthy individuals under physiological conditions, exercise will cause an increase in sympathetic tone. This causes a decrease in total peripheral resistance and an increase in venous return as well as a rise in heart rate and contractility. Enhanced contractility leads to a larger stroke volume, which together with an elevated heart rate results in an increased cardiac output (McKenna & Riddoch, 2003; Salmasi, 1993). This increase in blood flow will continue until oxygen delivery to the tissues matches demand, then it will plateau (Olive et al., 2003). This plateau typically occurs at moderate intensities (heart rate of 120 – 140 beats/min) with maximal stroke volume being attained, this is maintained during maximal exercise (heart rate of 180 – 190 beats per minute) (McKenna & Riddoch, 2003). Endurance training increases stroke volume and therefore cardiac output during maximal exercise (Bassett & Howley, 2000; McKenna & Riddoch, 2003). This is supported by a number of studies comparing endurance-trained athletes to non-athletes (Bevegård, Holmgren, & Jonsson, 1963; Dempsey, Hanson, & Henderson, 1984; Grimby, Nilsson, & Saltin, 1966; Harms et al., 1998; Levine, Lane, Buckey, Friedman, & Blomqvist, 1991; Pluim, Zwinderman, Laarse, & Wall, 2000). Dempsey et al (1984) suggested this difference in cardiac output may be as great as 20 L/min: 40L/min in trained individuals compared to 25L/min in those untrained (Dempsey, Hanson, & Henderson, 1984).

*VO<sub>2</sub> = cardiac output x arterio-venous difference*

Oxygen consumption is the product of cardiac output and arterio-venous oxygen difference, the former largely explaining the greater oxygen capacity in athletes (Bevegård et al., 1963; Cerretelli & Di Prampero, 2011; Dempsey, Romer, Rodman, Miller, & Smith, 2006; McKenna & Riddoch, 2003). As early as 1966, it was proposed by Grimby et al (1966) that arterio-venous difference might be the main limiting factor for oxygen uptake, with findings showing the arterio-venous oxygen difference was 45ml/L at rest and only increasing to 133ml/L with maximal exercise. This was attributed to a relatively low haemoglobin concentration combined with peripheral

factors. However, more recently it has been suggested that less interpersonal variation exists in maximal heart rate and systemic oxygen extraction, indicating that stroke volume and therefore cardiac output might be a more important limiting factor (Cerretelli & Di Prampero, 2011).

## **2.2. Exercise economy**

Exercise economy is defined as the oxygen uptake ( $VO_2$ ) required at a given submaximal exercise intensity (Jones & Carter, 2000). The rate of oxygen consumption indicates the underlying aerobic muscle activity and provides a measure of energy cost for a given steady-state submaximal exercise intensity, with lower  $VO_2$  values, upon repeated testing, reflecting greater efficiency (Dawes et al., 2003). Previously,  $VO_2$  max was considered to be an important predictor of endurance performance (Burgess & Lambert, 2010), this is reflected in a large number of studies investigating performance related outcome variables by using a  $VO_2$  max protocol (Buller & Poole-Wilson, 1990; Burdon, Killian, & Jones, 1983; Johnson, Saupe, & Dempsey, 1992; Legrand et al., 2007; Mancini et al., 1997; Yoshiga & Higuchi, 2003). In a heterogeneous sample of runners, a high correlation has been shown between  $VO_2$  max and performance, however, in a homogeneous sample of runners this becomes a poor correlation, with performance closely linked to submaximal oxygen consumption or economy (Conley & Krahenbuhl, 1980; Coyle, Sidossis, Horowitz, & Beltz, 1992; Daniels & Daniels, 1992; Lucia, Hoyos, Perez, Santalla, & Chicharro, 2002; Noakes, 2003). This is likely due to more economical athletes being able to work at a lower percentage of their maximal oxygen consumption, resulting in a lower blood lactate concentration at any given exercise intensity (Burgess & Lambert, 2010; Morgan, Martin, & Krahenbuhl, 1989). Conley and Krahenbuhl (1980) investigated the correlation between performance and economy in 12 elite male runners, finding that 65.4% of the variation observed in the 10km race time could be explained by running economy as all participants had a very similar  $VO_2$  max. Similar findings were observed in a sample of elite female runners (Conley, Krahenbuhl, Burkett, & Millar, 1981). Despite a poor relationship between  $VO_2$  max and 10km run performance in these homogeneous groups, all subjects demonstrated a high  $VO_2$  max. As  $VO_2$  max is indicative of the rate of the upper limit of Adenosine triphosphate production during exercise and in elite athletes is generally significantly higher than in healthy sedentary

subjects (Bassett & Howley, 2000; Joyner & Coyle, 2008; Levine et al., 1991), it is suggested that this variable may be an important determinant of achieving elite level performance (Conley & Krahenbuhl, 1980). In contrast, within this elite group, exercise economy may be a better indicator of performance, since, unlike VO<sub>2</sub> max, this is highly variable between athletes (Coyle et al., 1992; Losnegard, Myklebust, & Hallén, 2012; Saunders, Pyne, Telford, & Hawley, 2004). The mechanisms behind the variance in exercise economy are not well understood, therefore factors which may account for this during running and cycling have been thoroughly investigated. Coyle et al (1992) demonstrated that elite cyclists exercising at identical power outputs required different rates of oxygen uptake, apparent over a range of work rates. Using an instrumented pedal dynamometer, the researchers found that differences in economy were not due to cycling technique, but were strongly correlated with the percentage of type I slow twitch fibers in the vastus lateralis. The more economical cyclists were found to have a greater percentage of type I fibers. This was supported later by Lucia et al (2002) who proposed that increased type I and decreased type II muscle fiber distribution may contribute to improved economy. This is attributed to the increased oxygen and energy demand of type II fibers when compared to type I for a given amount of contractile work, as the former consumes ATP in a number of different processes thereby reducing economy (Coyle et al., 1992; Crow & Kushmerick, 1983). Endurance training has been shown to enhance the oxidative capacity of skeletal muscle (Coyle et al., 1992; Holloszy & Coyle, 1984; Paavolainen, Häkkinen, Hämmäläinen, Nummela, & Rusko, 1999), with Coyle et al (1992) observing a significant relationship between the number of years of endurance training and percentage of type I muscle fibers in participants, which was also strongly correlated with cycling economy. An improvement in running economy has also been noted in several studies following various training interventions (Bransford & Howley, 1976; Conley, Krahenbuhl, Burkett, & Millar, 1981; Franch, Madsen, Djurhuus, & Pederson, 1998; Morgan et al., 1995; Paavolainen et al., 1999; Pate, Sparling, Wilson, Cureton, & Miller, 1987; Pollock, Jackson, & Pate, 1980; Svedenhag & Sjodin, 1985). However, Lake and Cavanagh (1996) investigated the effect of a six-week training programme on the running economy and mechanics of 15 previously untrained males and found that although there was an increase in VO<sub>2</sub> max, as well as the fractional utilisation of VO<sub>2</sub> max, running performance and submaximal VO<sub>2</sub> following the intervention, running mechanics were unchanged (Lake & Cavanagh, 1996). These findings indicate that the changes in performance could be attributed to physiological rather

than biomechanical modifications. This is supported by findings of Losnegard et al. who found that technical skill and biomechanics during two different skiing techniques (V2 skating and double poling) and uphill running did not determine the economy of participants, as the most economic subjects in one exercise mode were also the most economic in the other exercise modes (Losnegard, Schäfer, & Hallén, 2014). Morgan et al (1995) suggested that routine, long-term exposure to distance running may be the stimulus for improvements in running economy, and this notion was supported by Jones (1998) who found that economy, as measured by submaximal  $\text{VO}_2$ , improved significantly each year in an elite female runner over 5 years of endurance training (Jones, 1998). These results suggest that the intervention used in Lake and Cavanagh's (1996) work may not have been long enough to see significant changes. Furthermore, endurance training can cause an increased utilization of fat as an energy production substrate during high intensity exercise, requiring a greater amount of oxygen for the synthesis of ATP when compared to carbohydrate metabolism (Daniels, 1985; Jones & Carter, 2000; Talanian, Galloway, Heigenhauser, Bonen, & Spriet, 2007), it is possible that this factor may have also influenced the findings of Lake and Cavanagh (1996).

Other factors that have been found to influence exercise economy include pedaling cadence while cycling (Coast, Cox, & Welch, 1986), diet (Poole & Henson, 1988), overtraining (Bahr, Opstad, Medbø, & Sejersted, 1991), proportion of mass in the legs relative to the trunk (Larsen, 2003; Lucia et al., 2006; Pate, Macera, Bailey, Bartoli, & Powell, 1992), sex (Helgerud, 1994) and breathing pattern (Jones, Dean, & Chow, 2003; Mueller, Petty, & Filley, 1970). The latter is of particular interest to this review as decreased efficiency and increased work of breathing contributes to the increased oxygen cost of breathing (Collett & Engel, 1986; Jones et al., 2003). The work of breathing is the product of the mean pressure generated by respiratory muscles and  $\text{VT}$ . The efficiency with which the respiratory muscles work is largely dependent on  $\text{VE}$ , airway resistance and lung compliance, posture and lung volume, factors which also determine the pattern of breathing (Fairbairn, 1989; Guenette et al., 2007; Jones et al., 2003). As respiratory muscle activity increases with  $\text{VE}$  and exercise intensity, these tissues need additional oxygen and blood flow to cope with increased metabolic demand, as well as the muscles involved in locomotion and cardiac tissue. At rest, the oxygen cost of breathing is around 2% of total body  $\text{O}_2$  consumption, increasing to 3 – 5 % during moderate exercise (Aaron, Seow, Johnson, & Dempsey, 1992). However,

during maximal exercise the metabolic and circulatory cost of breathing in the untrained person amounts to 10% of  $\text{VO}_2$  max and cardiac output, and up to as much as 16% in those who are highly trained (Aaron et al., 1992; Guenette et al., 2007; Harms et al., 1998). This can become problematic to athletes performing at high levels of intensity, as the ventilatory work that is required at such levels has a high oxygen cost potentially affecting oxygen supply to the locomotive muscles as well as contributing to the development of expiratory flow limitation and increased work of breathing (Guenette et al., 2007; Karp, 2008). These factors will be discussed at length in subsequent sections. Additionally, if the muscle activity exceeds oxygen delivery and depletes  $\text{O}_2$  stores in the muscle, aerobic production of ATP becomes inadequate for energy demands and anaerobic glycolysis is required to sustain availability of ATP. This energy production pathway results in an increased rate of lactic acid production, which allows further anaerobic ATP generation (Wasserman et al., 1973). Because lactic acid readily dissociates, in the physiological pH range it is almost completely buffered in the blood, with bicarbonate decreasing in similar quantities, resulting in increased  $\text{CO}_2$  and hydrogen ions leading to metabolic acidosis caused by lactic accumulation (Mateika & Duffin, 1995; Romer & Polkey, 2008; Wasserman et al., 1973). In this state, there is additional respiratory demand to influence compensatory tachypneic hyperventilation to minimize the drop of arterial pH and prevent arterial hypoxemia.

### **2.3. Limitation to exercise**

Traditionally, very little regard has been given to the possible limiting effects of ventilation on aerobic exercise in normoxic conditions. This is due to the respiratory systems considerable ventilatory reserve and ability to cope with increased metabolic demand during heavy exercise in young healthy untrained subjects (Dempsey et al., 1984; Fairbairn, 1989; Mota et al., 1999; Olafsson & Hyatt, 1969). It has been generally considered that mechanical limitations imposed by the properties of the airways, chest wall and the pressure generating capacity of the respiratory musculature are insufficient for flow and pressure responses during exercise to reach their maximum (Wilkie, Dominelli, Sporer, Koehle, & Sheel, 2015).

More recently, however, studies have indicated that endurance trained athletes with a higher  $\text{VO}_2$  max and maximal  $\text{CO}_2$  production, working at a high ventilatory demand

begin to approach mechanical limits for pressure and flow development. As a result, respiratory muscle fatigue, expiratory flow limitation and dynamic hyperinflation are fairly common in this population (Guenette & Sheel, 2007; Guenette, Witt, McKenzie, Road, & Sheel, 2007; Harms et al., 1998; Mota et al., 1999; Plowman & Smith, 2014; Romer & Polkey, 2008; Spengler, Roos, Laube, & Boutellier, 1999; Wilkie et al., 2015). Under these conditions the capacities of the cardiovascular and metabolic systems can exceed that of the respiratory system such that it could be considered a limitation to maximal work and therefore performance (Costill, 2001); as well as exacerbating the competitive relationship for blood flow between locomotor and respiratory muscles (Guenette, Witt, McKenzie, Road, & Sheel, 2007). These potential limitations to exercise will be discussed in depth below.

### **2.3.1. Expiratory flow limitation and dynamic hyperinflation**

Expiratory flow limitation (EFL) occurs, when for a given operational lung volume, expiratory flow plateaus despite increasing pressures (Guenette et al., 2007; Mota et al., 1999; Wilkie et al., 2015). This occurs when ventilatory demand exceeds the lung and chest walls capacity to generate flow through volume changes (Guenette et al., 2007).

While there is reserve available to increase ventilation during normal tidal flow, EFL occurs when tidal expiratory flow encroaches maximal expiratory flow and flow reserve is abolished (Agostoni, Pellegrino, Conca, Rodarte, & Brusasco, 2002; Pride & Macklem, 2011). However, Pellegrino (1999) found in an investigation of EFL in 9 stable male asthmatic patients that EFL occurred well before expiratory reserve was completely abolished (Pellegrino et al., 1999). This study used two methods of EFL detection to obtain these results, the partial flow method and negative expiratory pressure, (NEP) the latter being considered the most valid (Calverley & Koulouris, 2005; Valta et al., 1994). As the population in this study were all stable asthmatics, these results may not apply to healthy individuals, in whom EFL may occur when tidal flow encroaches maximal flow. In this situation, any further increase in expiratory flow must take place at an increased operational lung volume allowing higher expiratory flow and greater ventilation. In healthy subjects, EFL does not occur during tidal breathing at rest, as critical pressure is never achieved and expiratory flow is much less than maximal expiratory flow at the same lung volume (Murciano et al., 2000;

Pellegrino et al., 2001).

The occurrence of EFL has been extensively studied, particularly in those with COPD, elderly and obese subjects. In these studies, participants were tested for presence of EFL in seated and supine positions, all with varying results (Table 3). These erratic results were also apparent in the studies investigating EFL in healthy and trained individuals, generally during rest and incremental exercise to exhaustion (Table 3). The most obvious differences in these studies, which may account for the vast discrepancies in occurrence (ranging from 0 – 90% of participants) is the use of a number of different EFL detection methods and the various populations sampled. Although five techniques are mentioned in Table 3 the two most commonly used methods are the negative expiratory pressure technique and isovolume pressure/flow curve comparison (IPFV). Negative expiratory pressure involves application of a negative pressure at the mouth during a tidal expiration and comparing the ensuing expiratory flow volume curve with the previous control expiration (Koulouris et al., 1997). IPFV was, until recently, the conventional method since its proposal by Hyatt (Hyatt, 1961). This method involves tidal measurements of forced vital capacity (FVC) used to plot a flow-volume curve, which is compared to that of maximal flow (Koulouris et al., 1997).

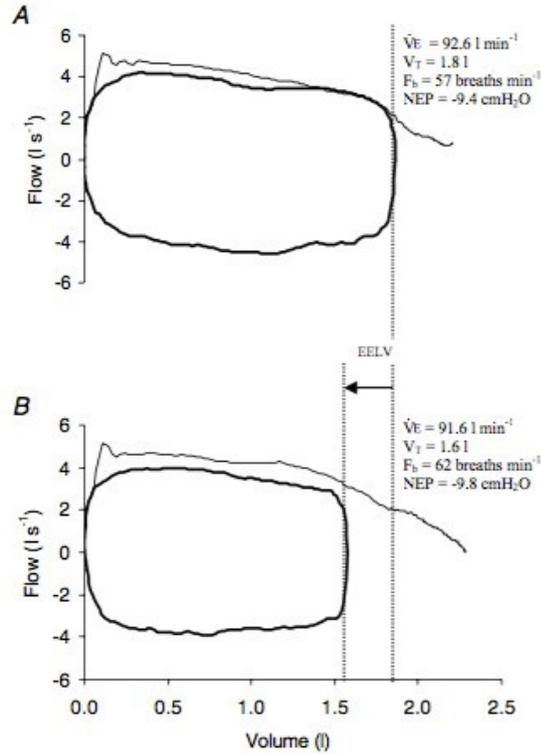
Despite the latter being frequently used, this method has many downfalls including, not allowing for thoracic gas compression artefacts, reliance on total lung capacity remaining constant during exercise, requires subjects to perform a true maximal inspiration, incorrect alignment of tidal flow volume, does not consider effect of previous volume and time history which at tidal breathing is different to a maximal expiratory maneuver and this method is technically complex and time consuming (Calverley & Koulouris, 2005; Ingram & Schilder, 1966; Koulouris et al., 1997; Tantucci et al., 1999). Koulouris (1997) concluded that even if measurements are done with a body plethysmograph to avoid gas compression artefacts, results can still be erroneous and this is likely due to the other limiting factors described. The introduction of the negative expiratory pressure method has simplified detection of EFL, by eliminating the need to use body plethysmography, not requiring patient co-operation and co-ordination, accounting for previous volume and time as it takes breath by breath analysis and because of its potential use in any position and during exercise (Calverley & Koulouris, 2005; Eltayara, Becklake, Volta, & Milic-Emili, 1996; Ingram &

Schilder, 1966; Koulouris et al., 1995; Tantucci et al., 1999). NEP is now considered the gold standard method, since validation by Valta et al (1994) in identifying EFL in mechanically ventilated intensive care patients by concomitant determination of iso-pressure flow volume relationships. However, consideration must be given to the generalisability of these results since these patients were mechanically ventilated, therefore obviating the effect of respiratory muscles.

It is quite clear that there are methodological concerns with methods of detection of EFL. Likely accounting for the conflicting results such as those found by Mota et al (1999) where flow limitation was found to be uncommon in male endurance athletes using the NEP detection method. On the other hand, Derchak et al (2000), McClaran et al (1998) and Guenette (2007) demonstrate the opposite; the last using NEP but the other investigators using the IPFV method (Table 3). Another possible cause for this variation in results is subjects overcoming EFL, a phenomenon that was not accounted for by Mota et al (1999) who only performed the NEP test during the last minute of each exercise level. In the study by Guenette (2007), multiple NEP recordings were obtained during each exercise stage so it could be determined if subjects were flow limited early in a steady-state exercise stage but could later, in the same stage, become non-flow limited. This was observed in 7 out of 18 subjects during the final exercise stage, when it appeared subjects were able to overcome EFL by altering their breathing patterns during high intensity exercise. Figure 1 shows a typical example of this adoption of a tachypnoeic breathing pattern, where participants tended to slightly decrease VT and increase respiratory rate such that VE did not change. As seen in Figure 1, when the tidal expiratory flow and maximal expiratory flow loops are aligned, it is demonstrated that the end-expiratory lung volume shifts, resulting in the avoidance of EFL (Guenette et al., 2007). This study also demonstrated EFL occurring in 90% of females tested, but only 43% of males (Guenette et al., 2007). Interestingly, Mota et al (1999) only investigated the presence of EFL in males, possibly giving another reason for the lower incidence of EFL reported in that study, since EFL seems to occur more frequently in females.

Despite aforementioned conflicting results, research suggests it can be assumed that EFL occurs more frequently in:

- The elderly - due to airway closing and senescence (Pride, 2005)
- COPD - due to airflow reduction from increased airway resistance, decreased lung compliance and airway collapsibility (Boczkowski et al., 1997; Eltayara et al., 1996; Mota et al., 1999)
- Obesity - due to breathing at low lung volumes, also seen with chronic congestive heart failure and sometimes restrictive pulmonary disease (Mota et al., 1999; Pankow et al., 1998)
- Highly trained endurance athletes during exercise - due to high ventilatory requirements during exercise causing an increase in mean tidal expiratory flow, reducing expiratory flow reserve by an increased rate and depth of breathing (Mota et al., 1999)
- Females - due to smaller caliber airways, smaller lung volumes and lower peak expiratory flow rates relative to age- and height- matched men causing EFL to occur at a lower VE (Guenette et al., 2007; McClaran, Harms, Pegelow, & Dempsey, 1998). This particularly appears to affect endurance trained females who are capable of working at a higher ventilatory demand as shown by McClaran et al (1998) and Guenette (2007) who found that 86% and 90% of trained females, respectively, demonstrated EFL during exercise (Guenette & Sheel, 2007; McClaran et al., 1998)
- Recumbency - due to decreasing lung volumes changing position from upright to supine, which can lead to airway closure as well as the effect of gravity (Castile, Mead, Jackson, Wohl, & Stokes, 1982; Pankow et al., 1998; Tucker & Sieker, 1960)



**Figure 1. Flow volume loops of tidal expiratory flow aligned with maximal expiratory flow, demonstrating EFL avoidance during exercise with figure A indicating the presence of EFL and figure B taken later in the exercise stage showing no presence of EFL (Guenette et al., 2007)**

**Table 3: Summary of studies investigating occurrence of EFL in order of subjects used**

Author	Subjects	EFL detection method	Results
(Baydur, Wilkinson, Mehdian, Bains, & Milic-Emili, 2004)	Total (132) Obstructive airway disease (56) Restrictive airway disease (47) Non-obese and healthy (20) Obese (9)	NEP (at rest – sitting and supine)	EFL detected in 10 (18%) patients with obstructive airway disease (4 only supine, 6 supine and sitting) No EFL detected in any other subjects
(Boczkowski et al., 1997)	Stable asthmatic patients (13)	NEP and IPVF (at rest – sitting and supine)	NEP – no patients EFL seated, 2 EFL supine (15%) IPVF – 6 EFL seated (46%)
(Boni et al., 2002)	COPD patients (20) 13M 7F	NEP (seated and during exercise)	EFL in 11 (55%) during exercise
(Dellacà et al., 2004)	Total (22) Stable COPD patients (15) Age matched healthy subjects (7)	Mead and Whittenberger method with forced oscillations (at rest)	EFL detected in 6 COPD patients (27%) during tidal ventilation Healthy subjects not flow limited
(Diaz et al., 2000)	COPD patients (52)	NEP (at rest)	EFL detected in 29 participants at rest (55%)
(Kosmas et al., 2004)	Stable asthmatics (20)	NEP (at rest and during incremental exercise)	EFL at rest in 1 patient (5%) EFL in 13 during exercise (65%)
(Koulouris et al., 1995)	COPD patients (26)	NEP (at rest – seated and supine)	EFL in 11 patients seated and supine (42%), 8 of these only while supine (31%)
(Koulouris et al., 1997)	Total (18) COPD (14) Healthy (4)	NEP (at rest and during exercise)	EFL at rest in 5 COPD patients (28%) EFL in 9 COPD patients at 33% work rate max ( $WR_{max}$ ), 12 at 66% $WR_{max}$ Healthy subjects were not flow limited up to 90% $WR_{max}$
(Ninane et al., 2012)	Total 17 COPD patients (12) Healthy subjects (5)	Manual compression of abdominal wall (MCA)	EFL in 6 COPD patients seated (50%) and 10 while supine (83%) Healthy subjects not flow limited
(Pellegrino et al., 1999)	Total (14) Males with mild to moderated fixed airflow obstruction (8) Healthy males (6)	IPVF	None were flow limited (0%)

(Valta et al., 1994)	Semi-recumbent mechanically ventilated patients in intensive care unit (12)	NEP and flow resistance by bypassing expiratory line of ventilator and exhaling into the atmosphere (ATM)	NEP – EFL in 9 (75%) ATM – agreement with NEP technique, however, gave erroneous results for 3 that were not flow limited
(Pankow et al., 1998)	<u>Total 14</u> Obese (8) Normal weight and age-matched (6)	NEP and IPVF (at rest – sitting and supine)	No flow limitation in 5 obese participants (63%) 2 EFL with both methods (20%), 1 EFL with IPVF but not NEP sitting EFL in 7 obese supine with both methods (88%)
(Ferretti, Giampiccolo, Cavalli, Milic-Emili, & Tantucci, 2001)	Healthy obese subjects (46) 18M 28F	NEP (at rest – seated and supine)	EFL detected in 10 subjects seated (22%), 4 of these were men EFL in 27 subjects supine (59%), 10 of these were men EFL detected in 56% of men and 61% of women supine
(De Bisschop et al., 2005)	2,612 elderly (Validated measurements from 1,318)	NEP (at rest)	EFL detected in 47% of participants 51.5% of F and 41.7% of M
(Olafsson & Hyatt, 1969)	Normal adult males (10)	IPVF and orifice technique	EFL in one patient (10%)
(McClaran et al., 1998)	<u>Total healthy female participants (29)</u> Fit females (14) Less fit females (15)	IPFV (during exercise)	EFL in 12 fit females (86%) and 4 of the less fit females (27%)
(Guenette et al., 2007)	Endurance trained subjects (18) 8M 10F	NEP (during incremental exercise)	EFL in 9 females (90%) and 3 males (43%) at high intensity exercise
(Derchak, Stager, Tanner, & Chapman, 2000)	College aged male athletes (16)	IPFV (during incremental exercise and following maximal exercise)	EFL detected in 8 participants (50%)
(Mota et al., 1999)	Male competition cyclists (10)	NEP (a rest and during exercise)	EFL present in 1 athlete (10%) at peak exercise

A number of studies have suggested that the increased lung volume associated with EFL leads to dynamic pulmonary hyperinflation (DH), as increasing ventilatory demand associated with EFL causes a premature reflex ending of expiration to increase end-expiratory lung volume in order to avoid dynamic compression of the airways. However, a compelling study by Landelli et al (2002) used a Starling resistor to externally impose EFL on six healthy male participants. This showed that reaching maximal expiratory flow doesn't always lead to dynamic hyperinflation in this population. Using an optoelectronic plethysmograph to track chest wall and lung volume changes, as well as various pressure and movement measurements during exercise, four subjects were determined to be hyperinflated towards the end of exercise, the remaining two were not. This result suggests the dynamic hyperinflation is not triggered by the development of and may occur discretely to EFL. This supports earlier work by Tantucci et al (1999) and Murciano et al (2000). Tantucci et al. (1999) found a significant relationship between dynamic hyperinflation and reported dyspnea using the Borg CR10 scale. The increase in dyspnea was reported to be more strongly related to the increase in end-expiratory lung volume (therefore hyperinflation), than the onset of EFL (Tantucci et al., 1999). This contradicts the findings of Landelli et al. (2002) who found that all participants (who were both hyperinflated and not hyperinflated), were performance limited at around 65% of maximal work rate (WR<sub>max</sub>) as determined by control exercise without the Starling resistor. Dyspnea was reported as the reason for termination in each case (all participants reported levels dyspnea between 9 – 10 on the Borg CR10 scale); whereas breathing sensation did not limit control exercise (reported dyspnea between 2 – 4 on the Borg CR10 scale) (Landelli, Aliverti, Kayser, Dellaca, et al., 2002). This study concluded that although DH reduced inspiratory reserve volume to almost zero in the four hyperinflated participants, this occurred late in the exercise protocol after Borg ratings had already increased significantly, suggesting the role of DH in exercise limitation may be overemphasized in previous literature (Landelli et al., 2002).

These results are supported by Murciano et al (2000) who found that most of their participants (eight single lung transplant patients) demonstrated EFL and DH, however, this was associated with high Borg ratings (scores 7 – 10) in only half of the participants. The others reported low exertional dyspnea with exercise capacity being limited by other factors such as peripheral muscle weakness and deconditioning.

Although a number of other previous papers reported a much more significant role of DH in exercise limitation (Bauerle, Chrusch, & Younes, 1998; Chen & Yan, 1999; O'Donnell & Webb, 1993; Tantucci et al., 1999), the study by landelli et al (2000) is particularly compelling with strong methodology and consistently using the most validated methods and equipment. Nonetheless, the strength of this investigation is limited by the sample size and the lack of female participants. Subsequent papers have gone on to appropriately suggest that EFL is instead 'associated with' or 'promotes' DH (Calverley & Koulouris, 2005; de Araujo, Karloh, Reis, Palú, & Mayer, 2015; Murciano et al., 2000; Tantucci, 2013).

Despite a lack of conclusive evidence as to the incidence of EFL and whether or not EFL causes DH, it has been commonly found and accepted that EFL and DH can impose limitations on aerobic exercise. This is likely due to effects on ventilatory capacity, breathing mechanics, ventilatory control, exertional dyspnea and exercise intolerance (Babb, 2013). Babb (2013) suggests that changes in these parameters can occur prior to EFL, possibly as early as VT approaching 50 – 60% of forced vital capacity (FVC). The presence of EFL during tidal breathing is associated with DH and intrinsic positive end-expiratory pressure (increased abdominal, pleural and alveolar pressures) (Aliverti et al., 2005; Calverley & Koulouris, 2005). The combined effects of increased flow resistance, hyperinflation and positive end-expiratory pressure increases elastic load on respiratory muscles, and since lung compliance decreases as lung volume increases and increased volume also forces the inspiratory respiratory muscles to contract from a shorter, less efficient length (Calverley & Koulouris, 2005; Grassino, Gross, Macklem, Roussos, & Zigelbaum, 1979; Guenette et al., 2007; Koulouris et al., 1995; Sheel, 2002). The resultant higher metabolic cost of breathing is associated with increased competition for blood flow and  $VO_2$ , possibly compromising perfusion to exercising limb locomotor muscles (Harms et al., 1998; Wilkie et al., 2015). This notion is supported in a study by Aliverti et al (2005), who found that under EFL conditions stroke volume decreased by 10%,  $PaO_2$  by 5% and  $O_2$  delivery to working muscles by around 15% when compared to control subjects, which then likely contributed to  $WR_{max}$  being 62.5% of that of the control subjects. Similar findings were reported by Komaz et al. (2004) who found that performance was limited to  $75 \pm 9\%$  of predicted  $WR_{max}$  in a sample of 13 stable asthmatics demonstrating EFL during exercise. Similarly, landelli et al (2002) who found that EFL performance was limited to 65% of  $WR_{max}$  of that of control, with exercise consistently limited by dyspnea.

Conditions of EFL, dynamic hyperinflation and positive end-expiratory pressure have also been suggested to lead to impaired venous return (Potter, Olafsson, & Hyatt, 1971), increased partial pressure of carbon dioxide (Bégin & Grassino, 1991; Calverley & Koulouris, 2005; Diaz et al., 2000; Haluszka, Chartrand, Grassino, & Milic-Emili, 1990) and decreased inspiratory capacity (Diaz et al., 2000; Murciano et al., 2000). The last of these is reported to be closely related to changes in self-reported breathlessness (O'Donnell, Bertley, Chau, & Webb, 1997).

### **2.3.3. Respiratory muscle fatigue and decreased blood flow to locomotor muscles**

Under normal physiological conditions, the increased demand for oxygen and blood flow by the respiratory muscles during maximal exercise can cause a substantial portion of cardiac output to be redirected to the respiratory muscles (Aaron et al., 1992; Guenette et al., 2007; Harms et al., 1998) and reduce blood flow to the working locomotor muscles. This is due to sympathetically mediated vasoconstriction, which helps to maintain the vital supply of respiratory muscle  $\text{VO}_2$  (Harms et al., 1997). At maximal intensity, the supply of energy is suggested to be inadequate to meet the needs of both respiratory and locomotor muscles (Aliverti & Macklem, 2001). This finding led to further investigation by Harms et al. (2000), who observed that decreasing the work of breathing consistently led to increased exercise tolerance, and increasing the work of breathing attenuated performance. These effects were largely attributed to the fatiguing effect of increased respiratory work on respiratory muscles and the limited blood flow distribution to limb locomotor muscles during very heavy exercise (Harms, Wetter, Croix, Pegelow, & Dempsey, 2000).

Findings reported by Harms et al (2002) are supported by a number of studies that showed that minimizing the work of breathing could increase exercise tolerance by reducing the oxygen cost of breathing and respiratory muscle fatigue, resulting in a greater proportion of total  $\text{VO}_2$  and cardiac output being utilized by locomotor muscles (Dempsey et al., 2006; Gallagher & Younes, 1989; Legrand et al., 2007; Levison & Cherniack, 1968, 1968; Mancini et al., 1997; Romer, Lovering Haverkamp, Pegelow, & Dempsey, 2006; Salvadego et al, 2015). This has been demonstrated in obese participants and those with chronic heart failure using helium rich air to unload

respiratory muscles, resulting in a decreased oxygen cost of exercise and a decreased perceived exertion during submaximal moderate and heavy exercise (Mancini et al., 1997; Salvadego et al., 2015). However, Mancini et al (1997) found that although the effects were pronounced on subjects with heart failure, the helium rich air minimally affected exercise performance measures in healthy subjects. The effect of respiratory muscle unloading and loading on locomotor blood flow has also been explored in healthy subjects of using proportion assisted ventilation. Harms et al (1998) found that unloading respiratory muscles in healthy trained subjects resulted in a decrease in total  $\text{VO}_2$  and cardiac output, with a concomitant increase in the proportion of total  $\text{VO}_2$  and cardiac output distributed to working locomotor muscles during maximal exercise (Harms et al., 1997) and a reduction in peripheral limb fatigue (Harms et al., 2000; Romer, Lovering, Haverkamp, Pegelow, & Dempsey, 2006). When the work of breathing was increased by loading the respiratory muscles, no change to cardiac output to the leg was observed (Wetter, Harms, Nelson, Pegelow, & Dempsey, 1999). However, the latter study was performed at submaximal exercise and it was subsequently proposed that the respiratory muscle work may not have been sufficient to cause respiratory muscle fatigue, though respiratory muscle fatigue must occur for unloading to improve performance (Gallagher & Younes, 1989; Krishnan, Zintel, McParland, & Gallagher, 1996). The generalisability of this study by Wetter et al (1999) may also be compromised due to the lack of female participants, since literature suggests that females may be more resistant than males to muscle fatigue (Hicks, Kent- Braun, & Ditor, 2001).

Muscle fatigue is defined as “a condition in which there is a loss in the capacity for developing force and/or velocity of a muscle, resulting from muscle activity under load and which is reversible by rest” (Plowman & Smith, 2014, p. 457). During periodic contraction, muscles benefit from an interval of recovery between contractions. This period of rest will prolong the time to muscle fatigue. This has been demonstrated in handgrip muscles developing a constant force showing that an increase in contraction time relative to relaxation time results in a decreased time to fatigue (Barcroft & Millen, 1939; Park & Rodbard, 1962; Rodbard & Pragay, 1968; Sjøgaard, Savard, & Juel, 1988). Bellemare and Grassino (1982) hypothesized based on these results that the diaphragm should fatigue more rapidly at any given tension if the ratio of inspiratory time over total breathing time ( $T_i/T_{tot}$ ) increases, a phenomenon shown to occur in asthmatics during exercise (Azab, El Mahalawy, Abd El Aal, & Taha, 2015). To

investigate this, four healthy male subjects breathed with a constant breathing pattern for 45 minutes or until transdiaphragmatic pressure (P<sub>di</sub>) could not be sustained, whichever occurred first. The breathing patterns included P<sub>di</sub> of 0.15 – 0.90 of maximal transdiaphragmatic pressure (P<sub>dimax</sub>), obtained by adjusting inspiratory resistance, and T<sub>i</sub>/T<sub>tot</sub> between 0.15 – 1. The product of P<sub>di</sub> and T<sub>i</sub>/T<sub>tot</sub> represents tension time of the diaphragm (TT<sub>di</sub>), it was found that breathing patterns that could be sustained for more than 45-minutes were found to have a TT<sub>di</sub> of about 0.15. It was concluded that the high TT<sub>di</sub> may result in fatigue due to diaphragmatic blood flow limitation, and therefore inspiratory time is an important determinant of diaphragmatic fatigue (Bellemare & Grassino, 1982). This work is supported by Johnson et al (1993) who later found that despite the substantial aerobic capacity of the diaphragm, it would exhibit fatigue during exercise to exhaustion at intensities greater than 80% of VO<sub>2</sub> max, this was demonstrated using bilateral phrenic nerve stimulation. With supra-maximal nerve stimulation immediately following exercise, the P<sub>di</sub> elicited was 25 – 50% below that of measurements prior to exercise and took 2 hours to recover (Johnson et al., 1992). Babcock et al (1995) later found that replicating fatiguing diaphragmatic force output in resting conditions was not sufficient to cause diaphragmatic fatigue, nor did it cause fatigue in a minimally used hand muscle. This suggests that diaphragm fatigue and peripheral muscle fatigue is likely due to additional stressors during whole body exercise. This is presumably a result of decreased blood flow as a consequence of competing for available cardiac output which then leads to inadequate oxygen transport and muscle fatigue (Sliwinski, Yan, Gauthier, & Macklem, 1996; Ward, Eidelman, Stubbing, Bellemare, & Macklem, 1988).

Previously a pattern with decreased breathing frequency and increased total breath cycle duration that is void of a tachypnoeic shift in elite athletes has been considered to result from resistance to respiratory muscle fatigue from years of training (Syabbalo, Krishnan, Zintel, & Gallagher, 1994) and a respiratory response to training to become metabolically and mechanically more efficient (Lucia, Hoyos, Joyos, & Chicharro, 2001). However, these ideas could be disputed by Kippelen et al (2005) who found that in a sample of 13 young male endurance trained athletes who had been training regularly for  $7 \pm 1.5$  years with a training volume of  $11 \pm 1$  hours per week, that from pre-competition to competitive periods the respiratory rate had increased and tidal volume had decreased (Kippelen et al., 2005), indicating that a low respiratory rate could be due to more than just adaptations to high volume training.

Respiratory rate can be influenced by an alteration in breathing pattern, as demonstrated by Jones et al (2003), who found that breathing frequency was significantly different during diaphragmatic ( $15.0 \pm 4.32$  breaths/min) and pursed lip ( $12.8 \pm 3.53$  breaths/min) breathing exercise when compared to spontaneous breathing ( $17.3 \pm 4.23$  breaths/min) in participants with COPD at rest. A similar change in respiratory rate was observed in COPD patients during low intensity exercise when pursed lip breathing was imposed (Mueller et al., 1970). Respiratory rate can also be influenced by participants awareness of their breathing being recorded, with several studies noting a change in respiratory time and rate with this awareness when compared to spontaneous breathing while participants are distracted or told that cardiac measures are being taken (Gilbert, Auchincloss, Brodsky, & Boden, 1972; Han, Stegen, Cauberghe, & Van de Woestijne, 1997; Mador & Tobin, 1991; O'Donnell et al., 1997; Paek & McCool, 1992; Perez & Tobin, 1985; Rodenstein, Mercenier, & Stănescu, 1985; Shea, Walter, Pelley, Murphy, & Guz, 1987; Western & Patrick, 1988). This effect on respiratory rate is particularly pronounced when breathing is monitored through a mouthpiece with a nose clip (Rodenstein et al., 1985), for this reason Jones et al (2003) used a canopy apparatus rather than a mouth piece; Muller et al (1970) did not report the method used to direct expired gas to the metabolic cart in their study, so it is likely that this would have been the more commonly used mouthpiece and nose clip. Due to the techniques used and lack of deception, changes observed by Jones and Muller (1970) in response to manipulated breathing patterns may not be truly reflective of what occurs during unmonitored breathing.

Due to compelling evidence that an increased work of breathing limits performance by increasing respiratory muscle fatigue (Johnson, Babcock, Suman, & Dempsey, 1993), decreasing blood flow to working locomotor muscles (Harms et al., 1997) and increasing dyspnea (Scano, Grazzini, Stendardi, & Gigliotti, 2006), many studies have investigated the effects of inspiratory muscle training on athletic performance of healthy individuals with varying results. Inspiratory muscle training (IMT) involves breathing against a variable resistance, with the aim of increasing the strength and endurance of the respiratory muscles. Volianitis et al (2001) investigated the effect of 11-weeks of inspiratory muscle training on rowing performance in 14 female competitive rowers, specifically performance during 6 minutes of all-out effort and a 5000-metre trial on a rowing ergometer. IMT consisted of 30 inspiratory efforts, twice daily, with inspiration against resistance equivalent to 50% peak inspiratory mouth

pressure; the control group used the same device but with an inspiratory resistance equivalent to 15% of peak inspiratory mouth pressure. Inspiratory muscle strength of the IMT group increased when compared to the control group ( $45.3 \pm 29.7\%$  vs  $5.3 \pm 9.8\%$ ), as did the distance covered during 6- minute all-out effort ( $3.5 \pm 1.2\%$  vs  $1.6 \pm 1.0\%$ ) and the time in the 5000-metre trial was decreased in the IMT group compared to control ( $3.1 \pm 0.8\%$  vs  $0.9 \pm 0.6\%$ ) (Volianitis et al., 2001). These results are similar to other studies using different exercise modalities, such as Fairbairn et al (1991) who found that time to exhaustion during a fixed intensity cycle at 90%  $\text{VO}_2$  max increased by 25% in the IMT group, compared to 4% in the control group; an 85% increase in swimming endurance time (Held & Pendergast, 2014); and an improvement in the 20-Metre Shuttle Run Test as measured by laps completed ( $16.3 \pm 3.9\%$ ) compared to pre-intervention testing (Tong et al, 2008). In contrast, despite using a very similar intervention methodology, Romer et al (2002) did not observe the same changes in 20-metre shuttle performance, with no improvement noted in the IMT or placebo group. However, total recovery time was reduced significantly in the IMT group following intervention when compared to the change in the control group ( $6.9 \pm 1.3\%$  vs  $0.7 \pm 1.3\%$ ). As all subjects included in the study by Tong et al (2008) were highly active individuals, it is plausible that the observed changes in 20-metre shuttle run performance following the intervention could, in part, be due to the effects of training, as no mention is made of ensuring consistent volume or intensity of training during the studies. McEntire et al (2016) later investigated the effects of moderate-intensity training and IMT on inspiratory muscle fatigue and performance. Fifteen subjects were randomly divided into the exercise training group ( $n = 8$ ) or the exercise training and IMT group ( $n = 7$ ). The exercise training and IMT group breathed through an inspiratory muscle trainer during exercise. An improvement in 5-mile time-trial performance was observed in both groups (exercise training =  $\sim 10\%$  and exercise training and IMT =  $\sim 18\%$ ) in addition to a decrease in inspiratory muscle fatigue (also significantly greater in expiratory training and IMT compared to exercise training group) (McEntire et al., 2016). Their results suggest that exercise training can have a significant effect on inspiratory muscle fatigue and performance, however, that it is more pronounced with the addition of IMT. Decreased respiratory muscle fatigue was largely attributed to increased respiratory muscle endurance related to IMT, with no mention of the influence on breathing pattern.

Volianitis et al (2001) was the only study to report an alteration in breathing pattern following IMT, noting an increased VE ( $119.9 \pm 12.8$  (SD) to  $122.5 \pm 12.3$  L/min<sup>-1</sup>) and increased VT ( $2.01 \pm 0.61$  to  $2.16 \pm 0.16$  L/min<sup>-1</sup>) following the intervention period. No significant change was noted in the exercise training control group, however, there was a tendency toward a tachypnoeic pattern with a 4.5% increase in breathing rate compared with only 1.5% in the IMT group. Hanel and Secher, (1991) reported similar findings of slight decreases in breathing frequency during maximal exercise following IMT (from 55 to 53 breaths per minute), whereas no change was observed in the control group. These results suggest that IMT could also decrease respiratory muscle fatigue through avoidance of a tachypnoeic shift and related DH and EFL.

## **Summary**

This review has highlighted the functions of breathing and how it can become dysfunctional, outlining the detrimental effects this can have both at rest and during exercise in healthy and pathological individuals. The difficulty in diagnosing dysfunctional breathing due to varying and intermittent symptoms has been discussed, reflected in the difficulty in successfully treating the disorder. Most importantly, the review has highlighted a lack of research into the effects of manual therapy on dysfunctional breathing patterns in otherwise healthy individuals and the effect this may have on endurance performance.

## Chapter Three: Methods

### 3.1. Participants

Prior to the study, calculations 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 9 points in SEBQ (Mitchell, Bacon, & Moran, 2016) or 0.2 L/min in VO<sub>2</sub> for economy (Bacon, Myers, & Karageorghis, 2012).

Before recruitment, a randomisation schedule was produced using a random number generator ([www.randomization.org](http://www.randomization.org)). This schedule was kept by an investigator who had no direct contact with prospective participants and allocation was sent upon notification of recruitment. Participants received four, weekly osteopathic treatments and six weeks of breathing retraining, either immediately (immediate-start) or after a 6-week delay (delayed-start), depending on allocation. They attended three or four laboratory sessions, respectively (Table 4). Participants in the delayed start group were led to believe the delay in starting was due to laboratory availability, however, the true purpose of this delay was revealed at the conclusion of their involvement.

This design allowed for two analyses. For the randomised controlled trial (RCT) analysis, the immediate-start group's first baseline (Baseline 1) and follow-up outcomes were compared with the first and second pre-intervention outcome measures (Baseline 1 and 2) of the delayed start group. For the cohort analysis, baseline measures (using the average of Baseline 1 and 2 for the delayed start group) were compared to the follow up measures for both groups

Table 4: Participation Schedule.

Week	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	
IS	Baseline 1	Intervention						Follow-up								
DS	Baseline 1	Delay						Baseline 2	Intervention						Follow-up	

Immediate Start (IS) and Delayed Start (DS). Familiarization session, at which maximal oxygen consumption test was performed, not shown, occurred prior to Baseline 1 stage.

Participants were recruited and screened for this study from the general public living within the greater Auckland region through direct contact with sport clubs and interaction with advertising on social media (Appendix 1). Respondents were then directed to an online inclusion criteria screening questionnaire (Appendix 2). For inclusion in the study, participants were required to be between 19 and 45 years of age. They also needed to be participating in aerobic activity for at least four hours per week and perceive that breathing might be limiting their sporting performance. Participants were excluded if they had smoked in the last six months, had any respiratory-related hospitalizations in the last six months, or any diagnosed cardiac, emotional or autonomic nervous system pathologies. Participants who suffered from asthma were included providing they had not had any hospitalizations related to this condition in the previous six-months and considered their asthma well controlled. Participants were asked to keep their activity levels consistent for the period of their involvement in the study. This was not monitored, but left to the participant’s discretion.

During recruitment, all participants were informed of the risks and requirements of participation both verbally and in written form. They were also required to give written informed consent in accordance with, and approved by, the Unitec Institute of Technology Ethics Committee guidelines (UREC 2013-1080). Participants were also asked to complete a general information questionnaire, which included relevant medical questions, prior to the commencement of their involvement. The study was registered with the Australia and New Zealand Clinical Trials Registry (ACTRN12614001301651).

## **3.2. Experimental Procedures**

This was part of a broader study, investigating the effects of OMT and BRT on a number of other outcome variables. All tests were performed at Unitec's Human Performance Laboratory located at the Unitec Mt Albert Campus, Auckland, New Zealand. Laboratory conditions were kept as standardized as possible by keeping all doors and windows closed and testing at similar times of the day; however, participants were given the choice of having a fan running during testing, which was accepted in most sessions. Although temperature was not recorded during laboratory sessions, the Auckland average maximum monthly temperatures varied from 29 degrees in February to 24 degrees in May, 2015, when most of the data collection took place.

### **3.2.1. Spirometry**

A hand-held spirometer was used for lung function measures. Each participant's forced vital capacity (FVC), forced expiratory volume in one second (FEV<sub>1</sub>) and peak expiratory flow (PEF) were taken at rest prior to ergometer bouts. Participants were seated and instructed to breathe out as hard and fast as they could through the spirometer. This was repeated three times with a short break in between, the average of these results were calculated.

### **3.1.1. Breath-hold time**

Resting breath-hold time (BHT) was taken prior to the ergometer bout in each session. Participants were instructed to start the breath hold time at the end of full exhalation and hold until they felt a diaphragmatic 'spasm' and/or an urge to breath.

### **3.1.1. Breathing dysfunction questionnaires**

Participants were required to fill out online versions of both the Nijmegen (NQ) and Self-Evaluation of Breathing Questionnaires (SEBQ) as part of their inclusion in the study and also at each submaximal testing session. Both questionnaires were completed at baseline and follow-up in the study. The Nijmegen Questionnaire (NQ) is the most validated and frequently used measure of dysfunctional breathing patterns to date, despite its initial conception as a test for hyperventilation (Rosalba Courtney & Greenwood, 2009). The use of the NQ as a measure in the validation or discrediting of novel approaches to assess hyperventilation is a common theme in studies found dating from 2001 to current years (Courtney, Cohen, & van Dixhoorn, 2011; Courtney & Cohen, 2008; Courtney & Greenwood, 2009; Hagman, Janson, & Emtner, 2008; Thomas et al., 2003; Thomas, McKinley, Freeman, & Foy, 2001). Self-Evaluation of Breathing Questionnaire (SEBQ) was developed to assess symptoms and behaviours affiliated with each dimension of dysfunctional breathing. This is achieved through a larger number of items specific to physiological, psychological and biomechanical dysfunction (Rosalba Courtney & Greenwood, 2009). Though it lacks sensitivity/specificity in the diagnosis of hyperventilation (Courtney & Greenwood, 2009), SEBQ has been demonstrated to have a high test-retest reliability (Mitchell et al., 2016) and has been suggested to be a useful complement to NQ in the assessment of a broader range of breathing symptoms (Courtney and van Dixhoorn, 2014).

### **3.1.1. Maximal oxygen consumption test**

Each participant was required to undergo an initial familiarization session, which involved familiarization with questionnaires, lung function tests, heart rate recording devices and procedures, and a maximal oxygen consumption test to exhaustion on the cycle ergometer. Prior to the maximal oxygen consumption test, each participants blood pressure was taken with a sphygmomanometer and stethoscope. In addition to this, their response to the General Information Questionnaire and a short verbal conversations with each participant about their own general health, to ensure their safety during the protocol. These were not systematically recorded for the study.

Participants were helped to set the ergometer seat and handle bar height suitably; these settings were recorded for use at each subsequent visit. All ergometer protocols began with a 5-minute warm-up with resistance set at either 50 Watts (W) or 100 W, after which the maximal oxygen consumption protocol would increase by 10, 15 or 20 W per minute depending on participant age, gender and estimated current level of fitness as determined by the participants reported level and load of training. Participants were required to continue cycling until volitional exhaustion or inability to maintain pedaling at 75 revolutions per minute, as displayed on a bicycle computer. Peak oxygen consumption was calculated as the average of the highest three breath-by-breath values. The test was determined to be maximal test if the participant achieved a respiratory exchange ratio greater than 1.15, a final heart rate within 10 beats per minute of age-predicted maximum ( $220 - \text{age}$  (in years)) and volitional exhaustion, as per criteria outlined in the British Association for Sports and Exercise Sciences guidelines for physiological testing of athletes (Winter, Jones, Davison, Bromley, & Mercer, 2006).

### **3.1.1. Fixed load submaximal test**

Baseline and follow-up laboratory sessions involved a 20-minute ergometer protocol of four, 5-minute bouts at fixed work rates of 30, 40, 55 and 70% of each participant's peak oxygen uptake, determined from  $\text{VO}_2$  and HR data collected in the familiarization session, and allowing for a 30s lag between workload changes and matched  $\text{VO}_2$ . Submaximal  $\text{VO}_2$  was recorded as the average  $\text{VO}_2$  during the final two minutes of each stage of exercise. Throughout the exercise bout, participants were required to pedal at a cadence of 75 revolutions per minute. At the conclusion of the protocol, the face-mask was removed and participants were required to sit and recover for 5 minutes, without moving or talking.

### **3.2.6. Equipment used in maximal and fixed load submaximal exercise tests**

#### **3.2.6.1 Ergometer**

All exercise bouts in the study were performed on an electro-magnetically braked cycle ergometer (Velotron, Racemate Inc, Seattle, USA). The braking system allowed the assessor to predetermine the level of resistance or power output, independent of pedal cadence, allowing for the creation of individualised ergometer 'protocols' for each participant that could be replicated during subsequent laboratory visits.

#### **3.2.6.2. Pulmonary gas exchange and spirometry**

During testing, participants wore a face-mask (Oro-Nasal 7450 V2 Mask, Hans Rudolph, Shawnee, U.S.A) connected to an automated breath-by-breath system (Moxus modular VO<sub>2</sub> system AEI Technologies, Pittsburgh, U.S.A) used to record pulmonary gas exchange measures. The Moxus equipment also recorded participants breathing frequency for the duration of the exercise bout and the mean breathing rate of the final two minutes of each exercise stage was used for analysis purposes.

Prior to each assessment, the gas analysis system was calibrated using a two-point calibration procedure as previously described by Vickery (2008). Following gas calibration, the flow-volume transducer was calibrated using a three-litre syringe until acceptable values ( $\pm 0.03\%$ ) were reached.

#### **3.2.6.3. Heart rate recording**

Heart rate was recorded using monitors (S810i or RS800x, Polar, Kempele, Finland) during all laboratory sessions and at home as part of a sub-study involved with this project.

### **3.2.1.1. Borg scale**

Rating of perceived exhaustion (RPE) was recorded using the 15-point (6 – 20) Borg scale (Appendix 3). Participants were introduced to the tool during a familiarization laboratory session where it was used at the end of the warm-up and every two minutes subsequently. At 4, 9, 14 and 19 minutes into the fixed-load submaximal protocol, participants were asked to rate their perceived exercise exertion on the Borg scale, held directly in front of them.

### **3.2.7. Breathing retraining and OMT intervention**

For the purpose of this study the breathing retraining (BRT) protocol involved breathing awareness, education, further instruction and practice (participants were required to practice 10 minutes, twice daily), positions to challenge breathing, tips on BRT and assessment of breathing pattern (Appendices 7, 8 & 9). The protocol was introduced immediately prior to the beginning of the intervention stage for each individual. This introduction involved an assessment of conscious breathing, as per the standardized BRT assessment protocol (Appendix 10), which was repeated at the follow-up laboratory session following their final osteopathic consultation. These visits also included delivery of each participant's initial and final BRT sessions, performed by the investigator present at the laboratory. Sessions 2 – 5 were delivered by an osteopath at the beginning of each participant's four, weekly consultations involving OMT, in addition to advice and cues to aid BRT.

The remainder of the 40-minute osteopathic consultation involved OMT following a protocol developed for the purpose of this study (Appendix 11). This protocol is a semi-standardised tool to direct the practitioner through assessment, diagnosis and treatment (Benjamin, Bacon, Verhoeff, & Moran, 2016). Practitioners assessed participants with observation of active movements, passive movements and palpation, and then came to a diagnosis based on any irregularities assessed. Irregularities could include a decrease in range of motion (ROM), change in texture, asymmetry or tenderness. Prescribed treatment allowed the practitioner to use any technique, or combination thereof that involved soft tissue massage, joint manipulation or functional osteopathic technique.

### **3.2.8. Practitioners**

Participants were assigned, based on availability and geographical convenience, to one of eight registered osteopaths. Practitioners all held a current New Zealand annual practicing certificate, worked in the Auckland region, actively treated breathing and attended a symposium to confer and put together a semi-standardised treatment. Following each visit, practitioners emailed the investigators each participant's BRT progress.

### **3.3. Analysis**

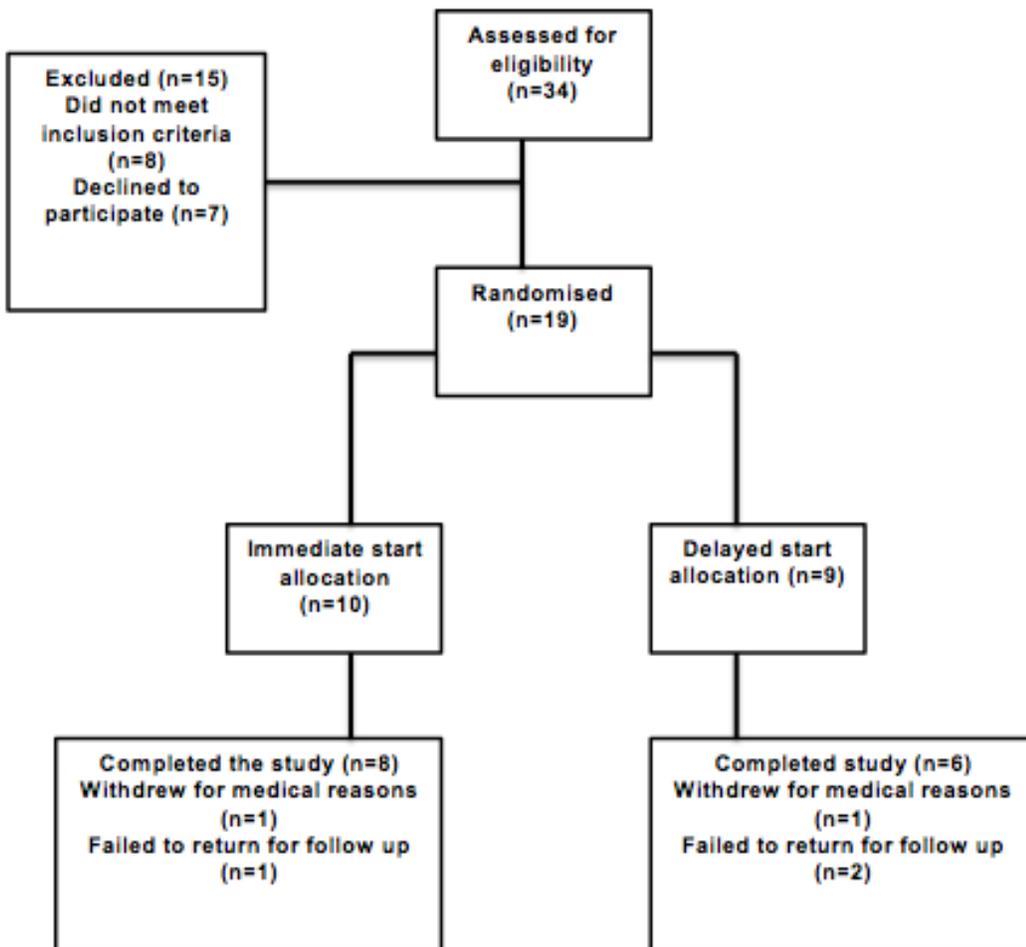
All statistical analysis was performed using SPSS (Version 22 IBM, Armonk, NY). Outcome variables were explored to check that assumptions of a normal distribution were not violated by examining z-scores for skewness and kurtosis, and the results of Shapiro-Wilks and Kolmogorov-Smirnoff tests for changes in pre- to post-intervention outcomes (Field, 2013).

Change in outcome measures were compared between those randomised to the immediate start group (who had received the intervention) and the delayed start group (who had not), using 2-way ANOVA or non-parametric equivalents. Data from both cohorts comparing pre- and post- intervention were then pooled to allow analysis of change in outcome variables for the entire group, using paired t-tests or non-parametric equivalents. The level of statistical significance was set at alpha <0.05.

## Chapter Four: Results

### 4.1. Participants

Thirty-four participants were recruited and screened, of whom 19 fitted the inclusion criteria and could commit to the requirements of the study. These 19 participants were randomly allocated into immediate- or delayed-start groups. Of all persons allocated groups, 14 participants completed this study with sufficient data for analysis, the rest withdrawing due to inability to attend all of the laboratory sessions required ( $n = 3$ ) and medical complications ( $n = 2$ ).



**Figure 2** Flow diagram of participant progress in study

All participants were asked to keep their activity level consistent during the period of the study, however, due to the various sporting codes and respective differences in season phase, this may have been more difficult for some.

**Table 5. Baseline Participant Anthropometric Characteristics**

	<b>Delayed*</b>	<b>Immediate*</b>
Age (yr)	25 ± 4	30 ± 8
Body Mass (kg)	74.6 ± 13.7	75.6 ± 8.9
Height (cm)	174 ± 11	173 ± 8

\*All values are mean ± SD

Participants characteristics are presented in Table 5.

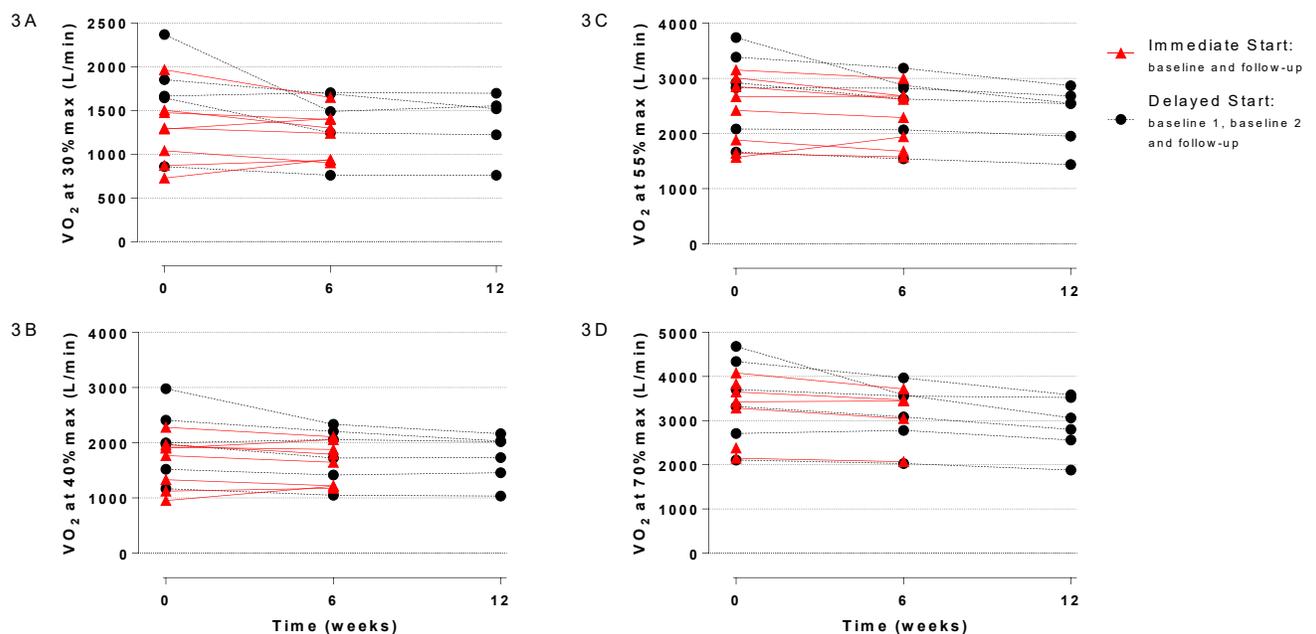
#### **4.2. Data**

An analysis of the distribution of changes of variables following intervention showed 55% of z- scores for skewness and kurtosis were within 95% confidence interval for normal distribution in the RCT analysis and 75% in the cohort analysis. Variables that violated assumptions of normality tended to be the high intensity exercise variables for VO<sub>2</sub>, breathing frequency, heart rate and Borg ratings of perceived exertion. Normality tests showed 45% of tests to be violated for change variables in the RCT analysis, for both groups, and 25% for baseline to follow-up change variables (cohort analysis). Given that violations for primary outcome variables were not severe, the results from parametric statistical analyses are reported.

## 4.2.1. RCT analysis

### Oxygen Consumption ( $VO_2$ )

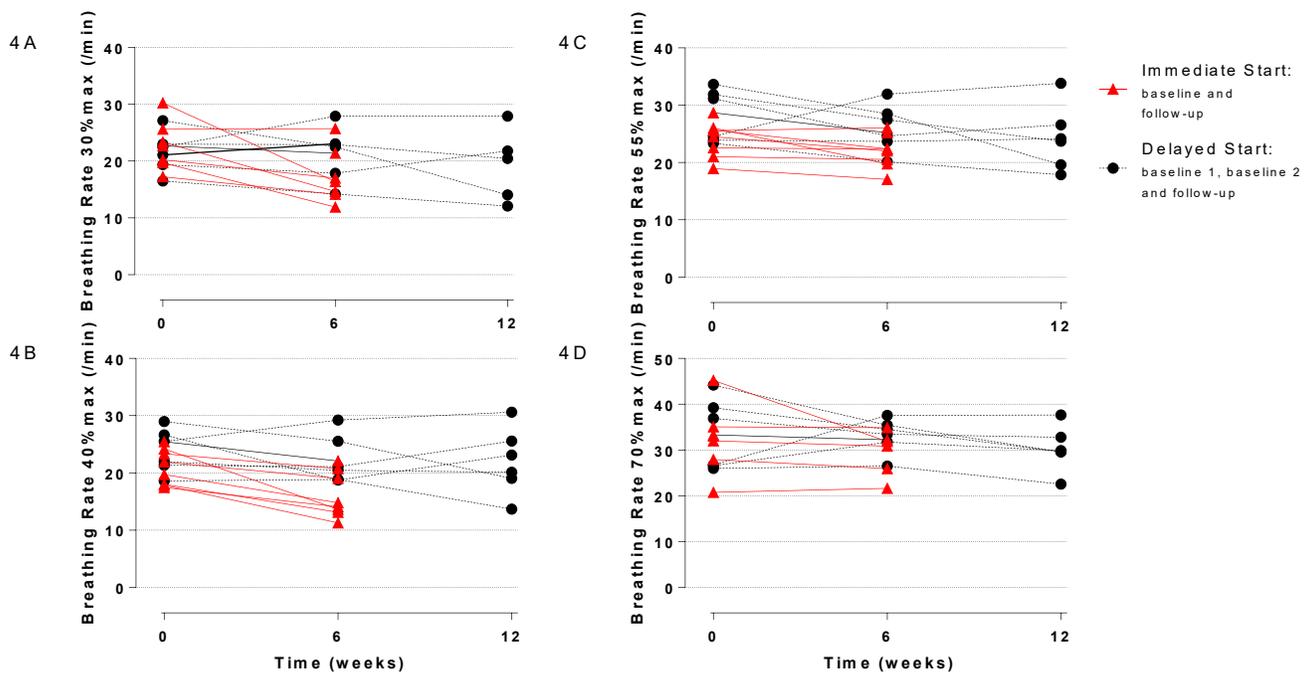
There were no differences between the immediate and delayed start groups from the first to the second visit for any of the  $VO_2$  variables at any intensity (percentage of  $VO_2$  max) (Figure 3A-D). Similarly, none of the other exercise, resting spirometry or questionnaire variables showed any between group differences in change from the first to the second visit visit. See Appendix 12 for graphs plotting all data variables.



**Figure 3 A-D** Plot of  $VO_2$  at individually-set workloads. For the immediate start group, Baseline 1 was at 0-weeks and follow-up at 6-weeks. For the delayed start group, Baseline 1 was at 0-weeks, Baseline 2 at 6-weeks and follow-up at 12-weeks. The RCT analysis addresses the change between weeks 0 and 6 for both groups. No significant difference in change between groups was shown. The cohort analysis addresses the change between baseline and follow up for both groups. Baseline for the immediate start group is Baseline 1, and for delayed start group it is the average of baseline 1 and 2. The P values for the change between baseline and follow-up visits (cohort analysis) were A.  $VO_2$  30%,  $P = 0.06$ ; B.  $VO_2$  40%,  $P = 0.12$ ; C.  $VO_2$  55%,  $P = 0.02$ ; and D.  $VO_2$  70%,  $P = 0.04$ .

## Breathing rate

Breathing rate at 40% VO<sub>2</sub> max during low intensity exercise showed an interaction trend between group and time that approached statistical significance ( $P = 0.08$ ). Immediate start group participants appeared to display a decreasing trend between first and second visits compared to those in the delayed start group for breathing rate at 40% VO<sub>2</sub> max, as shown by Figure 4B. Statistical significance was not attained at any other intensity.



**Figure 4 A-D** Plot of BR at individually-set workloads. For the immediate start group, Baseline 1 was at 0-weeks and follow-up at 6-weeks. For the delayed start, Baseline 1 was at 0-weeks, Baseline 2 at 6-weeks and follow-up at 12-weeks. The RCT analysis addresses the change between weeks 0 and 6 for both groups. No significant difference in change between groups was shown. The cohort analysis addresses the change between baseline and follow up for both groups. Baseline for the immediate start group is Baseline 1, and for the delayed start group it is the average of baseline 1 and 2. The P values for the change between baseline and follow-up visits (cohort analysis) were A. BR 30%,  $P = 0.03$ ; B BR 40%,  $P = 0.1$ ; C. BR 55%,  $P = 0.04$ ; and D. BR 70%,  $P = 0.08$ .

### 4.2.3. Cohort analysis

#### VO<sub>2</sub>

For the cohort analysis, , VO<sub>2</sub> at each intensity reduced from baseline to follow-up (Table 6 and Figure 3A-D), the higher intensities, 55 and 70% of VO<sub>2</sub> max, reaching statistical significance with P values of 0.02 and 0.04 respectively (Figure 3C-D). Statistical significance for these variables was retained with more conservative non-parametric analysis.

Table 6: Cohort analysis outcomes of oxygen consumption (VO<sub>2</sub>) variables (mL/min)

	<b>BASELINE</b>	<b>BASELINE SD</b>	<b>FOLLOW-UP</b>	<b>FOLLOW-UP SD</b>	<b>DIFFERENCE IN 95% CI</b>	<b>EFFECT SIZE</b>	<b>P VALUE</b>
VO <sub>2</sub> 30%	1370	414	1271	307	204 to 5.4	-0.57	0.06
VO <sub>2</sub> 40%	1762	503	1681	393	186 to 25	-0.44	0.12
VO <sub>2</sub> 55%	2505	637	2322	508	325 to 41	-0.74	0.02
VO <sub>2</sub> 70%	3053	1130	2370	1397	1319 to 48	-0.62	0.04

VO<sub>2</sub> percentages are of VO<sub>2</sub> max. Pre-intervention (PRE-INT). Pre-intervention standard deviation (PRE-INT SD). Post-intervention (POST-INT). Post-intervention standard deviation (POST-INT SD). Difference in 95% confidence interval (DIFFERENCE IN 95% CI).

## Breathing rate

Cohort analysis of breathing frequency demonstrated a trending decrease between pre-intervention and follow-up measurements (Table 5 and Figures 5 A-D). At lower intensities (30, 40 and 55% of VO<sub>2</sub> max) these values achieved statistical significance which was retained with non-parametric analysis (30% P = 0.03, 40% P = 0.01 and 55% P = 0.04). Breathing rate at 70% of VO<sub>2</sub> max approached statistical significance with parametric analysis (P = 0.08) and attained it with non-parametric analysis (P = 0.03).

Table 7: Cohort analysis outcomes of breathing rate variables (breaths per minute)

	<b>BASELINE</b>	<b>BASELINE SD</b>	<b>FOLLOW-UP</b>	<b>FOLLOW-UP SD</b>	<b>DIFFERENCE IN 95% CI</b>	<b>EFFECT SIZE</b>	<b>P VALUE</b>
BR 30%	22.1	3.97	18.5	5.23	6.78 to 0.38	-0.68	0.03
BR 40%	21.9	3.35	18.5	5.54	5.61 to 0.89	-0.8	0.01
BR 55%	25.4	3.53	22.9	4.28	4.74 to 0.16	-0.62	0.04
BR 70%	30.5	10.59	25.7	11.71	10.26 to 0.66	-0.51	0.08

Pre-intervention (PRE-INT). Pre-intervention standard deviation (PRE-INT SD). Post-intervention (POST-INT). Post-intervention standard deviation (POST-INT SD). Difference in 95% confidence interval (DIFFERENCE IN 95% CI). Breath rate per minute (BR). Percentage of VO<sub>2</sub> max (%).

## Heart rate, Borg rating of perceived exertion and resting variables

Analysis of remaining exercise variables and resting variables showed no significant difference following the intervention. See tables 8 – 10.

Table 8: Cohort analysis outcomes of heart rate variables (beats per minute)

	<b>BASELINE</b>	<b>BASELINE</b>	<b>FOLLOW-UP</b>	<b>FOLLOW-UP</b>	<b>DIFFERENCE</b>	<b>EFFECT</b>	<b>P VALUE</b>
		<b>SD</b>		<b>SD</b>	<b>IN 95% CI</b>	<b>SIZE</b>	
HR 40%	121.5	14.2	110	14.5	4.85 to 18.22	-1.00	0.003
HR 55%	143.5	13.6	139.7	7.4	-1.22 to 8.75	-0.44	0.1
HR 70%	149.5	44.4	140.1	60	-19.25 to 37.93	-0.19	0.5

Pre-intervention (PRE-INT). Pre-intervention standard deviation (PRE-INT SD). Post-intervention (POST-INT). Post-intervention standard deviation (POST-INT SD). Difference in 95% confidence interval (DIFFERENCE IN 95% CI). Heart rate (HR). Percentage of VO<sub>2</sub> max (%).

Table 9: Cohort analysis outcomes of Borg variables

	<b>BASELINE</b>	<b>BASELINE</b>	<b>FOLLOW-UP</b>	<b>FOLLOW-UP</b>	<b>DIFFERENCE</b>	<b>EFFECT</b>	<b>P VALUE</b>
		<b>SD</b>		<b>SD</b>	<b>IN 95% CI</b>	<b>SIZE</b>	
BORG 9M	10.1	1.47	9.8	1.35	-1.11 to 0.47	-0.23	0.4
BORG 14M	12.9	1.73	12.8	1.45	-1 to 0.89	-0.03	0.9
BORG 19M	12.6	4.6	12.9	7.2	-5.29 to 1.86	-0.28	0.3

Pre-intervention (PRE-INT). Pre-intervention standard deviation (PRE-INT SD). Post-intervention (POST-INT). Post-intervention standard deviation (POST-INT SD). Difference in 95% confidence interval (DIFFERENCE IN 95% CI). Borg

rating of perceived exertion (BORG). Minutes into cycle protocol (9M, 14M and 19M).

Table 10: Cohort analysis outcomes of resting variables

	<b>BASELINE</b>	<b>BASELINE</b>	<b>FOLLOW-UP</b>	<b>FOLLOW-UP</b>	<b>DIFFERENCE</b>	<b>EFFECT</b>	<b>P VALUE</b>
		<b>SD</b>		<b>SD</b>	<b>IN 95% CI</b>	<b>SIZE</b>	
SEBQ	23.29	11.55	16.29	7.17	-15.21 to 1.21	-0.49	0.09
NQ	12.71	6.93	13.71	6.44	-9.4 to 1.4	-0.43	0.1
PFR	520.97	146.97	519.05	138.4	-26.3 to 22.5	-0.05	0.9
FEV1	3.72	0.74	3.73	0.87	-0.18 to 0.16	0.03	0.9
FVC	4.07	0.98	4.1	1.08	-0.23 to 0.18	0.07	0.8
BHT	22.87	8.19	27.3	9	-9.66 to 0.78	0.54	0.08

Pre-intervention (PRE-INT). Pre-intervention standard deviation (PRE-INT SD). Post-intervention (POST-INT). Post-intervention standard deviation (POST-INT SD). Difference in 95% confidence interval (DIFFERENCE IN 95% CI). Self evaluation of breathing questionnaire (SEBQ). Nijmegen questionnaire (NQ). Peak flow rate (PFR). Forced expiratory volume in 1 second (FEV1). Forced vital capacity (FVC). Breath hold time (BHT).

## Chapter Five: Discussion

The primary finding from this study is that following six weeks of osteopathic manual therapy and breathing pattern retraining, a decreased  $\text{VO}_2$  consumption and breathing frequency during incremental steady state exercise was observed. These decreases were apparent through all exercise intensities (30, 40, 55 and 70% of  $\text{VO}_2$  max).

### *Breathing rate*

When the entire cohort was analysed, a 14.3% average decrease (from  $31 \pm 11$  breaths per minute before to  $26 \pm 12$  breaths per minute after at 70%  $\text{VO}_2$  max) in breathing frequency following the intervention, compared to before, was observed over all intensities of  $\text{VO}_2$  max (Table 7). A similar response was reported by Vickery (2008) who found that following four weeks of BRT participants' breathing frequency at maximal exercise decreased by 22% (from  $60 \pm 8$  breaths per minute at exhaustion before intervention to  $47 \pm 7$  breaths per minute at exhaustion following BRT) and  $V_T$  increased when compared to the breathing pattern adopted by the performance-matched control group. Similar breathing frequencies to Vickery were reported by Lucia et al (1999) who found that during maximal exercise professional and elite cyclists were breathing at approximately 51 and 58 breaths per minute, respectively. It should be noted that in the latter study, however, the subjects reached a greater maximal power output than those in the study by Vickery (>400W vs 335W).

An observed decrease in breathing frequency is significant as it may indicate a breathing pattern void of a tachypnoeic shift, which occurs when  $V_T$  plateaus and any further increase in  $V_E$  relies on an increased breathing rate (Gallagher, Brown, & Younes, 1987; Gravier, Delliaux, Delpierre, Guieu, & Jammes, 2013; Lucía, Carvajal, Calderón, Alfonso, & Chicharro, 1999; Vickery, 2008). Lucia et al (1999) suggested that the ability to maintain a breathing pattern void of a tachypnoeic shift was the most significant difference between amateur elite cyclists and the higher performing professionals studied. It was also noted that the professional cyclists had a lower maximal  $V_E$  at high workloads than the amateur elite cyclists (Lucía et al., 1999), a change that was also observed in Vickery's study following breathing retraining (Vickery, 2008) and attributed to the decrease in breathing frequency and increase in  $V_T$ . Ward (2007) suggests maintaining a low  $V_E$  may prevent expiratory flow limitation, a state which has been associated with decreased exercise tolerance (Diaz et al., 2000; Haluszka et al., 1990; Iandelli, Aliverti, Kayser, Dellaca, et al., 2002; McClaran et al., 1998; O'Donnell et al., 1997; Potter et al., 1971; Ward, 2007).

Vickery (2008) and Lucia et al (1999) both determined that the observed decreased respiratory frequency, therefore avoidance of a tachypnoeic breathing pattern at high intensities was attributed to a prolonged expiratory time and associated with increased performance (Lucía et al., 1999; Vickery, 2008). In the present study we did not record inspiratory or expiratory time or performance, therefore it cannot be ascertained that an increased expiratory time lead to a decrease in breathing frequency, however, based on research, this may be a likely cause. Generally, healthy humans increase the rate of breathing by decreasing expiratory time rather than inspiratory time (Folinsbee, Wallace, Bedi, & Horvath, 1983); a breathing pattern that can lead to breath stacking and hyperinflation over time with exercise (Chaitow et al., 2014). Increased breathing rate, expiratory flow limitation breath stacking and hyperinflation are considered to be detrimental to performance due to impaired alveolar ventilation (Aliverti et al., 2005; Chaitow et al., 2014; Otis, Fenn, & Rahn, 1950), respiratory muscle fatigue (Aaron et al., 1992; Grassino et al., 1979; Guenette et al., 2007; Otis et al., 1950; Sheel, 2002; St Croix, Morgan, Wetter, & Dempsey, 2000; Vogiatzis et al., 2009), dyspnea (Bauerle et al., 1998; Chen & Yan, 1999; landelli, Aliverti, Kayser, Dellacà, et al., 2002; O'Donnell & Webb, 1993; Tantucci et al., 1999) and higher metabolic cost of breathing associated with increased competition for blood flow, possibly compromising perfusion to exercising locomotor muscles (Harms et al., 1998; Lucía et al., 1999; Sheel et al., 2001; Wilkie et al., 2015).

Vickery (2008) attributes the decreased respiratory frequency observed to the emphasis on retraining and maintaining a diaphragmatic breathing pattern with forced exhalation during the intervention. Emphasis on diaphragmatic breathing was also apparent in the BRT protocol used in the present study, possibly similarly leading to the breathing frequency changes observed. Most participants made substantial progress in the breathing retraining protocol, on average progressing fourteen stages from baseline to follow-up (Appendix 7), indicating improved diaphragmatic use during this period. In the present study, BRT was supplemented by OMT which was not included in the investigation by Vickery. It is unable to be determined if the addition of OMT might have resulted in any differences in breathing frequency at high intensity exercise following intervention (26 breaths per minute in the present study vs 48 breaths per minute in Vickery's investigation) as the present study investigated breathing rates at 30, 40, 55 and 70% of  $\text{VO}_2$  max, whereas Vickery investigated the change in breathing rate during incremental exercise to volitional exhaustion. The lower exercise intensities in the present study likely account for the significantly lower breathing rates reported compared to Vickery, rather than the addition of OMT.

VO<sub>2</sub>

The present study measured VO<sub>2</sub> at 30, 40, 55 and 70% of VO<sub>2</sub> max to determine if the intervention could influence the O<sub>2</sub> cost or work of breathing associated with steady-state exercise at these intensities. Analysis of the entire cohort revealed a decrease (10.4%) in average oxygen consumption apparent at all reported exercise intensities (Table 6). Lower VO<sub>2</sub> at set submaximal intensities is indicative of more economical aerobic exercise, which is a strong predictor of endurance performance (Burgess & Lambert, 2010; Jones & Carter, 2000). Better exercise economy is beneficial to endurance performance as it results in utilisation of a lower percentage of VO<sub>2</sub> max for a given exercise intensity (Burgess & Lambert, 2010; Conley et al., 1981).

Endurance training is thought to improve exercise economy by increasing arterio-venous oxygen difference (Jones & Carter, 2000), increased muscle oxidative capacity and associated changes in motor recruitment patterns (Jones & Carter, 2000; Joyner & Coyle, 2008), decreased exercise ventilation and heart rate for exercise of the same intensity and improved movement technique (Jones & Carter, 2000). In the present study, well-trained participants were recruited to minimize the effect of endurance training on outcome measures, as presumably with the volume and load of training achieved prior to participation in the study, these training induced changes would have already occurred. With this in mind, it is more likely that the observed change in submaximal VO<sub>2</sub> was due to decreasing the work of breathing in participants by retraining breathing to a more efficient pattern. This is supported by the findings of Jones et al (2003), who found that manipulating breathing patterns resulted in a 4.8% reduction in VO<sub>2</sub> when compared to spontaneous breathing in patients with COPD. This decrease was slightly greater (1%) when participants performed pursed lip breathing exercise than diaphragmatic exercise. The former breathing technique was also investigated during low intensity exercise by Muller, with no significant effect on VO<sub>2</sub>. However, pursed lip breathing was associated with relief from dyspnea in 7 participants; this was attributed to decreased airway collapse, an increased V<sub>T</sub> and decreased respiratory rate and therefore avoidance of a tachypnoeic breathing pattern (Muller et al., 2012).

An increased work of breathing is likely associated with EFL and dynamic hyperinflation (Collett & Engel, 1986; Jones, Dean, & Chow, 2003; Sheel, 2002; Wilkie et al., 2015), therefore it is likely that the opposite is true for decreased respiratory work. Minimizing the work of breathing can be advantageous to performance as this reduces the oxygen cost of breathing and

respiratory muscle fatigue, resulting in a greater proportion of total  $\text{VO}_2$  and cardiac output being utilized by locomotor muscles, increasing exercise tolerance and capacity (Babcock et al., 2002; Dempsey et al., 2006; Gallagher & Younes, 1989; Harms et al., 1998, 2000; Legrand et al., 2007; Levison & Cherniack, 1968, 1968; Mancini et al., 1997; Mortensen et al., 2005; Romer et al., 2006; Salvadego et al., 2015). This was demonstrated in two investigations by Harms et al who found that unloading respiratory muscles using proportion assisted ventilation, decreased total  $\text{VO}_2$  and cardiac output with concomitant increases in  $\text{VO}_2$  and cardiac output directed to the leg during exercise (Harms et al., 1997, 1998). Similarly, the decrease in submaximal  $\text{VO}_2$  observed during exercise in the present study is likely due to a decrease in the metabolic cost of breathing. As no performance measures were taken it can not be conclusively determined if the decreased metabolic cost of exercise had any effect on performance, however research indicates there is generally a strong link between the two. The decrease in metabolic cost of exercise was not paralleled with a change in reported level of perceived exercise intensity.

Although changes in  $\text{VO}_2$  and breathing frequency were observed from baseline to follow up in the cohort analysis, there was no difference in these variables between the control and intervention groups in the RCT analysis, indicating that these changes may be due to factors beyond the intervention. It is possible that changes may have reflected participants' familiarisation with equipment and protocols or increased awareness of breathing following recruitment, but prior to intervention in the delayed start group. Increased awareness of breathing has been shown to have a substantial impact on breathing pattern. Using inductance plethysmography, Han et al (1997) found that an increase in inspiratory and expiratory time occurred resulting in a decreased respiratory rate when participants were aware of their breathing being recorded. This was compared to spontaneous breathing during the first 5 minutes where patients were informed that their breathing was not being recorded as the machine was being calibrated. Increased inspiratory and expiratory time was also observed, in addition to a markedly increased  $V_T$ , when the same participants breathed through a mouthpiece with an occluded nose, supporting similar results of earlier papers (Gilbert et al., 1972; Perez & Tobin, 1985; Rodenstein et al., 1985). These changes in ventilation have been attributed to the influence of additional dead space, stimulation of nasal and oral mucosa by the noseclip and mouthpiece, shift of respiratory route from unrestricted nose to mouth and the subjects attention on their breathing (Gilbert et al., 1972; Han et al., 1997). In the present study, a full face mask was used to take exercise gas exchange measures to avoid the influence of a mouthpiece and nose occlusion on respiratory pattern, thus minimizing all affects except for the

subjects awareness of their breath being recorded. During testing participants were aware that  $VO_2$  was being tested, but not of the recording of their respiratory rate, nor were they told anything about breathing mechanics to try to minimize the possible effects of increased respiratory awareness.

#### *Resting outcome measures*

Respiratory awareness could possibly account for some of the post-intervention change in self-reported symptoms measured by the NQ (14 after vs 13 before intervention) and SEBQ (16 vs 23). These changes in NQ scores were unexpected as the anticipated change in these measures was an overall decrease associated with the decreased respiratory rate and metabolic cost of exercise. However, many symptoms outlined in both questionnaires are either subtle and would likely go unnoticed by participants, or seem unrelated to breathing. Increased knowledge of dysfunctional breathing patterns and symptoms may make the participant more likely to notice and report these subsequently, possibly accounting for the unexpected increase in NQ scores following intervention. This effect of increased awareness was anecdotally recorded by several participants, but was not systematically recorded.

#### *Limitations*

It is possible that using a sample of primarily highly-trained athletes may have limited the effectiveness of the intervention, as it is reasonable to expect that higher level athletes may make smaller gains in cost of breathing due to them having smaller margins for improvement. This is supported by changes in performance noted by Hamlin and Hellemans, who discovered that following intermittent hypoxic training the greatest performance improvement was observed in the slowest athletes (Hamlin & Hellemans, 2007) and by respiratory function investigations by Boutellier et al. who found that respiratory training increased respiratory muscle endurance by 38% in trained subjects (Boutellier, Büchel, Kundert, & Spengler, 1992) compared to 50% in untrained subjects (Boutellier & Piwko, 1992). Endurance training has been shown to train the respiratory muscles and lower the ventilatory response to exercise (Boutellier et al, 1992); therefore it is also quite possible that in this trained population, a perception that breathing limited their performance could be due to breathing pattern and timing rather than structural limitations, potentially limiting the effectiveness of the manual therapy component of the intervention. There were a small number of participants recruited who were not such highly trained athletes, however, these people also made up a large number of those who dropped out of the study or had incomplete data sets due to their inability to complete 5 minutes of exercise

at 70%VO<sub>2</sub> max, therefore it cannot be determined whether or not these individuals experienced a greater change following the intervention when compared to the more highly-trained participants. Jones et al. suggest that in untrained patients with COPD, addressing the breathing pattern and timing at rest may be less beneficial than a manual therapy intervention (Jones et al., 2003). This is ascribed to respiratory mechanics and respiratory muscle efficiency playing a greater role in supporting spontaneous breathing than simply minimizing VO<sub>2</sub> and respiratory rate. Although changes in VO<sub>2</sub> and respiratory rate were observed following BRT during that study, effects of the intervention were not sustained, neither was the pattern adopted by the participants after the exercise had been performed. An intervention to optimize muscle mechanics to reduce biomechanical work and improve respiratory muscle efficiency was recommended to elicit a sustained improvement (Jones et al., 2003). It is apparent that further investigation is warranted to explore the difference in magnitude of change within different populations and using different interventions.

It is possible that the 6-week intervention may have been too short to demonstrate the full effects of improving exercise economy. Jones and Carter suggested that studies examining the effect of endurance training or breathing retraining on exercise economy, usually 6 – 12 weeks in duration, are generally not long enough to produce measurable improvements in metabolic efficiency, especially in those who are already trained (Jones & Carter, 2000). A lack of improvement, perhaps resulting from insufficient duration was observed by Vickery et al (2008), who found that VO<sub>2</sub> was unchanged during incremental exercise following 4 weeks of breathing pattern retraining, despite an increase in 20 km time trial performance. Nonetheless, some studies have shown significant improvements in exercise economy following 6 weeks of endurance and high intensity training in active and trained individuals (Billat, Flechet, Petit, Muriaux, & Koralsztein, 1999; Franch et al., 1998; Jones, Carter, & Doust, 1999). This may suggest that breathing retraining and adopting a breathing pattern efficient enough to bring about significant changes to exercise economy requires more time than an endurance training intervention, which may primarily elicit changes in mechanical efficiency. Although in the present study, a 6-week intervention was long enough to see significant change in some exercise variables, it is plausible that this change may have been greater given a longer intervention. To the authors knowledge, there are no previous papers exploring the effect of osteopathic manual therapy and breathing retraining on exercise economy, therefore it cannot be determined if the significant change in a short time was due to the compounding factor of manual therapy.

Throughout the duration of data collection, participants were asked to keep their activity consistent to minimize the effects of endurance training on outcome measures. This is in line with Boutellier et al (1992) who suggested that special attention must be paid to maintaining consistent levels of exercise, particularly for conditioned athletes, who in the present study were asked to train moderately and regularly with no intensive workouts. Keeping activity consistent in the present study was particularly difficult due to the variability of athletes recruited and the total time of trial data collection stretching over eight months and therefore various parts of the athlete's seasons. A number of participants were training for an upcoming world championship event and were therefore increasing the volume of their training for this. More intensive or more extensive training could interfere with the effects of respiratory training (Boutellier et al., 1992); the altered training volume could possibly have influenced outcome measures. In addition to this, other factors that could influence exercise economy were not monitored or considered in analysis, such as diet, proportion of mass in legs and sex of participant.

Whilst resting spirometric variables and breath hold time were taken as secondary outcome measures, none of these showed any significant change following the intervention. However, the strength of these measurements is limited by the lack of consistent protocols reported in previous literature, particularly for breath hold time. For resting spirometry values, no nose clip was used for nasal occlusion, and there was no set criteria for body position during measurement e.g. sitting or standing. To record breath hold time, although a consistent protocol was used, this may have been difficult for participants to replicate before and after treatment. Participants were asked to take as many spontaneous breaths as they would like, then fully exhale, at this point they were asked to indicate the beginning of the breath hold and to indicate again when they first felt the sensation of 'needing to breath' resulting in the conclusion of breath hold time. It is quite apparent that this method would be difficult to replicate over subsequent test days, as this could be influenced by motivation and varying degrees of sensation. For this reason, it may have been more appropriate to take this measurement multiple times with a rest between and calculate an average. Using such a method may also have minimized the effect of familiarisation on this outcome measure.

## **Conclusion**

It is unable to be determined if the observed decreased breathing frequency in this study was indicative of an absence of a tachypnoeic shift, EFL, dynamic hyperinflation and breath-stacking without direct measures (expiratory vs inspiratory time, NEP, breathing frequency vs  $V_T$ ). However, as previously described, research does suggest that a decreased breathing rate is associated with improved endurance performance.

The results of the present study suggest that combined osteopathic manual therapy and breathing pattern retraining has a positive effect on the metabolic cost of breathing during submaximal exercise. This was demonstrated by a significantly lower  $VO_2$  max and respiratory rate following intervention. The mechanism responsible for this improved economy of exercise remains unclear. Further research to enhance understanding of the role that BRT and OMT have in exercise economy in varying population groups is clearly warranted.

## References:

- Aaron, E. A., Seow, K. C., Johnson, B. D., & Dempsey, J. A. (1992). Oxygen cost of exercise hyperpnea: implications for performance. *Journal of Applied Physiology*, 72(5), 1818–1825.
- Agostoni, P., Pellegrino, R., Conca, C., Rodarte, J. R., & Brusasco, V. (2002). Exercise hyperpnea in chronic heart failure: relationships to lung stiffness and expiratory flow limitation. *Journal of Applied Physiology*, 92(4), 1409–1416.
- Aliverti, A., Dellaca, R. L., Lotti, P., Bertini, S., Duranti, R., Scano, G., ... Macklem, P. T. (2005a). Influence of expiratory flow-limitation during exercise on systemic oxygen delivery in humans. *European Journal of Applied Physiology*, 95(2-3), 229–242.
- Aliverti, A., Dellaca, R. L., Lotti, P., Bertini, S., Duranti, R., Scano, G., ... Macklem, P. T. (2005b). Influence of expiratory flow-limitation during exercise on systemic oxygen delivery in humans. *European Journal of Applied Physiology*, 95(2-3), 229–242.
- Aliverti, A., & Macklem, P. T. (2001). How and why exercise is impaired in COPD. *Respiration*, 68(3), 229–239.
- Arena, R., Myers, J., & Guazzi, M. (2008). The clinical importance of cardiopulmonary exercise testing and aerobic training in patients with heart failure. *Brazilian Journal of Physical Therapy*, 12(2), 75–87.
- Azab, N. Y., El Mahalawy, I. I., Abd El Aal, G. A., & Taha, M. H. (2015). Breathing pattern in asthmatic patients during exercise. *Egyptian Journal of Chest Diseases and Tuberculosis*, 64(3), 521–527.
- Babb, T. G. (2001). Breathing He-O<sub>2</sub> increases ventilation but does not decrease the work of breathing during exercise. *American Journal of Respiratory and Critical Care Medicine*, 163(5), 1128–1134.
- Babb, T. G. (2013). Exercise ventilatory limitation: the role of expiratory flow limitation. *Exercise and Sport Sciences Reviews*, 41(1), 11.
- Babcock, M. A., Pegelow, D. F., Harms, C. A., & Dempsey, J. A. (2002). Effects of respiratory muscle unloading on exercise-induced diaphragm fatigue. *Journal of Applied Physiology*, 93(1), 201–206. <http://doi.org/10.1152/jappphysiol.00612.2001>
- Bacon, C. J., Myers, T. R., & Karageorghis, C. I. (2012). Effect of music-movement synchrony on exercise oxygen consumption. *The Journal of Sports Medicine and Physical Fitness*, 52(4), 359–365.
- Bahr, R., Opstad, P. K., Medbø, J. I., & Sejersted, O. M. (1991). Strenuous prolonged

- exercise elevates resting metabolic rate and causes reduced mechanical efficiency. *Acta Physiologica Scandinavica*, 141(4), 555–563.
- Barcroft, H., & Millen, J. L. E. (1939). The blood flow through muscle during sustained contraction. *The Journal of Physiology*, 97(1), 17–31.
- Barker, N., & Everard, M. L. (2015). Getting to grips with “dysfunctional breathing.” *Paediatric Respiratory Reviews*, 16(1), 53–61.
- Bassett, D., & Howley, E. (2000). Limiting factors for maximum oxygen uptake and determinants of endurance performance. *Medicine and Science in Sports and Exercise*, 32(1), 70–84.
- Bauerle, O., Chrusch, C., & Younes, M. (1998). Mechanisms by which COPD affects exercise tolerance. *American Journal of Respiratory and Critical Care Medicine*, 157(1), 57–68.
- Baydur, A., Wilkinson, L., Mehdian, R., Bains, B., & Milic-Emili, J. (2004). Extrathoracic expiratory flow limitation in obesity and obstructive and restrictive disorders: effects of increasing negative expiratory pressure. *CHEST Journal*, 125(1), 98–105.
- Bégin, P., & Grassino, A. (1991). Inspiratory muscle dysfunction and chronic hypercapnia in chronic obstructive pulmonary disease. *American Review of Respiratory Disease*, 143(5\_pt\_1), 905–912.  
[http://doi.org/10.1164/ajrccm/143.5\\_Pt\\_1.905](http://doi.org/10.1164/ajrccm/143.5_Pt_1.905)
- Bellemare, F., & Grassino, A. (1982). Effect of pressure and timing of contraction on human diaphragm fatigue. *Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology*, 53(5), 1190–1195.
- Benjamin, J., Bacon, C., Verhoeff, W., & Moran, R. (2016). Preliminary development of a complex intervention for osteopathic management of dysfunctional breathing. *International Journal of Osteopathic Medicine*.
- Bevegård, B. S., Holmgren, A., & Jonsson, B. (1963). Circulatory studies in well trained athletes at rest and during heavy exercise, with special reference to stroke volume and the influence of body position. *Acta Physiologica Scandinavica*, 57(1-2), 26–50.  
<http://doi.org/10.1111/j.1748-1716.1963.tb02572.x>
- Billat, V. L., Flechet, B., Petit, B., Muriaux, G., & Koralsztein, J.-P. (1999). Interval training at VO<sub>2</sub>max: effects on aerobic performance and overtraining markers. *Medicine and Science in Sports and Exercise*, 31(1), 156–163.
- Boczkowski, J., Murciano, D., Pichot, M.-H., Ferretti, A., Pariente, R., & Milic-Emili, J. (1997). Expiratory flow limitation in stable asthmatic patients during resting breathing.

- American Journal of Respiratory and Critical Care Medicine*, 156(3), 752–757.
- Bodin, P., Kreuter, M., Bake, B., & Olsén, M. F. (2003). Breathing patterns during breathing exercises in persons with tetraplegia. *Spinal Cord*, 41(5), 290–295. <http://doi.org/10.1038/sj.sc.3101472>
- Bodin, P., Olsen, M. F., Bake, B., & Kreuter, M. (2005). Effects of abdominal binding on breathing patterns during breathing exercises in persons with tetraplegia. *Spinal Cord*, 43(2), 117–122.
- Bonetti, D. L., & Hopkins, W. G. (2009). Sea-level exercise performance following adaptation to hypoxia. *Sports Medicine*, 39(2), 107–127.
- Boni, E., Corda, L., Franchini, D., Chirolì, P., Damiani, G. P., Pini, L., ... Tantucci, C. (2002). Volume effect and exertional dyspnoea after bronchodilator in patients with COPD with and without expiratory flow limitation at rest. *Thorax*, 57(6), 528–532.
- Borge, C. R., Mengshoel, A. M., Omenaas, E., Moum, T., Ekman, I., Lein, M. P., ... Wahl, A. K. (2015). Effects of guided deep breathing on breathlessness and the breathing pattern in chronic obstructive pulmonary disease: A double-blind randomized control study. *Patient Education and Counseling*, 98(2), 182–190.
- Borghi-Silva, A., Carrascosa, C., Oliveira, C. C., Barroco, A. C., Berton, D. C., Vilaça, D., ... Neder, J. A. (2008). Effects of respiratory muscle unloading on leg muscle oxygenation and blood volume during high-intensity exercise in chronic heart failure. *American Journal of Physiology - Heart and Circulatory Physiology*, 294(6), H2465–H2472. <http://doi.org/10.1152/ajpheart.91520.2007>
- Bott, J., Blumenthal, S., Buxton, M., Ellum, S., Falconer, C., Garrod, R., ... others. (2009). *Guidelines for the physiotherapy management of the adult, medical, spontaneously breathing patient*. BMJ Publ. Group. Retrieved from [https://www.researchgate.net/profile/Rachel\\_Garrod/publication/24393894\\_British\\_Thoracic\\_Society\\_Physiotherapy\\_guideline\\_development\\_Group\\_Guidelines\\_for\\_the\\_physiotherapy\\_management\\_of\\_the\\_adultmedicalspontaneously\\_breathing\\_patients/links/0912f51484ec2790c1000000.pdf](https://www.researchgate.net/profile/Rachel_Garrod/publication/24393894_British_Thoracic_Society_Physiotherapy_guideline_development_Group_Guidelines_for_the_physiotherapy_management_of_the_adultmedicalspontaneously_breathing_patients/links/0912f51484ec2790c1000000.pdf)
- Boutellier, U., Büchel, R., Kundert, A., & Spengler, C. (1992). The respiratory system as an exercise limiting factor in normal trained subjects. *European Journal of Applied Physiology and Occupational Physiology*, 65(4), 347–353.
- Boutellier, U., & Piwko, P. (1992). The respiratory system as an exercise limiting factor in normal sedentary subjects. *European Journal of Applied Physiology and Occupational Physiology*, 64(2), 145–152.

- Bowler, S. D., Green, A., Mitchell, C. A., & others. (1998). Buteyko breathing techniques in asthma: a blinded randomised controlled trial. *Medical Journal of Australia*, 169, 575–578.
- Bradley, H., & Esformes, J. D. (2014). Breathing pattern disorders and functional movement. *International Journal of Sports Physical Therapy*, 9(1), 28.
- Bradstreet, T. C., & Parkman, T. J. (2016). Behavioral medicine treatment for patients with chronic obstructive pulmonary disease. *The Southwest Respiratory and Critical Care Chronicles*, 4(13), 45–48.
- Bransford, D. R., & Howley, E. T. (1976). Oxygen cost of running in trained and untrained men and women. *Medicine and Science in Sports*, 9(1), 41–44.
- Brown, R., DiMarco, A. F., Hoit, J. D., & Garshick, E. (2006). Respiratory dysfunction and management in spinal cord injury. *Respiratory Care*, 51(8), 853–870.
- Bruton, A., & Thomas, M. (2011). The role of breathing training in asthma management. *Current Opinion in Allergy and Clinical Immunology*, 11(1), 53–57.
- Buller, N. P., & Poole-Wilson, P. A. (1990). Mechanism of the increased ventilatory response to exercise in patients with chronic heart failure. *British Heart Journal*, 63(5), 281–283.
- Burdon, J. G., Killian, K. J., & Jones, N. L. (1983). Pattern of breathing during exercise in patients with interstitial lung disease. *Thorax*, 38(10), 778–784.
- Burgess, J., Ekanayake, B., Lowe, A., Dunt, D., Thien, F., & Dharmage, S. C. (2011). Systematic review of the effectiveness of breathing retraining in asthma management. *Expert Review of Respiratory Medicine*, 5(6), 789–807.
- Burgess, T. L., & Lambert, M. I. (2010). The effects of training, muscle damage and fatigue on running economy. *International SportMed Journal*, 11(4).
- Calverley, P. M. A., & Koulouris, N. G. (2005). Flow limitation and dynamic hyperinflation: key concepts in modern respiratory physiology. *European Respiratory Journal*, 25(1), 186–199.
- Carra, J., Candau, R., Keslacy, S., Giolbas, F., Borrani, F., Millet, G. P., ... Ramonatxo, M. (2003). Addition of inspiratory resistance increases the amplitude of the slow component of O<sub>2</sub> uptake kinetics. *Journal of Applied Physiology*, 94(6), 2448–2455.
- Caruana-Montaldo, B., Gleeson, K., & Zwillich, C. W. (2000). The control of breathing in clinical practice. *CHEST Journal*, 117(1), 205–225.
- Casciari, R. J., Fairshter, R. D., Harrison, A., Morrison, J. T., Blackburn, C., & Wilson, A. F. (1981). Effects of Breathing Retraining in Patients With Chronic Obstructive

- Pulmonary Disease. *Chest*, 79(4), 393–398.
- Castile, R., Mead, J., Jackson, A., Wohl, M. E., & Stokes, D. (1982). Effects of posture on flow-volume curve configuration in normal humans. *Journal of Applied Physiology*, 53(5), 1175–1183.
- Cerretelli, P., & Di Prampero, P. E. (2011). Gas Exchange in Exercise. In *Comprehensive Physiology*. John Wiley & Sons, Inc.
- Ceugniet, F., Cauchefer, F., & Gallego, J. (1996). Voluntary decrease in breathing frequency in exercising asthmatic subjects. *European Respiratory Journal*, 9(11), 2273–2279.
- Chaitow, L. (2007). Influences on the bodywork client: understanding breathing. *Massage & Bodywork*, 28–40.
- Chaitow, L., Gilbert, C., & Bradley, D. (2014). *Recognizing and Treating Breathing Disorders: A Multidisciplinary Approach* (2 edition). Churchill Livingstone.
- Chatham, K., Baldwin, J., Griffiths, H., Summers, L., & Enright, S. (1999). Inspiratory muscle training improves shuttle run performance in healthy subjects. *Physiotherapy*, 85(12), 676–683.
- Chen, R. C., & Yan, S. (1999). Perceived inspiratory difficulty during inspiratory threshold and hyperinflationary loadings. *American Journal of Respiratory and Critical Care Medicine*, 159(3), 720–727.
- Ciesla, N. D. (1996). Chest physical therapy for patients in the intensive care unit. *Physical Therapy*, 76(6), 609–625.
- CliftonSmith, T., & Rowley, J. (2011). Breathing pattern disorders and physiotherapy: inspiration for our profession. *Physical Therapy Reviews*, 16(1), 75–86.
- Coast, J. R., Cox, R. H., & Welch, H. G. (1986). Optimal pedalling rate in prolonged bouts of cycle ergometry. *Medicine and Science in Sports and Exercise*, 18(2), 225–230.
- Collett, P. W., & Engel, L. A. (1986). Influence of lung volume on oxygen cost of resistive breathing. *Journal of Applied Physiology*, 61(1), 16–24.
- Conley, D., & Krahenbuhl, C. (1980). Running economy and distance running performance of highly trained athletes. *Medicine and Science in Sports and Exercise*, 12(5), 357–360.
- Conley, D. L., Krahenbuhl, G. S., Burkett, L. N., & Millar, A. (1981). Training for aerobic capacity and running economy. *Physician Sportsmed*, 9(4), 107–115.
- Conley, D. L., Krahenbuhl, G. S., Burkett, L. N., & Millar, A. L. (1981). Physiological

- correlates of female road racing performance. *Research Quarterly for Exercise and Sport*, 52(4), 441–448.
- Cooper, S., Osborne, J., Newton, S., Harrison, V., Coon, J. T., Lewis, S., & Tattersfield, A. (2003). Effect of two breathing exercises (Buteyko and pranayama) in asthma: a randomised controlled trial. *Thorax*, 58(8), 674–679.
- Corrêa, E. C., & Bérzin, F. (2007). Efficacy of physical therapy on cervical muscle activity and on body posture in school-age mouth breathing children. *International Journal of Pediatric Otorhinolaryngology*, 71(10), 1527–1535.
- Costanzo, L. (2013). *Physiology* (5 edition). Philadelphia Pa.: Saunders.
- Costill, D. (2001). *Inside Running: Basics of Sports Physiology*. Carmel IN: Cooper Pub Group.
- Courtney, R. (2009). The functions of breathing and its dysfunctions and their relationship to breathing therapy. *International Journal of Osteopathic Medicine*, 12(3), 78–85.
- Courtney, R., & Cohen, M. (2008). Investigating the Claims of Konstantin Buteyko, M.D., Ph.D.: The Relationship of Breath Holding Time to End Tidal CO<sub>2</sub> and Other Proposed Measures of Dysfunctional Breathing. *The Journal of Alternative and Complementary Medicine*, 14(2), 115–123.
- Courtney, R., Cohen, M., & van Dixhoorn, J. (2011). Relationship between dysfunctional breathing patterns and ability to achieve target heart rate variability with features of “coherence” during biofeedback. *Alternative Therapies in Health and Medicine*, 17(3), 38.
- Courtney, R., & Greenwood, K. M. (2009). Preliminary investigation of a measure of dysfunctional breathing symptoms: The Self Evaluation of Breathing Questionnaire (SEBQ). *International Journal of Osteopathic Medicine*, 12(4), 121–127.
- Courtney, R., van Dixhoorn, J., Greenwood, K. M., & Anthonissen, E. L. M. (2011). Medically unexplained dyspnea: partly moderated by dysfunctional (thoracic dominant) breathing pattern. *Journal of Asthma*, 48(3), 259–265.
- Cowie, R. L., Conley, D. P., Underwood, M. F., & Reader, P. G. (2008). A randomised controlled trial of the Buteyko technique as an adjunct to conventional management of asthma. *Respiratory Medicine*, 102(5), 726–732.
- Coyle, E. F. (1994). Integration of the physiological factors determining endurance performance ability. *Exercise and Sport Sciences Reviews*, 23, 25–63.
- Coyle, E. F., Sidossis, L. S., Horowitz, J., & Beltz, J. D. (1992). Cycling efficiency is

- related to the percentage of type I muscle fibers. *Medicine and Science in Sports and Exercise*, 24(7), 782–788.
- Crow, M. T., & Kushmerick, M. J. (1983). Correlated reduction of velocity of shortening and the rate of energy utilization in mouse fast-twitch muscle during a continuous tetanus. *The Journal of General Physiology*, 82(5), 703–720.
- Da Costa, J. M. (1871). Irritable heart; a clinical study of a form of functional cardiac disorder and its consequences. *The American Journal of the Medical Sciences*, 121(1), 2–52.
- Daniels, J., & Daniels, N. (1992). Running economy of elite male and elite female runners. *Medicine and Science in Sports and Exercise*, 24(4), 483–489.
- Daniels, J. T. (1985). A physiologist's view of running economy. *Medicine and Science in Sports and Exercise*, 17(3), 332–338.
- Davies, A. (1995). Hyperventilation syndrome. *British Homeopathic Journal*, 84(2), 80–87.
- Dawes, H., Bateman, A., Culpan, J., Scott, O., Wade, D. T., Roach, N., & Greenwood, R. (2003). The effect of increasing effort on movement economy during incremental cycling exercise in individuals early after acquired brain injury. *Clinical Rehabilitation*, 17(5), 528–534.
- de Araujo, C. L. P., Karloh, M., Reis, C. M. D., Palú, M., & Mayer, A. F. (2015). Pursed-lips breathing reduces dynamic hyperinflation induced by activities of daily living test in patients with chronic obstructive pulmonary disease: a randomized cross-over study. *Journal of Rehabilitation Medicine*, 47(10), 957–962.
- De Bisschop, C., Marty, M. L., Tessier, J. F., Barberger-Gateau, P., Dartigues, J. F., & Guenard, H. (2005). Expiratory flow limitation and obstruction in the elderly. *European Respiratory Journal*, 26(4), 594–601.
- Dechman, G., & Wilson, C. R. (2004). Evidence underlying breathing retraining in people with stable chronic obstructive pulmonary disease. *Physical Therapy*, 84(12), 1189–1197.
- DeGuire, S., Gevirtz, R., Hawkinson, D., & Dixon, K. (1996). Breathing retraining: a three-year follow-up study of treatment for hyperventilation syndrome and associated functional cardiac symptoms. *Biofeedback and Self-Regulation*, 21(2), 191–198.
- Dellacà, R. L., Santus, P., Aliverti, A., Stevenson, N., Centanni, S., Macklem, P. T., ... Calverley, P. M. A. (2004). Detection of expiratory flow limitation in COPD using the forced oscillation technique. *European Respiratory Journal*, 23(2), 232–240.

- De Meersman, R. (1993). Heart rate variability and aerobic fitness. *American Heart Journal*, 125(3), 726–731.
- Dempsey, J. A., Hanson, P. G., & Henderson, K. S. (1984a). Exercise-induced arterial hypoxaemia in healthy human subjects at sea level. *The Journal of Physiology*, 355, 161.
- Dempsey, J. A., Hanson, P. G., & Henderson, K. S. (1984b). Exercise-induced arterial hypoxaemia in healthy human subjects at sea level. *The Journal of Physiology*, 355, 161–175.
- Dempsey, J. A., Romer, L., Rodman, J., Miller, J., & Smith, C. (2006). Consequences of exercise-induced respiratory muscle work. *Respiratory Physiology & Neurobiology*, 151(2-3), 242–250.
- Derchak, P. A., Stager, J. M., Tanner, D. A., & Chapman, R. F. (2000). Expiratory flow limitation confounds ventilatory response during exercise in athletes. *Medicine and Science in Sports and Exercise*, 32(11), 1873–1879.
- Dhungel, K. U., Malhotra, V., Sarkar, D., & Prajapati, R. (2008). Effect of alternate nostril breathing exercise on cardiorespiratory functions. *Nepal Med Coll J*, 10(1), 25–27.
- Diaz, O., Villafranca, C., Ghezzi, H., Borzone, G., Leiva, A., Milic-Emil, J., & Lisboa, C. (2000). Role of inspiratory capacity on exercise tolerance in COPD patients with and without tidal expiratory flow limitation at rest. *European Respiratory Journal*, 16(2), 269–275.
- Di Prampero, P. E. (2003). Factors limiting maximal performance in humans. *European Journal of Applied Physiology*, 90(3-4), 420–429.
- Eltayara, L., Becklake, M. R., Volta, C. A., & Milic-Emili, J. (1996). Relationship between chronic dyspnea and expiratory flow limitation in patients with chronic obstructive pulmonary disease. *American Journal of Respiratory and Critical Care Medicine*, 154(6), 1726–1734.
- Engelen, M., Porszasz, J., Riley, M., Wasserman, K., Maehara, K., & Barstow, T. J. (1996). Effects of hypoxic hypoxia on O<sub>2</sub> uptake and heart rate kinetics during heavy exercise. *Journal of Applied Physiology*, 81(6), 2500–2508.
- Fairbairn, M. S., Coutts, K. C., Pardy, R. L., & McKenzie, D. C. (1991). Improved respiratory muscle endurance of highly trained cyclists and the effects on maximal exercise performance. *International Journal of Sports Medicine*, 12(01), 66–70.
- Ferretti, A., Giampiccolo, P., Cavalli, A., Milic-Emili, J., & Tantucci, C. (2001). Expiratory flow limitation and orthopnea in massively obese subjects. *CHEST*, 119(5), 1401.

- Field, A. (2013). *Discovering Statistics Using IBM SPSS Statistics, 4th Edition* (4th edition). Los Angeles: SAGE Publications Ltd.
- Folinsbee, L. J., Wallace, E. S., Bedi, J. F., & Horvath, S. M. (1983). Respiratory pattern in trained athletes. *Elseiver, New York*, 205–212.
- Franch, J., Madsen, K., Djurhuus, M., & Pederson, P. (1998). Improved running economy following intensified training correlates with reduced ventilatory demands. *Medicine and Science in Sports and Exercise*, 30(8), 1250–1256.
- Gail, D. (1990). *Respiratory muscle fatigue - report of the respiratory muscle fatigue workshop group*. Amer Lung Assoc 1740 Broadway, New York, NY 10019.
- Gallagher, C. G., Brown, E., & Younes, M. (1987). Breathing pattern during maximal exercise and during submaximal exercise with hypercapnia. *Journal of Applied Physiology*, 63(1), 238–244.
- Gallagher, C. G., & Younes, M. (1989). Effect of pressure assist on ventilation and respiratory mechanics in heavy exercise. *Journal of Applied Physiology*, 66(4), 1824–1837.
- Gilbert, R., Auchincloss, J. H., Brodsky, J., & Boden, W. (1972). Changes in tidal volume, frequency, and ventilation induced by their measurement. *Journal of Applied Physiology*, 33(2), 252–254.
- Gosling, C., & Williams, K. A. (2004). Comparison of the effects of thoracic manipulation and rib raising on lung function of asymptomatic individuals. *J Osteopath Med*, 7, 103.
- Graham, W. G., Bradley, D. A., Kleczek, R., & Bartlett, D. (1978). Efficacy of chest physiotherapy and intermittent positive-pressure breathing in the resolution of pneumonia. *New England Journal of Medicine*, 299(12), 624–627.
- Grammatopoulou, E. P., Skordilis, E. K., Stavrou, N., Myrianthefs, P., Karteroliotis, K., Baltopoulos, G., & Koutsouki, D. (2011). The effect of physiotherapy-based breathing retraining on asthma control. *Journal of Asthma*, 48(6), 593–601.
- Grassino, A., Gross, D., Macklem, P. T., Roussos, C. H., & Zigelbaum, G. (1979). Inspiratory muscle fatigue as a factor limiting exercise. *Bulletin Européen de Physiopathologie Respiratoire*, 15(1), 105.
- Gravier, G., Delliaux, S., Delpierre, S., Guieu, R., & Jammes, Y. (2013). Inter-individual differences in breathing pattern at high levels of incremental cycling exercise in healthy subjects. *Respiratory Physiology & Neurobiology*, 189(1), 59–66.
- Grimby, G., Nilsson, N. J., & Saltin, B. (1966). Cardiac output during submaximal and maximal exercise in active middle-aged athletes. *Journal of Applied Physiology*,

21(4), 1150–1156.

- Grossman, P., De Swart, J. C. G., & Defares, P. B. (1985). A controlled study of a breathing therapy for treatment of hyperventilation syndrome. *Journal of Psychosomatic Research*, 29(1), 49–58. [http://doi.org/10.1016/0022-3999\(85\)90008-X](http://doi.org/10.1016/0022-3999(85)90008-X)
- Guenette, J. A., & Sheel, A. W. (2007). Physiological consequences of a high work of breathing during heavy exercise in humans. *Journal of Science and Medicine in Sport*, 10(6), 341–350.
- Guenette, J. A., Witt, J. D., McKenzie, D. C., Road, J. D., & Sheel, A. W. (2007a). Respiratory mechanics during exercise in endurance-trained men and women. *The Journal of Physiology*, 581(3), 1309–1322.
- Guenette, J. A., Witt, J. D., McKenzie, D. C., Road, J. D., & Sheel, A. W. (2007b). Respiratory mechanics during exercise in endurance-trained men and women: Respiratory mechanics. *The Journal of Physiology*, 581(3), 1309–1322.
- Hagman, C., Janson, C., & Emtner, M. (2008). A comparison between patients with dysfunctional breathing and patients with asthma: Dysfunctional breathing versus asthma. *The Clinical Respiratory Journal*, 2(2), 86–91.
- Hagman, C., Janson, C., & Emtner, M. (2011). Breathing retraining-A five-year follow-up of patients with dysfunctional breathing. *Respiratory Medicine*, 105(8), 1153–1159.
- Haluszka, J., Chartrand, D. A., Grassino, A. E., & Milic-Emili, J. (1990). Intrinsic PEEP and Arterial PCO<sub>2</sub> in Stable Patients with Chronic Obstructive Pulmonary Disease. *American Review of Respiratory Disease*, 141(5\_pt\_1), 1194–1197.
- Hamlin, M. J., & Hellems, J. (2007). Effect of intermittent normobaric hypoxic exposure at rest on haematological, physiological, and performance parameters in multi-sport athletes. *Journal of Sports Sciences*, 25(4), 431–441.
- Hanel, B., & Secher, N. H. (1991). Maximal oxygen uptake and work capacity after inspiratory muscle training: a controlled study. *Journal of Sports Sciences*, 9(1), 43–52.
- Han, J. N., Stegen, K., Cauberghs, M., & Van de Woestijne, K. P. (1997). Influence of awareness of the recording of breathing on respiratory pattern in healthy humans. *European Respiratory Journal*, 10(1), 161–166.
- Han, J. N., Stegen, K., De Valck, C., Clement, J., & Van de Woestijne, K. P. (1996). Influence of breathing therapy on complaints, anxiety and breathing pattern in patients with hyperventilation syndrome and anxiety disorders. *Journal of*

- Psychosomatic Research*, 41(5), 481–493.
- Hanon, C., Dorel, S., Delfour-Peyrethon, R., Leprêtre, P.-M., Bishop, D. J., Perrey, S., & Thomas, C. (2013). Prevalence of cardio-respiratory factors in the occurrence of the decrease in oxygen uptake during supra-maximal, constant-power exercise. *SpringerPlus*, 2(1), 651.
- Harms, C. A., Babcock, M. A., McClaran, S. R., Pegelow, D. F., Nিকেle, G. A., Nelson, W. B., & Dempsey, J. A. (1997a). Respiratory muscle work compromises leg blood flow during maximal exercise. *Journal of Applied Physiology*, 82(5), 1573–1583.
- Harms, C. A., Babcock, M. A., McClaran, S. R., Pegelow, D. F., Nিকেle, G. A., Nelson, W. B., & Dempsey, J. A. (1997b). Respiratory muscle work compromises leg blood flow during maximal exercise. *Journal of Applied Physiology*, 82(5), 1573–1583.
- Harms, C. A., Wetter, T. J., Croix, C. M. S., Pegelow, D. F., & Dempsey, J. A. (2000). Effects of respiratory muscle work on exercise performance. *Journal of Applied Physiology*, 89(1), 131–138.
- Harms, C. A., Wetter, T. J., McClaran, S. R., Pegelow, D. F., Nিকেle, G. A., Nelson, W. B., ... Dempsey, J. A. (1998). Effects of respiratory muscle work on cardiac output and its distribution during maximal exercise. *Journal of Applied Physiology*, 85(2), 609–618.
- Haseler, L. J., Hogan, M. C., & Richardson, R. S. (1999). Skeletal muscle phosphocreatine recovery in exercise-trained humans is dependent on O<sub>2</sub> availability. *Journal of Applied Physiology*, 86(6), 2013–2018.
- Häussermann, S., Schulze, A., Katz, I. M., Martin, A. R., Herpich, C., Hunger, T., & Texereau, J. (2015). Effects of a helium/oxygen mixture on individuals' lung function and metabolic cost during submaximal exercise for participants with obstructive lung diseases. *International Journal of Chronic Obstructive Pulmonary Disease*, 10, 1987.
- Hazlett-Stevens, H., & Craske, M. G. (2009). 14 Breathing retraining and diaphragmatic breathing. *General Principles and Empirically Supported Techniques of Cognitive Behavior Therapy*, 166.
- Held, H. E., & Pendergast, D. R. (2014). The effects of respiratory muscle training on respiratory mechanics and energy cost. *Respiratory Physiology & Neurobiology*, 200.
- Helgerud, J. (1994). Maximal oxygen uptake, anaerobic threshold and running economy in women and men with similar performances level in marathons. *European Journal of Applied Physiology and Occupational Physiology*, 68(2), 155–161.
- Hermant, E., Pichon, A., Lhuissier, F. J., & Richalet, J.-P. (2015). Periodic breathing in

- healthy humans at exercise in hypoxia. *Journal of Applied Physiology*, 118(1), 115–123.
- Hicks, A. L., Kent-Braun, J., & Ditor, D. S. (2001). Sex differences in human skeletal muscle fatigue. *Exercise and Sport Sciences Reviews*, 29(3), 109–112.
- Holloszy, J. O., & Coyle, E. F. (1984). Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. *Journal of Applied Physiology*, 56(4), 831–838.
- Holloway, E. A., & West, R. J. (2007a). Integrated breathing and relaxation training (the Papworth method) for adults with asthma in primary care: a randomised controlled trial. *Thorax*, 62(12), 1039–1042.
- Holloway, E. A., & West, R. J. (2007b). Integrated breathing and relaxation training (the Papworth method) for adults with asthma in primary care: a randomised controlled trial. *Thorax*, 62(12), 1039–1042.
- Howell, R. K., Allen, T. W., & Kappler, R. E. (1975). The influence of osteopathic manipulative therapy in the management of patients with chronic obstructive lung disease. *The Journal of the American Osteopathic Association*, 74(8), 757–760.
- Hyatt, R. E. (1961). The interrelationships of pressure, flow, and volume during various respiratory maneuvers in normal and emphysematous subjects. *American Review of Respiratory Disease*, 83(5), 676–683.
- landelli, I., Aliverti, A., Kayser, B., Dellaca, R., Cala, S. J., Duranti, R., ... others. (2002). Determinants of exercise performance in normal men with externally imposed expiratory flow limitation. *Journal of Applied Physiology*, 92(5), 1943–1952.
- landelli, I., Aliverti, A., Kayser, B., Dellacà, R., Cala, S. J., Duranti, R., ... others. (2002). Determinants of exercise performance in normal men with externally imposed expiratory flow limitation. *Journal of Applied Physiology*, 92(5), 1943–1952.
- Ingram, R. H., & Schilder, D. P. (1966). Effect of gas compression on pulmonary pressure, flow, and volume relationship. *Journal of Applied Physiology*, 21(6), 1821–1826.
- Johnson, B. D., Babcock, M. A., Suman, O. E., & Dempsey, J. A. (1993). Exercise-induced diaphragmatic fatigue in healthy humans. *The Journal of Physiology*, 460(1), 385–405.
- Johnson, B. D., Saupe, K. W., & Dempsey, J. A. (1992). Mechanical constraints on exercise hyperpnea in endurance athletes. *Journal of Applied Physiology*, 73(3), 874–886.

- Jones, A., & Carter, H. (2000). The effect of endurance training on parameters of aerobic fitness. *Sports Medicine*, 29(6), 373–386.
- Jones, A., Dean, E., & Chow, C. (2003). Comparison of the oxygen cost of breathing exercises and spontaneous breathing in patients with stable chronic obstructive pulmonary disease. *Physical Therapy*, 83(5), 424–431.
- Jones, A. M. (1998). A five year physiological case study of an Olympic runner. *British Journal of Sports Medicine*, 32(1), 39–43.
- Jones, A. M., Carter, H., & Doust, J. H. (1999). Effect of six weeks of endurance training on parameters of aerobic fitness. *Medicine & Science in Sports & Exercise*, 31(5), S280.
- Jones, A. Y., Dean, E., & Chow, C. C. (2003). Comparison of the oxygen cost of breathing exercises and spontaneous breathing in patients with stable chronic obstructive pulmonary disease. *Physical Therapy*, 83(5), 424–431.
- Jones, M., Troup, F., Nugus, J., Roughton, M., Hodson, M., Rayner, C., ... Pryor, J. (2015). Does manual therapy provide additional benefit to breathing retraining in the management of dysfunctional breathing? A randomised controlled trial. *Disability and Rehabilitation*. Retrieved from <http://www.tandfonline.com/doi/abs/10.3109/09638288.2014.941020>
- Joyner, M. J., & Coyle, E. F. (2008). Endurance exercise performance: the physiology of champions: Factors that make champions. *The Journal of Physiology*, 586(1), 35–44.
- Karam, M., Kaur, B. P., & Baptist, A. P. (2016). A modified breathing exercise program for asthma is easy to perform and effective. *Journal of Asthma*, (just-accepted), 00–00.
- Karp, J. (2008). An in-depth look at running economy. *Track Coach*, 182, 5801–5806.
- Kippelen, P., Caillaud, C., Robert, E., Connes, P., Godard, P., & Prefaut, C. (2005). Effect of endurance training on lung function: a one year study. *British Journal of Sports Medicine*, 39(9), 617–621.
- Kosmas, E. N., Milic-Emili, J., Polychronaki, A., Dimitroulis, I., Retsou, S., Gaga, M., ... Koulouris, N. G. (2004). Exercise-induced flow limitation, dynamic hyperinflation and exercise capacity in patients with bronchial asthma. *European Respiratory Journal*, 24(3), 378–384.
- Koulouris, N. G., Dimopoulou, I., Valta, P., Finkelstein, R., Cosio, M. G., & Milic-Emili, J. (1997). Detection of expiratory flow limitation during exercise in COPD patients. *Journal of Applied Physiology*, 82(3), 723–731.

- Koulouris, N. G., Valta, P., Lavoie, A., Corbeil, C., Chasse, M., Braidy, J., & Milic-Emili, J. (1995). A simple method to detect expiratory flow limitation during spontaneous breathing. *European Respiratory Journal*, *8*(2), 306–313.
- Kraft, A. R., & Hoogduin, C. A. (1984). The hyperventilation syndrome. A pilot study on the effectiveness of treatment. *The British Journal of Psychiatry*, *145*(5), 538–542.
- Krishnan, B., Zintel, T., McParland, C., & Gallagher, C. G. (1996). Lack of importance of respiratory muscle load in ventilatory regulation during heavy exercise in humans. *The Journal of Physiology*, *490*(Pt 2), 537.
- Lake, M., & Cavanagh, P. (1996). Six weeks of training does not change running mechanics or improve running economy. *Medicine and Science in Sports and Exercise*, *28*(7), 860–869.
- Larsen, H. B. (2003). Kenyan dominance in distance running. *Comparative Biochemistry and Physiology Part A: Molecular & Integrative Physiology*, *136*(1), 161–170.
- Legrand, R., Marles, A., Prieur, F., Lazzari, S., Blondel, N., & Mucci, P. (2007). Related trends in locomotor and respiratory muscle oxygenation during exercise. *Medicine and Science in Sports and Exercise*, *39*(1), 91.
- Levine, B. D., Lane, L. D., Buckey, J. C., Friedman, D. B., & Blomqvist, C. G. (1991). Left ventricular pressure-volume and Frank-Starling relations in endurance athletes. Implications for orthostatic tolerance and exercise performance. *Circulation*, *84*(3), 1016–1023.
- Levison, H., & Cherniack, R. M. (1968). Ventilatory cost of exercise in chronic obstructive pulmonary disease. *Journal of Applied Physiology*, *25*(1), 21–27.
- López, C. J., Navarro, M. A., Barbany, J. R., Garcia, M. J., Bonnin, M. R., & Valero, J. (1993). Salivary steroid changes and physical performance in highly trained cyclists. *International Journal of Sports Medicine*, *14*(3), 111–117.
- Losnegard, T., Myklebust, H. avar, & Hallén, J. (2012). No differences in O<sub>2</sub>-cost between V1 and V2 skating techniques during treadmill roller skiing at moderate to steep inclines. *The Journal of Strength & Conditioning Research*, *26*(5), 1340–1347.
- Losnegard, T., Schäfer, D., & Hallén, J. (2014). Exercise economy in skiing and running. *Frontiers in Physiology*, *5*. <http://doi.org/10.3389/fphys.2014.00005>
- Louvaris, Z., Zakynthinos, S., Aliverti, A., Habazettl, H., Vasilopoulou, M., Andrianopoulos, V., ... Vogiatzis, I. (2012). Heliox increases quadriceps muscle oxygen delivery during exercise in COPD patients with and without dynamic hyperinflation. *Journal of Applied Physiology*, *113*(7), 1012–1023.

- Lucía, A., Carvajal, A., Calderón, F. J., Alfonso, A., & Chicharro, J. L. (1999). Breathing pattern in highly competitive cyclists during incremental exercise. *European Journal of Applied Physiology and Occupational Physiology*, 79(6), 512–521.  
<http://doi.org/10.1007/s004210050546>
- Lucia, A., Esteve-Lanao, J., Oliván, J., Gómez-Gallego, F., San Juan, A. F., Santiago, C., ... Foster, C. (2006). Physiological characteristics of the best Eritrean runners—exceptional running economy. *Applied Physiology, Nutrition, and Metabolism*, 31(5), 530–540.
- Lucia, A., Hoyos, J., Joyos, J., & Chicharro, J. L. (2001). Physiology of Professional Road Cycling. *Sports Medicine*, 31(5), 339.
- Lucía, A., Hoyos, J., Perez, M., Santalla, A., & Chicharro, J. (2002). Inverse relationship between VO<sub>2</sub> max and economy/efficiency in world-class cyclists. *Medicine and Science in Sports and Exercise*, 34(12), 2079–2084.
- Mador, M. J., & Tobin, M. J. (1991). Effect of alterations in mental activity on the breathing pattern in healthy subjects. *American Review of Respiratory Disease*, 144(3\_pt\_1), 481–487.
- Mancini, D., Donchez, L., & Levine, S. (1997). Acute unloading of the work of breathing extends exercise duration in patients with heart failure. *Journal of the American College of Cardiology*, 29(3), 590–596.
- Markov, G., Spengler, C. M., Knoèpfli-Lenzin, C., Stuessi, C., & Boutellier, U. (2001). Respiratory muscle training increases cycling endurance without affecting cardiovascular responses to exercise. *European Journal of Applied Physiology*, 85(3-4), 233–239.
- Mateika, J., & Duffin, J. (1995). A review of the control of breathing during exercise. *European Journal of Applied Physiology and Occupational Physiology*, 71(1), 1–27.
- McClaran, S. R., Harms, C. A., Pegelow, D. F., & Dempsey, J. A. (1998). Smaller lungs in women affect exercise hyperpnea. *Journal of Applied Physiology*, 84(6), 1872–1881.
- McEntire, S. J., Smith, J. R., Ferguson, C. S., Brown, K. R., Kurti, S. P., & Harms, C. A. (2016). The effect of exercise training with an additional inspiratory load on inspiratory muscle fatigue and time-trial performance. *Respiratory Physiology & Neurobiology*, 230, 54–59.
- McHugh, P., Aitcheson, F., Duncan, B., & Houghton, F. (2003). Buteyko Breathing Technique for asthma: an effective intervention. *The New Zealand Medical Journal*

- (Online), 116(1187).
- McIlwaine, M. (2007). Chest physical therapy, breathing techniques and exercise in children with CF. *Paediatric Respiratory Reviews*, 8(1), 8–16.
- McKenna, J., & Riddoch, C. (2003). *Perspectives on Health and Exercise*. Palgrave.
- Miller, W. D. (1975). Treatment of visceral disorders by manipulative therapy. *The Research Status of Spinal Manipulative Therapy*, 295–301.
- Mitchell, A. J., Bacon, C. J., & Moran, R. W. (2016). Reliability and determinants of self-evaluation of breathing questionnaire (SEBQ) score: A symptoms-based measure of dysfunctional breathing. *Applied Psychophysiology and Biofeedback*, 41(1), 111–120.
- Morgan, D. W., Bransford, D. R., Costill, D. L., Daniels, J. T., Howley, E. T., & Krahenbuhl, G. S. (1995). Variation in the aerobic demand of running among trained and untrained subjects. *Medicine and Science in Sports and Exercise*, 27(3), 404–409.
- Morgan, D. W., Martin, P. E., & Krahenbuhl, G. S. (1989). Factors affecting running economy. *Sports Medicine*, 7(5), 310–330.
- Mortensen, S. P., Dawson, E. A., Yoshiga, C. C., Dalsgaard, M. K., Damsgaard, R., Secher, N. H., & González-Alonso, J. (2005). Limitations to systemic and locomotor limb muscle oxygen delivery and uptake during maximal exercise in humans. *The Journal of Physiology*, 566(1), 273–285.
- Mota, S., Casan, P., Drobnic, F., Giner, J., Ruiz, O., Sanchis, J., & Milic-Emili, J. (1999). Expiratory flow limitation during exercise in competition cyclists. *Journal of Applied Physiology*, 86(2), 611–616.
- Mueller, R. E., Petty, T. L., & Filley, G. F. (1970). Ventilation and arterial blood gas changes induced by pursed lips breathing. *Journal of Applied Physiology*, 28(6), 784–789.
- Muller, M. D., Kim, C.-H., Bellar, D. M., Ryan, E. J., Seo, Y., Muller, S. M., & Glickman, E. L. (2012). Effect of cold acclimatization on exercise economy in the cold. *European Journal of Applied Physiology*, 112(2), 795–800.
- Murciano, D., Ferretti, A., Boczkowski, J., Sleiman, C., Fournier, M., & Milic-Emili, J. (2000). Flow limitation and dynamic hyperinflation during exercise in COPD patients after single lung transplantation. *CHEST Journal*, 118(5), 1248–1254.
- Ninane, V., Leduc, D., KAFI, S. A., Nasser, M., Houa, M., & Sergysels, R. (2012). Detection of expiratory flow limitation by manual compression of the abdominal wall. *American Journal of Respiratory and Critical Care Medicine*. Retrieved from

<http://www.atsjournals.org/doi/abs/10.1164/ajrccm.163.6.2004150>

- Noakes, T. (2003). *Lore of running*. Human Kinetics.
- Noll, D. R., Johnson, J. C., Baer, R. W., & Snider, E. J. (2009). The immediate effect of individual manipulation techniques on pulmonary function measures in persons with chronic obstructive pulmonary disease. *Osteopathic Medicine and Primary Care*, 3, 9.
- Nosedá, A., Carpiaux, J. P., Vandepuut, W., Prigogine, T., & Schmerber, J. (1986). Resistive inspiratory muscle training and exercise performance in COPD patients. A comparative study with conventional breathing retraining. *Bulletin Europeen de Physiopathologie Respiratoire*, 23(5), 457–463.
- O'Donnell, D. E., Bertley, J. C., Chau, L. K., & Webb, K. A. (1997). Qualitative aspects of exertional breathlessness in chronic airflow limitation: pathophysiologic mechanisms. *American Journal of Respiratory and Critical Care Medicine*, 155(1), 109–115.
- O'Donnell, D. E., & Webb, K. A. (1993). Exertional breathlessness in patients with chronic airflow limitation: the role of lung hyperinflation. *American Review of Respiratory Disease*, 148(5), 1351–1357.
- Olafsson, S., & Hyatt, R. E. (1969). Ventilatory mechanics and expiratory flow limitation during exercise in normal subjects. *Journal of Clinical Investigation*, 48(3), 564.
- Olive, J. L., Slade, J. M., Dudley, G. A., & McCully, K. K. (2003). Blood flow and muscle fatigue in SCI individuals during electrical stimulation. *Journal of Applied Physiology*, 94(2), 701–708.
- Opat, A. J., Cohen, M. M., Bailey, M. J., & Abramson, M. J. (2000). A clinical trial of the Buteyko Breathing Technique in asthma as taught by a video. *Journal of Asthma*, 37(7), 557–564.
- Ortley, G. R., Sarnwick, R. D., Dahle, R. E., Roode, T. D., Zink, J. G., & Kilmore, M. A. (1980). Recording of physiologic changes associated with manipulation in healthy-subjects. In *Journal of The American Osteopathic Association*, 80, 228–229.
- Otis, A. B., Fenn, W. O., & Rahn, H. (1950). Mechanics of breathing in man. *Journal of Applied Physiology*, 2(11), 592–607.
- Paavolainen, L., Häkkinen, K., Hämmäläinen, I., Nummela, A., & Rusko, H. (1999). Explosive-strength training improves 5-km running time by improving running economy and muscle power. *Journal of Applied Physiology*, 86(5), 1527–1533.
- Paek, D., & McCool, D. (1992). Breathing patterns during varied activities. *Journal of Applied Physiology*, 73(3), 887–893.
- Pankow, W., Podszus, T., Gutheil, T., Penzel, T., Peter, J.-H., & Von Wichert, P. (1998).

- Expiratory flow limitation and intrinsic positive end-expiratory pressure in obesity. *Journal of Applied Physiology*, 85(4), 1236–1243.
- Papazian, L., Thomas, P., & Garbe, L. (2006). VENTILATION, PERFUSION MATCHING. *Medicine*, 152, 1982–1991.
- Park, S. R., & Rodbard, S. (1962). Effects of load and duration of tension on pain induced by muscular contraction. *American Journal of Physiology–Legacy Content*, 203(4), 735–738.
- Pate, R., Macera, C., Bailey, S., Bartoli, W., & Powell, K. (1992). Physiological, anthropometric, and training correlates of running economy. *Medicine and Science in Sports and Exercise*, 24(10), 1128–1133.
- Pate, R. R., Sparling, P. B., Wilson, G. E., Cureton, K. J., & Miller, B. J. (1987). Cardiorespiratory and metabolic responses to submaximal and maximal exercise in elite women distance runners. *International Journal of Sports Medicine*, 8(S 2), S91–S95.
- Pellegrino, R., Biggi, A., Papaleo, A., Camuzzini, G., Rodarte, J. R., & Brusasco, V. (2001). Regional expiratory flow limitation studied with Technegas in asthma. *Journal of Applied Physiology*, 91(5), 2190–2198.
- Pellegrino, R., Villosio, C., Milanese, U., Garelli, G., Rodarte, J., & Brusasco, V. (1999). Breathing during exercise in subjects with mild-to-moderate airflow obstruction: effects of physical training. *Journal of Applied Physiology*, 87(5), 1697–1704.
- Perez, W., & Tobin, M. J. (1985). Separation of factors responsible for change in breathing pattern induced by instrumentation. *Journal of Applied Physiology*, 59(5), 1515–1520.
- Perri, M., & Halford, E. (2004). Pain and faulty breathing: a pilot study. *Journal of Bodywork and Movement Therapies*, 8(4), 297–306.
- Plowman, S., & Smith, D. (2014). *Exercise Physiology for Health Fitness and Performance* (Fourth edition). Edinburgh ; New York: Lippincott Williams & Wilkins.
- Pluim, B. M., Zwinderman, A. H., Laarse, A. van der, & Wall, E. E. van der. (2000). The Athlete's Heart A Meta-Analysis of Cardiac Structure and Function. *Circulation*, 101(3), 336–344.
- Pollock, M. L., Jackson, A. S., & Pate, R. R. (1980). Discriminant analysis of physiological differences between good and elite distance runners. *Research Quarterly for Exercise and Sport*, 51(3), 521–532.
- Poole, D. C., & Henson, L. C. (1988). Effect of acute caloric restriction on work

- efficiency. *The American Journal of Clinical Nutrition*, 47(1), 15–18.
- Potter, W. A., Olafsson, S., & Hyatt, R. E. (1971). Ventilatory mechanics and expiratory flow limitation during exercise in patients with obstructive lung disease. *Journal of Clinical Investigation*, 50(4), 910–919.
- Pride, N. B. (2005). Ageing and changes in lung mechanics. *European Respiratory Journal*, 26(4), 563–565.
- Pride, N. B., & Macklem, P. T. (2011). Lung Mechanics in Disease. In *Comprehensive Physiology*. John Wiley & Sons, Inc. Retrieved from <http://onlinelibrary.wiley.com/doi/10.1002/cphy.cp030337/abstract>
- Ram, F. S. F., Holloway, E. A., & Jones, P. W. (2003). Breathing retraining for asthma. *Respiratory Medicine*, 97(5), 501–507.
- Resnick, E. S., Bielory, B. P., & Bielory, L. (2008). Complementary therapy in allergic rhinitis. *Current Allergy and Asthma Reports*, 8(2), 118–125.
- Ritz, T., von Leupoldt, A., & Dahme, B. (2006). Evaluation of a Respiratory Muscle Biofeedback Procedure—Effects on Heart Rate and Dyspnea. *Applied Psychophysiology and Biofeedback*, 31(3), 253–261.
- Roberts, S. E., Schreuder, F. M., Watson, T., & Stern, M. (2016). Do COPD patients taught pursed lips breathing (PLB) for dyspnoea management continue to use the technique long-term? A mixed methodological study. *Physiotherapy*. Retrieved from <http://www.sciencedirect.com/science/article/pii/S0031940616300268>
- Rodbard, S., & Pragay, E. B. (1968). Contraction frequency, blood supply, and muscle pain. *Journal of Applied Physiology*, 24(2), 142–145.
- Rodenstein, D. O., Mercenier, C., & Stănescu, D. C. (1985). Influence of the Respiratory Route on the Resting Breathing Pattern in Humans 1, 2. *American Review of Respiratory Disease*, 131(1), 163–166.
- Romer, L. M., Lovering, A. T., Haverkamp, H. C., Pegelow, D. F., & Dempsey, J. A. (2006a). Effect of inspiratory muscle work on peripheral fatigue of locomotor muscles in healthy humans. *The Journal of Physiology*, 571(2), 425–439.
- Romer, L. M., Lovering, A. T., Haverkamp, H. C., Pegelow, D. F., & Dempsey, J. A. (2006b). Effect of inspiratory muscle work on peripheral fatigue of locomotor muscles in healthy humans. *The Journal of Physiology*, 571(2), 425–439.
- Romer, L. M., McConnell, A. K., & Jones, D. A. (2002). Effects of inspiratory muscle training on time-trial performance in trained cyclists. *Journal of Sports Sciences*, 20(7), 547–590.

- Romer, L. M., & Polkey, M. I. (2008). Exercise-induced respiratory muscle fatigue: implications for performance. *Journal of Applied Physiology*, *104*(3), 879–888.
- Saladin, K. (2007). *Kenneth Saladin's Anatomy & Physiology 4th* (4 edition edition). McGraw-Hill Science/Engineering/Math.
- Salmasi, A.-M. (1993). Cardiac output during exercise. In A.-M. Salmasi & A. S. Iskandrian (Eds.), *Cardiac Output and Regional Flow in Health and Disease* (pp. 91–96). Springer Netherlands. Retrieved from [http://link.springer.com/chapter/10.1007/978-94-011-1848-4\\_9](http://link.springer.com/chapter/10.1007/978-94-011-1848-4_9)
- Salvadeo, D., Sartorio, A., Agosti, F., Tringali, G., Patrizi, A., Mauro, A. L., ... Grassi, B. (2015). Acute respiratory muscle unloading by normoxic helium–O<sub>2</sub> breathing reduces the O<sub>2</sub> cost of cycling and perceived exertion in obese adolescents. *European Journal of Applied Physiology*, *115*(1), 99–109.
- Saunders, P. U., Pyne, D. B., Telford, R. D., & Hawley, J. A. (2004). Factors affecting running economy in trained distance runners. *Sports Medicine*, *34*(7), 465–485.
- Saunders, P. U., Telford, R. D., Pyne, D. B., Hahn, A. G., & Gore, C. J. (2009). Improved running economy and increased hemoglobin mass in elite runners after extended moderate altitude exposure. *Journal of Science and Medicine in Sport*, *12*(1), 67–72.
- Scano, G., Grazzini, M., Stendardi, L., & Gigliotti, F. (2006). Respiratory muscle energetics during exercise in healthy subjects and patients with COPD. *Respiratory Medicine*, *100*(11), 1896–1906.
- Shea, S. A., Walter, J., Pelley, C., Murphy, K., & Guz, A. (1987). The effect of visual and auditory stimuli upon resting ventilation in man. *Respiration Physiology*, *68*(3), 345–357.
- Sheel, A. (2002). Respiratory muscle training in healthy individuals: physiological rationale and implications for exercise performance. *Sports Medicine*, *32*(9), 567–581.
- Sheel, A. W., Derchak, P. A., Morgan, B. J., Pegelow, D. F., Jacques, A. J., & Dempsey, J. A. (2001). Fatiguing inspiratory muscle work causes reflex reduction in resting leg blood flow in humans. *The Journal of Physiology*, *537*(1), 277–289.
- Sjøgaard, G., Savard, G., & Juel, C. (1988). Muscle blood flow during isometric activity and its relation to muscle fatigue. *European Journal of Applied Physiology and Occupational Physiology*, *57*(3), 327–335.
- Slader, C. A., Reddel, H. K., Spencer, L. M., Belousova, E. G., Armour, C. L., Bosnic-Anticevich, S. Z., ... Jenkins, C. R. (2006). Double blind randomised controlled trial of two different breathing techniques in the management of asthma. *Thorax*, *61*(8), 651–

656.

- Slatkowska, L., Jensen, D., Davies, G. A., & Wolfe, L. A. (2006). Phasic menstrual cycle effects on the control of breathing in healthy women. *Respiratory Physiology & Neurobiology*, *154*(3), 379–388.
- Sliwinski, P., Yan, S., Gauthier, A. P., & Macklem, P. T. (1996). Influence of global inspiratory muscle fatigue on breathing during exercise. *Journal of Applied Physiology*, *80*(4), 1270–1278.
- Spahija, J. A., & Grassino, A. (1996). Effects of pursed-lips breathing and expiratory resistive loading in healthy subjects. *Journal of Applied Physiology*, *80*(5), 1772–1784.
- Spengler, C. M., Roos, M., Laube, S. M., & Boutellier, U. (1999). Decreased exercise blood lactate concentrations after respiratory endurance training in humans. *European Journal of Applied Physiology and Occupational Physiology*, *79*(4), 299–305.
- Stanton, A. E., Vaughn, P., Carter, R., & Bucknall, C. E. (2008). An observational investigation of dysfunctional breathing and breathing control therapy in a problem asthma clinic. *Journal of Asthma*, *45*(9), 758–765.
- Stark-Leyva, K. N., Beck, K. C., & Johnson, B. D. (2004). Influence of expiratory loading and hyperinflation on cardiac output during exercise. *Journal of Applied Physiology*, *96*(5), 1920–1927.
- St Croix, C. M., Morgan, B. J., Wetter, T. J., & Dempsey, J. A. (2000). Fatiguing inspiratory muscle work causes reflex sympathetic activation in humans. *The Journal of Physiology*, *529*(2), 493–504.
- Stickford, A. S., Johnston, J. D., & Stager, J. M. (2015). Lower-Leg Compression, Running Mechanics, and Economy in Trained Distance Runners. *International Journal of Sports Physiology and Performance*, *10*(1), 76–83.
- Stone, R., & Stone, J. (2011). *Atlas of Skeletal Muscles* (7 edition). New York: McGraw-Hill Education.
- Svedenhag, J., & Sjodin, B. (1985). Physiological characteristics of elite male runners in and off-season. *Can J Appl Sport Sci*, *10*(3), 127–33.
- Syabbalo, N. C., Krishnan, B., Zintel, T., & Gallagher, C. G. (1994). Differential ventilatory control during constant work rate and incremental exercise. *Respiration Physiology*, *97*(2), 175–187.
- Taguchi, T., Adachi, H., Hoshizaki, H., Oshima, S., & Kurabayashi, M. (2015). Effect of

- physical training on ventilatory patterns during exercise in patients with heart disease. *Journal of Cardiology*, 65(4), 343–348.
- Talanian, J. L., Galloway, S. D., Heigenhauser, G. J., Bonen, A., & Spriet, L. L. (2007). Two weeks of high-intensity aerobic interval training increases the capacity for fat oxidation during exercise in women. *Journal of Applied Physiology*, 102(4), 1439–1447.
- Tantucci, C. (2013). Expiratory flow limitation definition, mechanisms, methods, and significance. *Pulmonary Medicine, Pulmonary Medicine*, 2013. Retrieved from <http://doi.org/10.1155/2013/749860>, 10.1155/2013/749860
- Tantucci, C., Ellaffi, M., Duguet, A., Zelter, M., Similowski, T., Derenne, J. P., & Milic-Emili, J. (1999). Dynamic hyperinflation and flow limitation during methacholine-induced bronchoconstriction in asthma. *European Respiratory Journal*, 14(2), 295–301.
- Tharion, E., Samuel, P., Rajalakshmi, R., Gnanasenthil, G., & Subramanian, R. K. (2012). Influence of deep breathing exercise on spontaneous respiratory rate and heart rate variability: a randomised controlled trial in healthy subjects. Retrieved from <http://imsear.li.mahidol.ac.th/handle/123456789/146093>
- Thomas, M., Bruton, A., & Ainsworth, B. (2015). Use of the Nijmegen Questionnaire in asthma. *ERJ Open Research*, 1(1), 33.
- Thomas, M., McKinley, R. K., Freeman, E., & Foy, C. (2001). Prevalence of dysfunctional breathing in patients treated for asthma in primary care: cross sectional survey. *Bmj*, 322(7294), 1098–1100.
- Thomas, M., McKinley, R. K., Freeman, E., Foy, C., Proddger, P., & Price, D. (2003). Breathing retraining for dysfunctional breathing in asthma: a randomised controlled trial. *Thorax*, 58(2), 110–115.
- Thomas, M., McKinley, R. K., Mellor, S., Watkin, G., Holloway, E., Scullion, J., ... Pavord, I. (2009). Breathing exercises for asthma: a randomised controlled trial. *Thorax*, 64(1), 55–61.
- Tong, T. K., Fu, F. H., Chung, P. K., Eston, R., Lu, K., Quach, B., ... So, R. (2008). The effect of inspiratory muscle training on high-intensity, intermittent running performance to exhaustion. *Applied Physiology, Nutrition, and Metabolism*, 33(4), 671–681.
- Tucker, D. H., & Sieker, H. O. (1960). The Effect of Change in Body Position on Lung Volumes and Intrapulmonary Gas Mixing in Patients with Obesity, Heart Failure, and

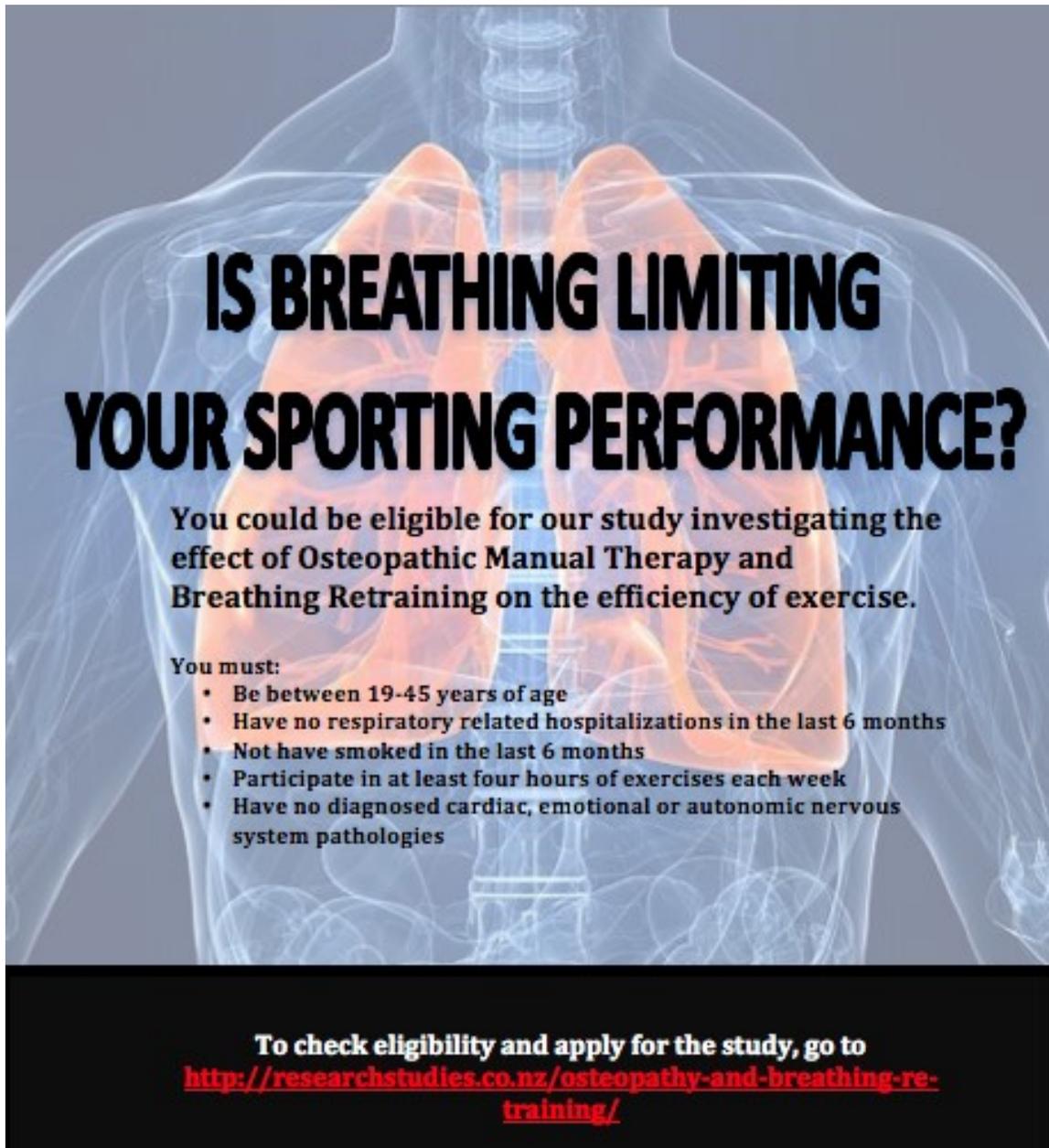
- Emphysema 1, 2. *American Review of Respiratory Disease*, 82(6), 787–791.
- Valta, P., Corbeil, C., Lavoie, A., Campodonico, R., Koulouris, N., Chassé, M., ... Milic-Emili, J. (1994). Detection of expiratory flow limitation during mechanical ventilation. *American Journal of Respiratory and Critical Care Medicine*, 150(5), 1311–1317.
- Van Dixhoorn, J., & Duivenvoorden, H. J. (1985). Efficacy of Nijmegen questionnaire in recognition of the hyperventilation syndrome. *Journal of Psychosomatic Research*, 29(2), 199–206.
- Vickery, R. (2008). *The effect of breathing pattern retraining on performance in competitive cyclists* (Doctoral dissertation). Auckland University of Technology. Retrieved from <http://aut.researchgateway.ac.nz/handle/10292/83>
- Viskoper, R., Shapira, I., Priluck, R., Mindlin, R., Chornia, L., Laszt, A., ... Alter, A. (2003). Nonpharmacologic treatment of resistant hypertensives by device-guided slow breathing exercises. *American Journal of Hypertension*, 16(6), 484–487.
- Vogiatis, I., Athanasopoulos, D., Habazettl, H., Kuebler, W. M., Wagner, H., Roussos, C., ... Zakynthinos, S. (2009). Intercostal muscle blood flow limitation in athletes during maximal exercise. *The Journal of Physiology*, 587(14), 3665–3677.
- Volianitis, S., McConnell, A. K., Koutedakis, Y., McNaughton, L. R., Backx, K., & Jones, D. A. (2001). Inspiratory muscle training improves rowing performance. Retrieved from <http://wlv.openrepository.com/wlv/handle/2436/7229>
- Ward, M. E., Eidelman, D., Stubbing, D. G., Bellemare, F., & Macklem, P. T. (1988). Respiratory sensation and pattern of respiratory muscle activation during diaphragm fatigue. *Journal of Applied Physiology*, 65(5), 2181–2189.
- Ward, S. A. (2007). Ventilatory control in humans: constraints and limitations. *Experimental Physiology*, 92(2), 357–366.
- Wasserman, K., Whipp, B. J., Koyl, S. N., & Beaver, W. L. (1973). Anaerobic threshold and respiratory gas exchange during exercise. *Journal of Applied Physiology*, 35(2), 236–243.
- Westerdahl, E., Lindmark, B., Eriksson, T., Friberg, O., Hedenstierna, G., & Tenling, A. (2005). Deep-breathing exercises reduce atelectasis and improve pulmonary function after coronary artery bypass surgery. *CHEST Journal*, 128(5), 3482–3488.
- Western, P. J., & Patrick, J. M. (1988). Effects of focusing attention on breathing with and without apparatus on the face. *Respiration Physiology*, 72(1), 123–130.
- West, J. B. (2008). *Respiratory Physiology: The Essentials* (Eighth edition). Philadelphia: LWW.

- Wetter, T. J., Harms, C. A., Nelson, W. B., Pegelow, D. F., & Dempsey, J. A. (1999). Influence of respiratory muscle work on VO<sub>2</sub> and leg blood flow during submaximal exercise. *Journal of Applied Physiology*, *87*(2), 643–651.
- Wilkie, S. S., Dominelli, P. B., Sporer, B. C., Koehle, M. S., & Sheel, A. W. (2015). Heliox breathing equally influences respiratory mechanics and cycling performance in trained males and females. *Journal of Applied Physiology*, *118*(3), 255–264.
- Wüthrich, T. U., Notter, D. A., & Spengler, C. M. (2013). Effect of inspiratory muscle fatigue on exercise performance taking into account the fatigue-induced excess respiratory drive: Prior-induced inspiratory muscle fatigue and performance. *Experimental Physiology*, *98*(12), 1705–1717.  
<http://doi.org/10.1113/expphysiol.2013.073635>
- Yoshiga, C. C., & Higuchi, M. (2003). Oxygen uptake and ventilation during rowing and running in females and males. *Scandinavian Journal of Medicine & Science in Sports*, *13*(6), 359–363.

## Appendices:

### Appendix 1

#### Recruitment Poster



The poster features a central image of a human torso with the respiratory system highlighted in orange. The text is overlaid on this image. The main title is in large, bold, black letters. Below it, a paragraph describes the study's focus. A list of eligibility criteria follows, and a call to action is provided at the bottom in a black box with white and red text.

# IS BREATHING LIMITING YOUR SPORTING PERFORMANCE?

You could be eligible for our study investigating the effect of Osteopathic Manual Therapy and Breathing Retraining on the efficiency of exercise.

You must:

- Be between 19-45 years of age
- Have no respiratory related hospitalizations in the last 6 months
- Not have smoked in the last 6 months
- Participate in at least four hours of exercises each week
- Have no diagnosed cardiac, emotional or autonomic nervous system pathologies

To check eligibility and apply for the study, go to  
<http://researchstudies.co.nz/osteopathy-and-breathing-re-training/>

## Appendix 2

### General Information Questionnaire

Name:

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 your 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 emotion disorders or cardiac dysfunctions?	Yes / No
Do you have any autonomic nervous system pathologies?	Yes / No

### Appendix 3

#### Borg (RPE) Scale

Rating	Perceived Exertion
6	No exertion
7	Extremely light
8	
9	Very light
10	
11	Light
12	
13	Somewhat hard
14	
15	Hard
16	
17	Very hard
18	
19	Extremely hard
20	Maximal exertion

Table 1. The Borg Rating of Perceived Exertion Scale

## Appendix 4

### Nijmegen Questionnaire (NQ)

#### 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>					

## Appendix 5

### Self Evaluation of Breathing Questionnaire (SEBQ)

<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 6

### Breathing Retraining Protocol – For practitioners

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, Wesley Verhoeff and Lacey Barnett.**

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 = **N**asal 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 = **Evenness**

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/Sn41aI0qCZg>
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/8Hd73qIpiGM>
10. <http://youtu.be/IH68IbjgDNM>

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 [cbacon@unitec.ac.nz](mailto:cbacon@unitec.ac.nz)

## **Appendix 7**

### **Breathing Retraining protocol – For patients**

#### **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, Wesley Verhoeff and Lacey Barnett.

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 characteristic 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 = **N**asal 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/Sn41aI0qCZg>

13. <http://youtu.be/ykKsyz4W6Gg>

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/8Hd73qIpiGM>

20. <http://youtu.be/IH68IbjgDNM>

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[jaydenbeginheim@gmail.com](mailto:jaydenbeginheim@gmail.com)

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

Catherine Bacon [cbacon@unitec.ac.nz](mailto:cbacon@unitec.ac.nz)

# Appendix 8

## Breathing Retraining Resource Sheet

### Breathing Re-Training Quick Reference

**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. 

**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. 

### Nasal Out Pause Evenness

**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. 

**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  
J. Bergman, R. Moran, M. Vanhoff & C. Barrett

# Appendix 9

# Breathing Retraining Resource Sheet – 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: ‘Out’ breath longer than in breath.

The length of your out breath should be longer than your 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 characteristic 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

Stage 1 Breathing Re-Training Quick Reference Version 1.1

© Benjamin, R. Moore, W. Marshall

## Appendix 10

### 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*

Second, please observe your patient for the following, at the stage indicated above.

*Note: Steps 1 -6 are to be assessed as individual components (a full assessment is not required)*

---

*Notes....*

1. Mouth, nose or both used with both inhalation and exhalation?		
2. Abdomen, thorax or both expanding with inhalation?		
3. Abdomen, thorax or both falling with exhalation?		
4. Longer exhalation or inhalation?		
5. Pause present after exhalation, inhalation or not present?		
6. Consistent or inconsistent of breath rhythm?		
7. Breathing rate appears normal or abnormal?		
8. 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 11

### OMT Assessment & Treatment Protocol

Patient:

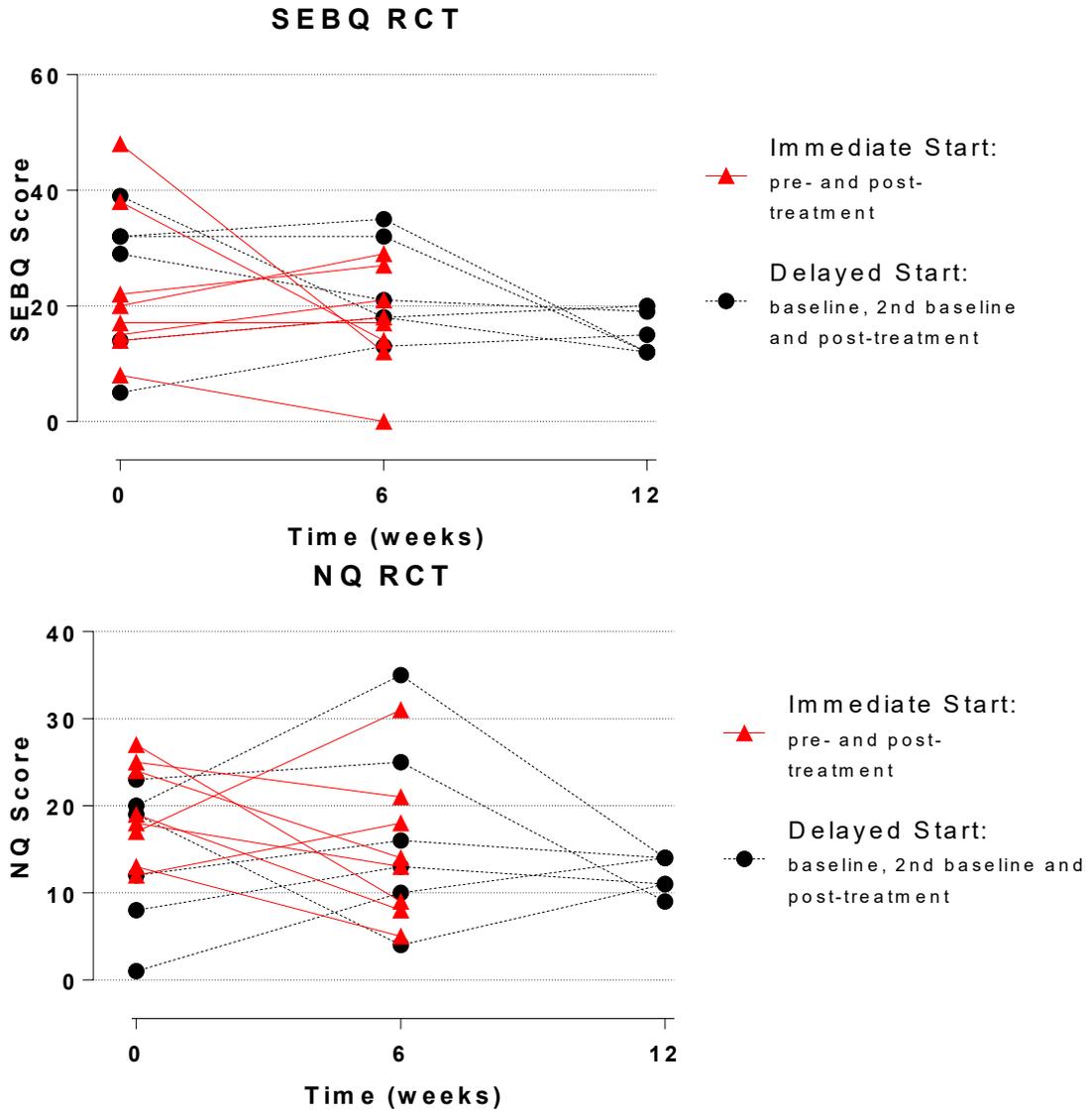
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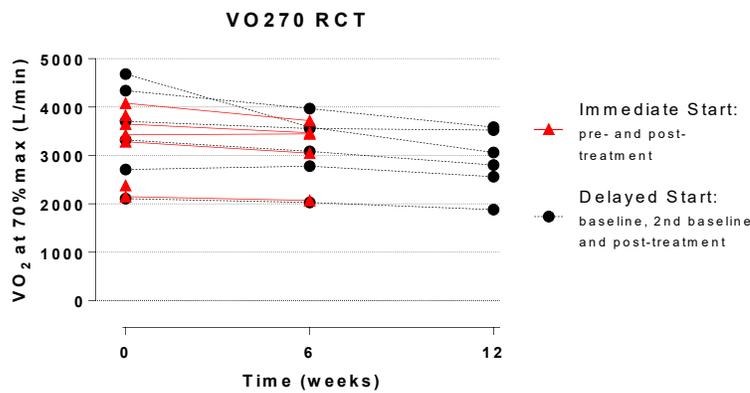
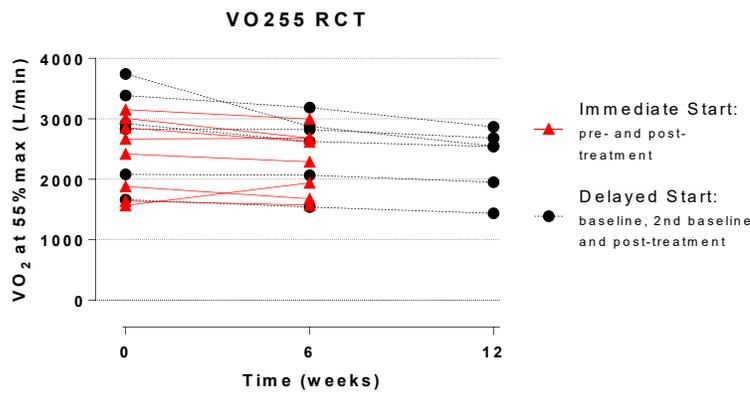
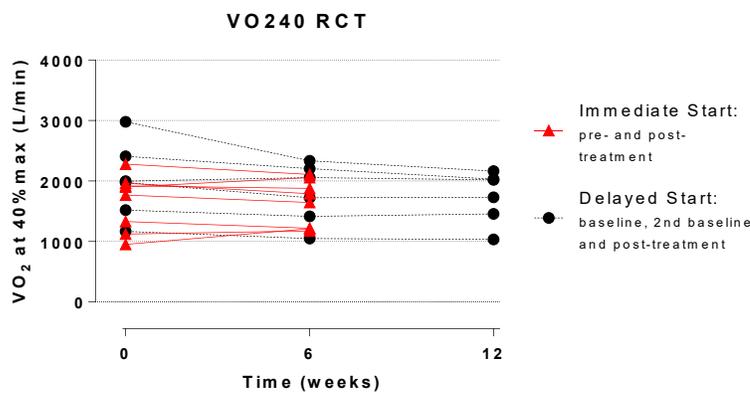
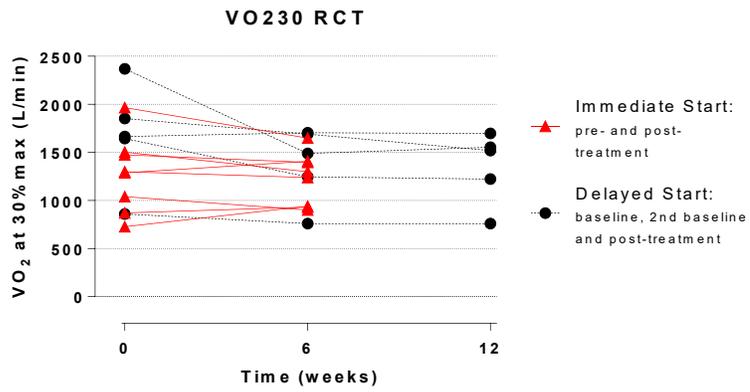
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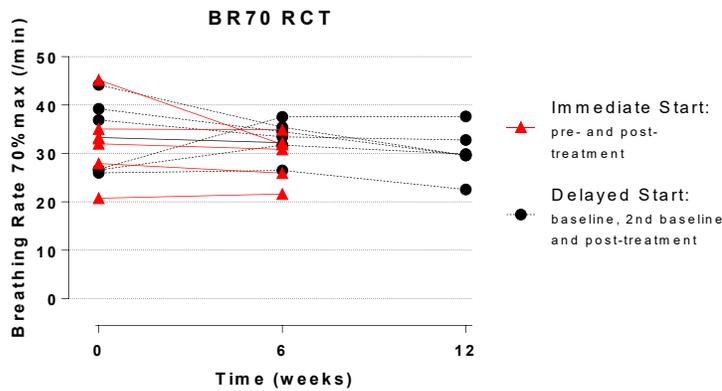
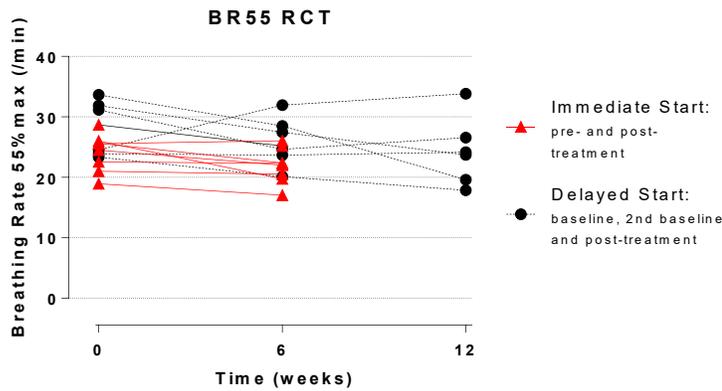
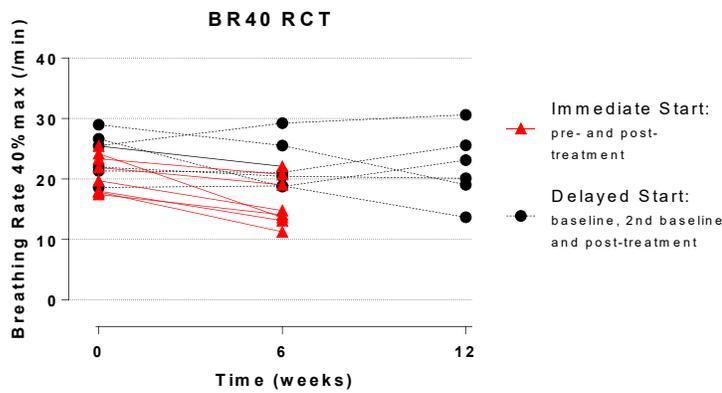
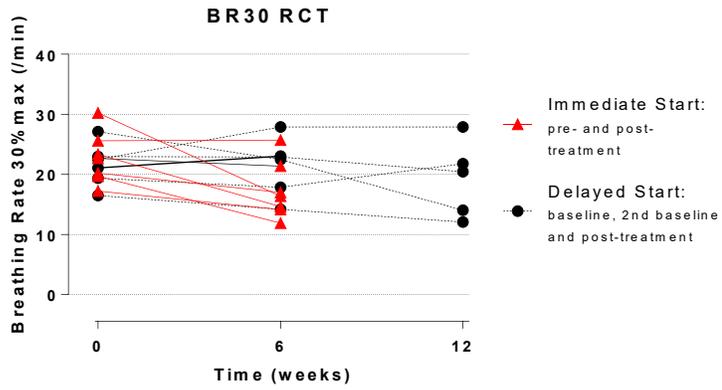
Region to be assessed	Notable Joint/soft tissue of the region	Presence of Dysfunction Y/N? (please circle)	Dysfunction present in? (please circle)	Technique(s) used? (please circle)
Cspine	OA-AA	Y/N T.A.R.T	Observation, Active, Passive, Palpation.	S/T, Inhibition, Myofascial release, M.E.T Manipulation. 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				

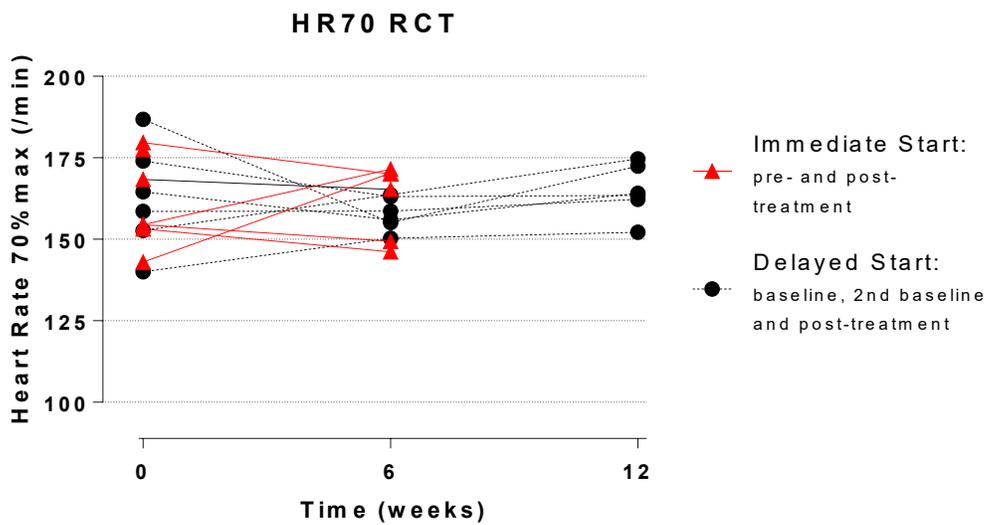
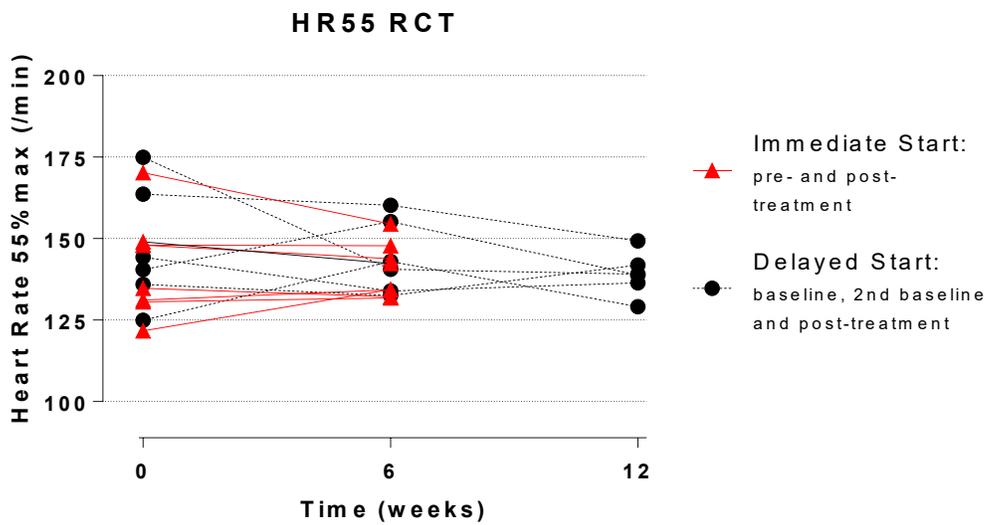
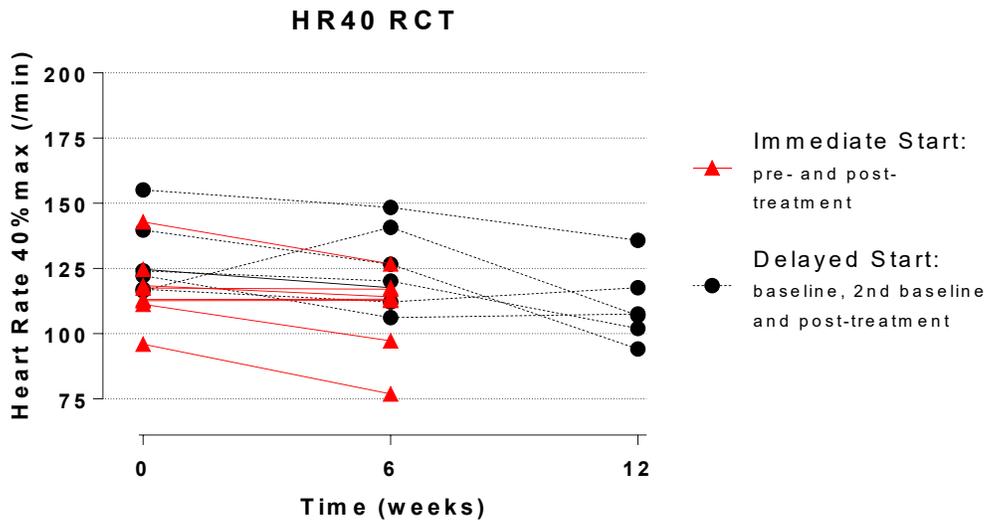
BLT = Balance ligamentous technique, BMT = Balance muscle technique, M.E.T = Muscle energy technique, MWM = Mobilisation with movement, SCS = Strain counter strain, S/T = Soft tissue manipulation, T<sup>1</sup>.A.R.T<sup>2</sup> = Texture, Asymmetry, Range of movement & Tenderness

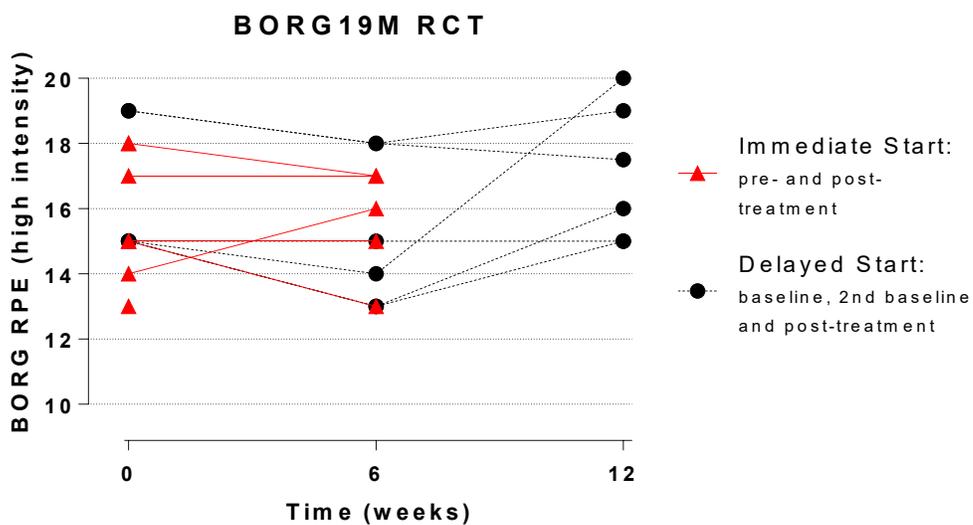
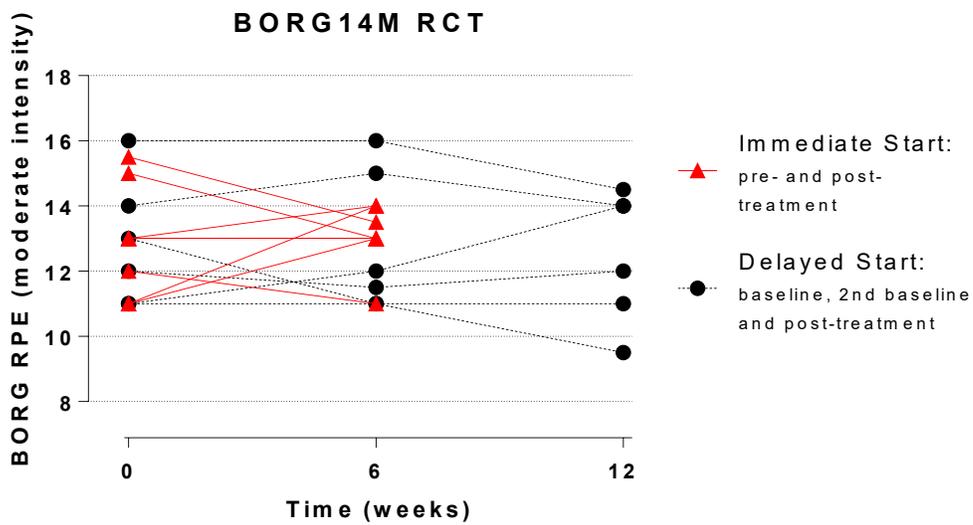
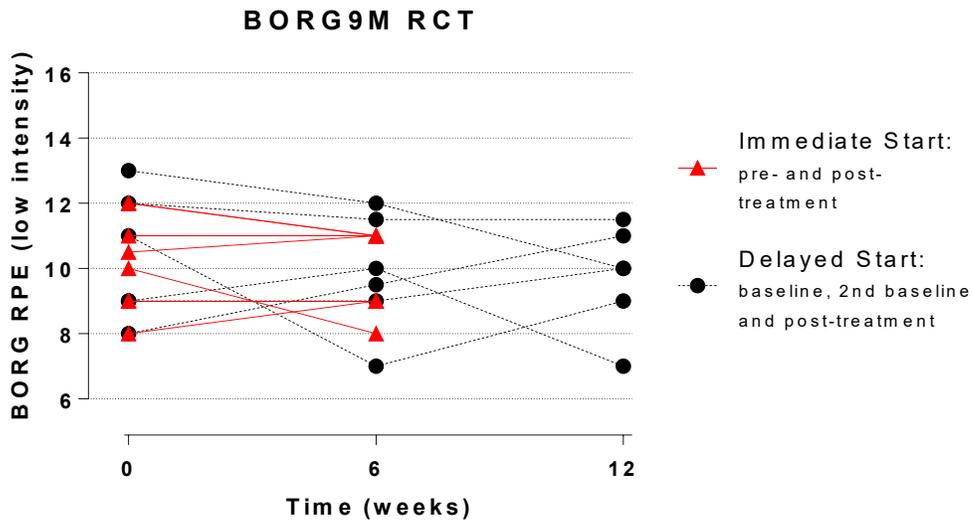
# Appendix 12 Graphed results

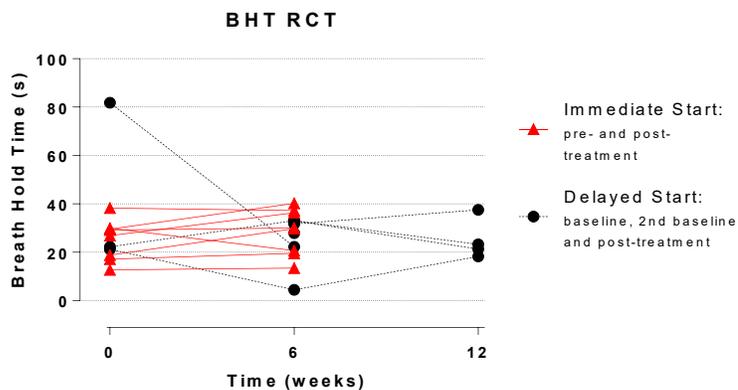
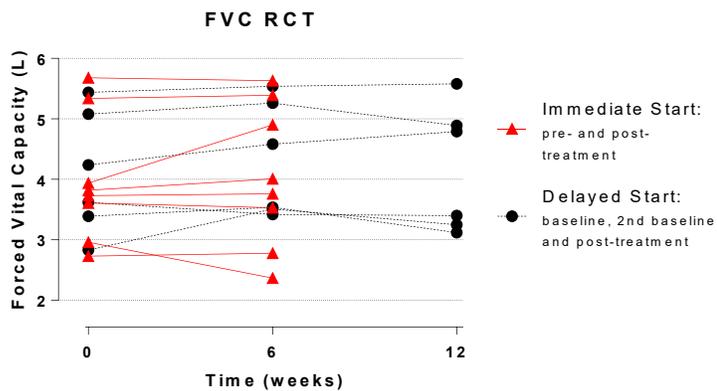
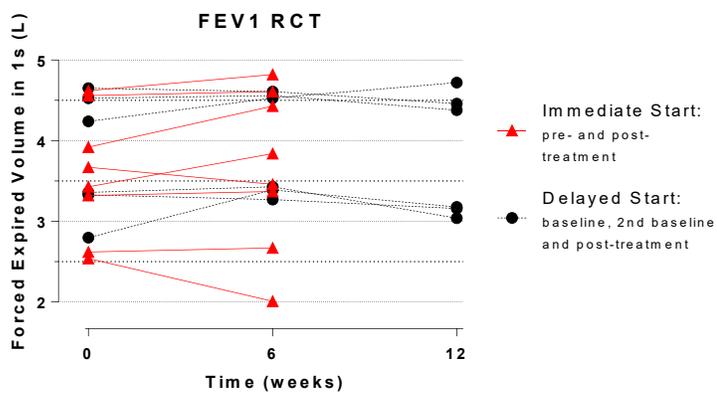
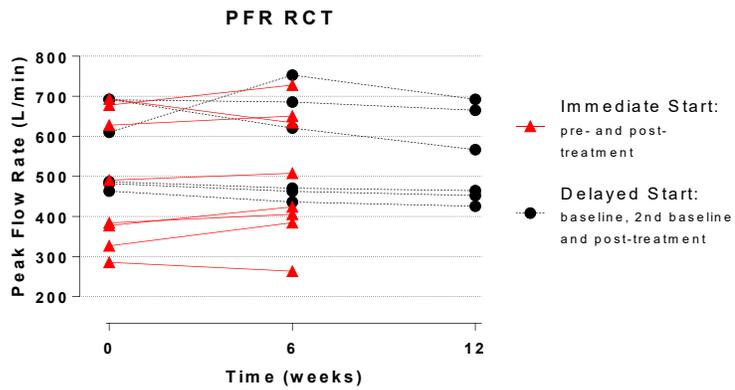














## Declaration

**Name of candidate:** Lacey Barnett

This Thesis/Dissertation/Research Project entitled The Effect of Osteopathic Manual Therapy with Breathing Retraining on Dysfunctional Breathing and Exercise Economy: A Randomised Controlled Trial is submitted in partial fulfilment for the requirements for the Unitec degree of Master of Osteopathy.

### CANDIDATE'S DECLARATION

I confirm that:

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- 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: UREC 2013-1080

Candidate Signature: .....Date: 1/8/16

Student number: 1367726



Full name of author: Lacey Ellen Barnett

ORCID number (Optional): .....

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

The Effect of Osteopathic Manual Therapy with Breathing Retraining on Dysfunctional Breathing and Exercise Economy: A Randomised Controlled Trial

Practice Pathway: osteopathy

Degree: Master of Osteopathy

Year of presentation: 2016

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Associate Supervisor: Robert Moran

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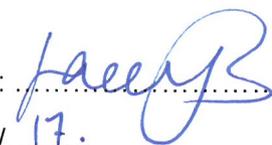
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