The Effect of Breathing Pattern Retraining on Performance in Competitive Cyclists

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Attestation of Authorship

I hereby declare that this submission is my own work and that, to the best of my knowledge and belief, it contains no material previously published or written by another person nor material which to a substantial extent has been accepted for the qualification of any degree or diploma of a university or other institution of higher learning, except where due acknowledgement is made in the acknowledgements.

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Dedication

This thesis is dedicated in the first instance to Neil Dearberg, one of life's true gentlemen, whose love, support, encouragement and patience know no boundaries.

I also dedicate this thesis to my parents, Evan and Heather Vickery, who are not only incredible parents, but are amazing, inspirational people. I am proud to be your daughter.

Acknowledgements

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Abstract

Background: The increased work of breathing associated with intense cycling has been identified as a factor that may negatively affect cycling performance. The aerodynamic position, abnormal respiratory mechanics either at rest or during exercise, and the development of a tachypnoeic breathing pattern are factors known to increase the work of breathing. Breathing pattern retraining aims to decrease the work of breathing by delaying the onset of dynamic hyperinflation and the recruitment of accessory breathing muscles. To date no studies have investigated the performance, physiological and perceptual consequences of manipulating breathing pattern in trained cyclists. **Purpose**: The aim of the present study was to investigate the effect of breathing pattern retraining on 20-km time trial performance and respiratory and metabolic measures in competitive cyclists. **Method:** Twenty-four competitive male cyclists (age 37.7 ± 8.6 years, mean ± SD; $\dot{V}O_{2\,peak}$ 4.34 ± 0.47 L·min^{-1}) were match paired on 20-km time trial performance and assigned at random to either an intervention group (breathing pattern retraining; N = 12) or control group (N = 12). 20-km time trial performance, pulmonary function and the physiological and perceptual response during a maximal incremental cycle step test were assessed pre- and post-intervention. The intervention group underwent four weeks of specific breathing pattern retraining using exercises designed to reduce dynamic hyperinflation and optimise respiratory mechanics. The control group attended the laboratory once a week during this period and performed a 10 minute sub-maximal ride wearing a biofeedback breathing harness. The control group was led to believe the purpose for their participation was to investigate the effect that maximal exercise had on breathing pattern, and to test the reliability of the breathing harness. There was no attempt to modify the breathing pattern of the control group. Data were analysed using an MS Excel spreadsheet designed for statistical analysis. The uncertainty in the effect was expressed as 90% confidence limits and a smallest worthwhile effect of 1.0% was assumed. **Results:** The intervention group showed substantial improvements in 20-km time trial performance (-1.5 ± 1.1%) and incremental power (3.2 ± 3%). Additionally, breathing frequency (-13.2 ± 8.9%; -9.5 ± 8.4%), tidal volume (10.6 ± 8.5%; 9.4 ± 7.6%), inspiratory time (10.1 ± 8%; 9.4 ± 7.7%), breathing RPE (-30 ± 33.9%; -24.7 ± 28.1%) and leg RPE (-27.9 ± 38.5%; -24.7 ± 28.2%) were all positively affected at lactate threshold and lactate turn point. No positive changes were observed in the control group for 20-km time trial performance (0.0 ± 1.0%), incremental power (-1.4 ± 3.5%), breathing frequency (-1.6 ± 8.0%; -2.0 ± 7.9%), tidal volume (0.9 ± 7.2%; 2.9 ± 9.4%), breathing RPE (16.1 ± 50.2%, 24.8 ± 43%) or leg RPE (13.4 ± 39.6%; 19.9 ± 43.2%) . **Conclusion:** These results provide evidence of the performance enhancing effect of four weeks of breathing pattern retraining in cyclists. Furthermore, they suggest breathing pattern can be retrained to exhibit a controlled pattern, without a tachypnoeic shift, during high intensity cycling. Additionally, these results indicate breathing pattern retraining attenuates the respiratory and peripheral perceived effort during incremental exercise.

Key words: Breathing pattern disorders, retraining, blood stealing, cycling, performance, power output, respiratory mechanics, perceived exertion, 20km-TT

Chapter One: Introduction

In the 2004 Olympic men's road cycling time trial (48km), the time difference between winning the gold medal and second place was 18.84 seconds (0.5%). Because small improvements in performance at the top level of cycling can make a significant difference to overall results, cyclists, coaches and sports scientists are continually looking for ways to facilitate these performance gains such as employing new physical training methods (Paton & Hopkins, 2005), consuming nutritional supplements (Wiles, Coleman, Tegerdine, & Swaine, 2006) and manipulating equipment (Belen, Habrard, Micallef, & Le Gallais, 2007). An area that appears to be overlooked is the effect that the mechanics of breathing may have on a cyclist's performance.

Abnormal breathing patterns, or breathing pattern disorders, may be the long term consequence of altered respiratory mechanics. Dysfunctional respiratory mechanics during exercise may be due to:

i)   pathological factors such as exercise-induced asthma, exercise-induced bronchospasm, vocal cord dysfunction, acute respiratory illness;

ii)   emotional factors, such as anxiety;

iii)   biomechanical factors for example the postures adopted by athletes in their sport and/or;

iv)   physiological factors, such as the changes that occur to breathing frequency and tidal volume during the normal ventilatory response to exercise.

Either alone or in combination, these factors may cause the development of a chronic breathing pattern disorder. Of particular interest to this study is the development, and potential

consequences, of a chronically abnormal breathing pattern in cyclists whose breathing pattern becomes maladaptive.

The consequences of a breathing pattern disorder may have a detrimental effect on a cyclist's performance for several reasons. Altered respiratory mechanics during exercise may lead to a tachypnoeic shift (increased breathing frequency at the expense of maintaining tidal volume; Lucia, Carvajal, Calderon, Alfonso, & Chicharro, 1999), increased dynamic hyperinflation (Johnson, Saupe, & Dempsey, 1992; McConnell, 2005) and a subsequent increase in the work of breathing (Harms et al., 1998). Consequently, an increased work of breathing may cause blood stealing (Sheel et al., 2001), a phenomenon where blood is diverted from working peripheral muscles to the respiratory muscles to meet increased demand. Such decreased blood flow may negatively affect the ability of the peripheral muscles to generate power (Romer & Dempsey, 2006). Additionally, an increased work of breathing can result in respiratory muscle fatigue (Johnson, Aaron, Babcock, & Dempsey, 1996) and increased dyspnoea (Grazzini, Stendardi, Gigliotti, & Scano, 2005), both of which may have an adverse affect on performance.

Traditionally, the idea that breathing may be a limiting factor in high intensity exercise has been disregarded due to the apparent breathing reserve observed in athletes even at the highest level of exertion (Dempsey, 1986). However, with evidence suggesting expiratory flow limitation may occur in elite athletes (Johnson, Saupe, & Dempsey, 1992) and that the work of breathing during intense exercise may cause blood stealing (Harms, Wetter, St Croix, Pegelow, & Dempsey, 2000; Sheel et al., 2001), attention has been brought to the possibility that breathing may be a limitation to performance in athletes (Dempsey, 2006b). The apparent success of techniques such as inspiratory muscle training (IMT) (Dellaca et al., 2001; Romer, McConnell, & Jones, 2002a) and

voluntary isocapnic hyperpnoea (VIH) (Holm, Sattler, & Fregosi, 2004; Leddy et al., 2007; McMahon, Boutellier, Smith, & Spengler, 2002) in improving performance in endurance sport suggests that specific intervention strategies can be employed to overcome this limitation. Although strong evidence exists to suggest that IMT is effective in increasing performance by increasing the strength and endurance of the respiratory muscles and reducing inspiratory muscle fatigue (Romer, McConnell, & Jones, 2002a, , 2002b; Volianitis et al., 2001), IMT in itself does not address the pattern of breathing. This could be likened to increasing the leg strength and power of a cyclist, without addressing pedalling technique.

The ability to maintain an even, controlled breathing pattern during high exercise intensities, without defaulting to a tachypnoeic shift, has been identified as an important characteristic of professional cyclists (Lucia, Carvajal, Calderon, Alfonso, & Chicharro, 1999). It is not known, however, whether this breathing pattern is a physiological adaptation (e.g. to years of high intensity training,) or whether it could be likened to a learned skill and could therefore be manipulated.

Breathing pattern retraining is a technique used by physiotherapists to correct abnormal respiratory mechanics and breathing pattern disorders (Blager, 2000; Chaitow, Bradley, & Gilbert, 2002; Jones, Dean, & Chow, 2003). Although used typically in the management of patients with respiratory pathology, the concept and application is similar for athletes. Breathing pattern retraining may benefit cyclists in two ways - firstly by correcting any abnormal respiratory mechanics or breathing pattern disorders that are present at rest, and secondly by attenuating the consequences of poor respiratory mechanics that often occur during intense exercise (Fallon, 2004; 2007).

Whilst breathing pattern retraining techniques are used clinically by physiotherapists to address breathing pattern disorders in athletes, there are no known published studies documenting the ergogenic potential of this technique in this population. Further, the effect that optimising respiratory mechanics may have on exercise performance in cyclists, by attempting to attenuate the negative consequences of an increased work of breathing, has not been reported. The aim of Chapter Two (Review of Literature) is to introduce the reader to the concept and consequences of abnormal breathing patterns in athletes and to discuss how breathing pattern retraining may not only improve the efficiency of breathing in cyclists, but also facilitate increased cycling performance. In consideration of how or why breathing pattern retraining may potentially enhance cycling performance, a brief description of normal and abnormal breathing patterns is provided and the prevalence of breathing disorders in athletes will be discussed. In addition, the theories and evidence that underpins the ventilatory response to exercise, subsequent changes to the work of breathing and consequences of this, will be presented. Finally, strategies that may decrease the effect of altered respiratory mechanics during exercise, including respiratory muscle training and breathing pattern retraining, will be explored.

Using breathing pattern retraining to manipulate breathing in athletes is a novel area of research. Indeed there is a paucity of literature that objectively defines breathing pattern retraining and its effectiveness in addressing breathing pattern disorders. There is currently no evidence to either support or refute its effectiveness as a performance enhancing technique in athletic populations. The aim of this thesis is to address these gaps by investigating the use of breathing pattern retraining techniques in cyclists.

1.1. Aim of The Study

The aim of this study was to investigate the effect of four weeks of breathing pattern retraining on 20-km time-trial performance, and on respiratory and metabolic measures during an incremental step test in competitive cyclists.

Chapter Two: Literature Review. The Development, Consequence and Management of Abnormal Respiratory Mechanics in Athletes

2.1. Cycling Performance, Ventilation and Breathing

The difference between winning an Olympic gold medal and second place in the men's 48km time trial in 2004 was 18.84 seconds (0.5%) (www.cyclingnews.com). Even the smallest improvement in performance at the top level can make a significant difference to the overall result (Jeukendrup & Martin, 2001). Cycling performance is dependent on physiological factors which influence mechanical power production (for example training modification, nutrition and possibly breathing pattern) (Lucia et al., 1999), in addition to mechanical and environmental factors that affect power demand (for example body mass, body position, bicycle) (Jeukendrup & Martin, 2001). These factors are even more important in an individual time trial (TT) where race tactics play a lesser part (Jeukendrup & Martin, 2001) and physical performance factors come to the fore. For example, the effects of modified training on a 40km-TT in already well-trained individuals has been reported to be 2-4% (Jeukendrup & Martin, 2001) which could make a difference of 70-140 seconds over 40km. This time difference applied to the 2004 Olympic results described above would be the difference between first and fourteenth place (www.cyclingnews.com).

Competitive road cyclists may take part in a one day race, or multistage events which are characterised by back-to-back days of racing including mass start races, individual and team time trials. Of all the road race formats, the individual time trial is considered the most physically demanding (Mujika & Padilla, 2001) and is a component of major tour races (e.g. the Tour de France), or in some cases is a stand-alone event (e.g. World Championships and Olympic Games). An individual time trial demands each cyclist cover the required distance in the shortest

possible time by sustaining the highest average power output that will allow them to gain a time advantage over their opponents (McDonald, 2007).

Mass start races allow drafting, which means a cyclist can ride in the slipstream of the rider(s) ahead to decrease their resistance (Faria, Parker, & Faria, 2005b). Drafting can decrease energy utilization by up to 40% (Faria, Parker, & Faria, 2005b), however drafting is not allowed in time trial events, meaning cyclists must be able to sustain high energy outputs for prolonged periods. Although many factors influence cycling performance, including years of training (Coyle et al., 1991), cycling economy (Lucia, Hoyos, & Perez, 2002) and $\dot{V}O_{2\,max}$ (Lucia et al., 2006), power output at lactate threshold and peak power output indicating a power-to-weight ratio of greater or equal to 5.5W/kg have been suggested to be the physiological markers most predictive of cycling performance (Faria, Parker, & Faria, 2005).

In an attempt to make small improvements to performance, numerous studies have investigated ways in which power output can be maximised in cyclists. These include hypoxic training (Clark et al., 2007), recovery strategies (Maxwell, Castle, & Spencer, 2007), aerodynamics (Grappe, Candau, & Belli, 1997) and inspiratory muscle training (Johnson, Sharpe, & Brown, 2007). Whilst attention has been paid to ventilatory aspects, including the changes that occur in breathing pattern during exercise (Dempsey, 2006a; Lucia, Carvajal, Calderon, Alfonso, & Chicharro, 1999; Lucia, Hoyos, & Perez, 2002; Lucia et al., 2006), no attention has been paid to manipulating breathing pattern on a long term basis. Given the reported high prevalence of breathing disorders in athletes (Fallon, 2004; Mendelli, Lounana, Messan, Menuet, & Petitjean, 2006; Rundell & Spiering, 2003) and the changes to respiratory mechanics that occur during exercise (Johnson,

Saupe, & Dempsey, 1992; Verges, Notter, & Spengler, 2006; Ward, 2007), it is surprising that no study has focused on the potential improvements in cycling work output and performance that can be made as a result of a breathing pattern intervention, or by improving a maladaptive breathing pattern which is commonly induced during high intensity exercise.

2.2. Breathing Pattern Disorders and Abnormal Respiratory Mechanics

A breathing pattern disorder (BPD) is a general term used to describe the consequences of long-standing abnormal respiratory mechanics. Little is published in the medical and scientific literature describing BPDs; what they are, how they are established and how they affect otherwise healthy individuals. Even less is known about how BPDs and abnormal respiratory mechanics may affect athletes and, specifically to this review, cyclists. Breathing in cyclists may be compromised in a number of ways (e.g. altered mechanics of breathing caused by the cyclists unique exercising posture, changes in respiratory timing and/or muscle recruitment pattern due to the normal physiological response to exercise, ventilation-perfusion mismatching, diffusion limitations and increased intra- and post-pulmonary shunt) (Dempsey & Wagner, 1999; Stickland & Lovering, 2006), and while most of these are beyond the scope of this review, it is possible that with regard to respiratory mechanics, a cyclist's breathing may be compromised by the presence of a breathing pattern disorder at rest, or the changes to respiratory timing and respiratory muscle recruitment pattern that occurs with the normal respiratory response to exercise. To gain further understanding of the aetiology of BPDs, this section will focus on the mechanics of normal and abnormal breathing at rest and during exercise. The prevalence of abnormal breathing patterns in athletes and contributing factors will also be discussed.

2.2.1. Normal Breathing at Rest

Breathing at rest, in people without respiratory illness, requires less than five percent of whole-body oxygen consumption ($\dot{V}O_2$) (Jones, Dean, & Chow, 2003). This proportion of $\dot{V}O_2$ enables the respiratory muscles to generate appropriate pressure changes within the thoracic cavity to overcome a combination of airway resistance and lung compliance, resulting in inspiration. As the primary muscle of inspiration, the diaphragm is normally responsible for 95% of the work of quiet, tidal breathing, with minimal contribution from the accessory muscles (Faithfull, Jones, & Jordan, 1979) (*Table 1*).

Table 1. Muscles and Structures of Respiration (From Chaitow et al., 2002)

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

Assuming optimal respiratory mechanics, the diaphragm descends into the abdominal cavity during inspiration, pushing the abdominal contents downwards and forwards, increasing the vertical dimensions of the chest cavity, as the ribs are lifted and move out to increase the transverse dimensions (Chaitow & Bradley, 2002). Expiration during quiet breathing is normally passive and as the diaphragm relaxes the abdomen and chest wall return to their starting position.

Minute ventilation (V_E) is the volume of gas ventilated in one minute and is the product of tidal volume and respiratory rate. The primary aim of V_E is to meet the metabolic demands of the body by moving oxygen (O_2) into and carbon dioxide (CO_2) out of the lungs in the most efficient way (Abernethy, Mackinnon, Neal, Kippers, & Hanrahan, 1996). It is suggested that meeting the metabolic demands of the body at rest (which requires approximately 6 L•min-1; Webber & Pryor, 1994) in the most efficient way, is achieved using a steady, rhythmical pattern of the diaphragm; a respiratory rate of 10-14 breaths·min^{-1}; and an inspiratory to expiratory ratio of 1:1.5-2 (Chaitow & Bradley, 2002). Ideally, a person naturally adopts an optimal breathing pattern that requires the least amount of mechanical stress and work from the respiratory musculature (Jones, Dean, & Chow, 2003). However in some instances this optimal pattern can be disrupted such that abnormal, and potentially inefficient, respiratory mechanics become the default pattern.

2.2.2. Abnormal Breathing at Rest

According to Chaitow et al. (2002) abnormal respiratory mechanics in a healthy human at rest have been defined as:

i) breathing to the upper chest (apical breathing);

ii) a breathing frequency (f_b) of greater than 16 breaths·min^{-1};

iii) an inspiratory to expiratory ratio of greater than 1.5:1;

iv) chronic mouth breathing, and;

v) absence of the end-expiratory pause.

Abnormal respiratory mechanics may occur as the result of, but not limited to, an acute or chronic illness, prolonged exposure to stressful situations, occupational factors (for example occupations

that require large amounts of talking), or poor posture (Blager, 2000; Chaitow, Bradley, & Gilbert, 2002).

One consequence of abnormal respiratory mechanics, especially an apical pattern with an increased f_b, is an acute decrease in the level of CO_2 in the bloodstream (Garssen, de Ruiter, & van Dyke, 1992). The physiology underpinning how breathing pattern may be altered chronically is not known. One theory that has been presented however, is related to the body's sensitivity to CO_2 (Garssen, de Ruiter, & van Dyke, 1992; Gilbert, 2005). Whilst the body has compensatory strategies to buffer the decreased CO_2 in the short term, it is thought that if abnormal respiratory mechanics persist chronically, the brainstem respiratory centres are 'reset' to respond to the new, decreased level (Garssen, de Ruiter, & van Dyke, 1992). Accordingly, the centrally mediated automatic breathing pattern is 'reprogrammed' such that an apical breathing pattern with an elevated f_b is accepted as 'normal' and a decreased level of circulating blood CO_2 is maintained. Consequently, a chronic BPD is established. Symptoms and signs of a BPD can include shortness of breath inappropriate to the level of activity, difficulty inhaling during exercise, inspiratory wheeze or stridor, air hunger, chest wall pain and heart palpitations (Chaitow & Bradley, 2002; Fallon, 2004). It is not known why some people develop BPDs whilst others exposed to the same situation or environment do not.

Diagnosis of a BPD is usually made by clinical observation comparing a patient's breathing pattern with 'normal' respiratory mechanics (Chaitow, Bradley, & Gilbert, 2002). Attention is paid to a patient's breathing frequency, whether they are breathing apically or diaphragmatically, via their mouth or nose, the degree to which their breathing pattern is rhythmical and the presence of frequent sighs, yawns and/or abdominal splinting (Chaitow & Bradley, 2002). There is currently

no formal, objective, reliable and valid single test to clinically diagnose abnormal respiratory mechanics and a BPD. Spirometry can give an indication of compromised respiratory mechanics, for example airflow limitations, as can a bronchoscopy, however both of these tests require a patient to be symptomatic at the time of testing (Blager, 2000). The Nijmegen questionnaire (Appendix 1) has been used as a supportive diagnostic tool for identifying breathing pattern disorders in research studies (Han, Stegen, De Valck, Clement, & Van de Woestijne, 1996; Thomas et al., 2003), and is used by some clinicians to confirm diagnosis of hyperventilation syndrome (HVS) (Chaitow, Bradley, & Gilbert, 2002). This noninvasive questionnaire identifies anxiety related breathing pattern disorders. It has a sensitivity of 91% and a specificity of 95% in patients with a clinical diagnosis of HVS, and enables patients to see the widespread nature of symptoms and progress with treatment (Van Dixhoorn & Duivenvoorden, 1984).

The prevalence of abnormal respiratory mechanics and BPDs in athletes, in the absence of respiratory pathology, has not been reported in the literature. It would be reasonable to speculate that if abnormal respiratory mechanics and/or BPDs are present in athletes at rest, the effect and consequences of such might be exaggerated during exercise due to the increased physiological demands upon the respiratory, cardiovascular and musculoskeletal systems during exercise. Before addressing these factors in more detail, a brief review of the normal respiratory response to exercise is provided.

2.2.3. Respiratory Response to Exercise

The increased metabolic demands experienced by the body during exercise must be met by significantly increasing V_E (Gallagher, Brown, & Younes, 1987). Compared to untrained individuals who elicit a V_E of 112 ± 19.9 L·min^{-1} (Sheel et al., 2006) during maximal intensity

exercise (V_{Emax}), studies of top level athletes have demonstrated that V_E can increase to 190 L$\cdot$min^{-1} in cycling (Harms, Wetter, St Croix, Pegelow, & Dempsey, 2000), and often above 200 L$\cdot$min^{-1} in rowers (Sheel, Lama, Potvin, Coutts, & McKenzie, 1996), sometimes peaking as high as 270 L$\cdot$min^{-1} (Shephard, 1998). To achieve a breathing pattern that will enable the ventilatory demand of high intensity exercise to be met usually causes an athlete's respiratory pattern to shift from diaphragmatic via the nose, to apical via the mouth in an attempt to shift larger volumes of air faster (Bradley, 2002). The workload at which this occurs has not been documented in the literature. It is possible that this shift in pattern corresponds with the respiratory compensation point – the onset of hyperventilation during incremental exercise that is thought to represent the body's attempt to compensate for metabolic acidosis (Meyer, Faude, Urhausen, & Kindermann, 2004). During exercise V_E can be met by increasing f_b and tidal volume (V_T) and it is not known whether the shift from diaphragmatic to apical breathing is a physiological necessity, or a 'learned habit'. Further, an optimal breathing pattern during exercise (as opposed to the 'normal physiological response') has not been identified.

To achieve such large increases in V_E, the normal respiratory response to exercise is to increase the depth of breathing (increased V_T) and as the workload further increases, by increasing f_b (Gallagher, Brown, & Younes, 1987). The initial increase in V_T may cause the end-expiratory lung volume (EELV) to decrease below tidal EELV (Johnson, Saupe, & Dempsey, 1992). This lengthens the diaphragm and other inspiratory muscles at end-expiration, allowing them to operate near their optimal length for force generation as exercise intensity increases (Ward, Eidelman, & Stubbing, 1988). In addition, a decreased EELV (below functional residual volume) may increase the proportion of "passive" inspiration due to the elastic recoil of the chest wall,

further enhancing the attainment of a high V_E whilst preserving respiratory muscle length (Verges, Notter, & Spengler, 2006).

As ventilatory demands increase further, f_b continues to rise, and V_T will either plateau at approximately 50-60% of vital capacity (Clark, Hagerman, & Gelfand, 1983; Hey, Lloyd, Cunningham, Jukes, & Bolton, 1966) or start to decrease (Spengler, Knopfli-Lenzin, Birchler, Trapletti & Boutellier, 2000; Gallagher, Brown & Younes, 1987). Potentially EELV may increase above resting levels resulting in dynamic hyperinflation of the lungs (Johnson, Saupe, & Dempsey, 1992). Although dynamic hyperinflation permits increases in expiratory flow rates (Miller et al., 2005), which may assist the attainment of the high V_E necessary during maximal exercise, it comes at the expense of an increased work of breathing (the effect of which will be discussed later in this review) (Witt, Guenette, Rupert, McKenzie, & Sheel, 2007).

It has been observed that as exercise intensity rises, f_b increases at the expense of maintaining V_T (this is commonly referred to as a tachypnoeic shift or a tachypnoeic pattern) and an irregular, inefficient pattern develops (Gallagher, Brown, & Younes, 1987; Scheuermann & Kowalchuk, 1999). A tachypnoeic shift is considered to be highly inefficient, both mechanically and metabolically (Lucia, Carvajal, Calderon, Alfonso, & Chicharro, 1999). The likely consequence of a tachypnoeic shift is dynamic hyperinflation due to the decreased expiratory time and subsequent breath stacking which ultimately increases the work of breathing caused by breathing at increased lung volumes. Further, V_E may remain unchanged despite further changes to f_b and V_T, whilst the amount of O_2 actually reaching the alveoli for gas exchange decreases due to increased dead space volume (Hanson, Claremont, Dempsey, & Reddan, 1982) (*Table 2*).

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Table 2. Effect of Breathing Rate and Depth on Alveolar Ventilation in Three Scenarios (from Marieb, 1992)

Breathing pattern	Dead space volume	Tidal volume (TV)	Respiratory rate	Minute respiratory volume	Alveolar ventilation rate	% of TV = dead space volume
I - Normal rate and depth	150ml	500ml	20/min	10,000 ml/min	7000 ml/min	30%
II - Slow, deep breathing	150ml	1000ml	10/min	10,000 ml/min	8500 ml/min	15%
III - Rapid, shallow breathing	150ml	250ml	40/min	10,000 ml/min	4000 ml/min	60%

It could be suggested that the ability to maintain an even, controlled breathing pattern, rather than allowing a tachypnoeic shift to develop, would be advantageous to athletes during high exercise intensities. Given the significant increase in V_E (often 10-15 times resting; Johnson, Aaron, Babcock, & Dempsey, 1996) that must be achieved during maximal exercise, it would seem advantageous to achieve this in the most efficient way. Certainly, it appears that some athletes can maintain an effective respiratory pattern (as defined by lack of tachypnoeic shift; Lucia, Carvajal, Calderon, Alfonso, & Chicharro, 1999) whilst others do not. In the few studies that have compared the respiratory pattern of different levels of athletes, it appears that only the very highly trained or experienced athletes demonstrate this desired characteristic (Lucia, Carvajal, Calderon, Alfonso, & Chicharro, 1999; Mahler, Shuhart, Brew, & Stukel, 1991; Vaughan, 1984). More specifically, a clear difference in respiratory pattern has been observed between professional and non professional cyclists (Lucia, Carvajal, Calderon, Alfonso, & Chicharro, 1999) at high exercise intensities. Lucia et al. (1999) compared f_b, V_T, V_E, inspiratory and expiratory time in 14 professional Tour de France level male road cyclists ($\dot{V}O_{2\ peak}$: 73.2 ± 6.6 ml·kg·min^{-1}, P_{max}: 471 ± 30.9W) and 11 amateur, elite male road cyclists ($\dot{V}O_{2\ peak}$: 73.8 ± 5 ml·kg·min^{-1}, P_{max}: 437 ± 25.2W) during incremental exercise from sub-maximal to maximal intensities (exhaustion). The

V_{Emax} did not differ between the two groups (170 vs. 175 L·min^{-1}, Figure 1), although at intensities between 300 and 400W professional cyclists exhibited a 15% (P < 0.01) lower V_E than elites. At power outputs less than 200W f_b tended to be higher in professionals than elite cyclists (for example 25 versus 20 breaths·min^{-1}; P < 0.05 at 100W, Figure 1) but from 200W to maximum intensities the elite group had a significantly higher f_b than professionals (58 vs. 51 breaths·min^{-1} at maximal; P < 0.05, Figure 1). Inspiratory time reached a plateau in professionals at low intensities (300W; 63.7% P_{max}) and then decreased with increasing exercise intensity, whereas it consistently decreased in elite from the lowest to the highest intensities (50 to 437.5W; Figure 2). Expiratory time was significantly higher in professionals than elites at most moderate-to-high power outputs (P < 0.05 for 250 and 300W, and P < 0.01 for 350 and 400W, Figure 2). These findings indicate that although the maximum volume of air delivered to the lungs was not statistically different between the two groups, professional cyclists did not adopt a tachypnoeic shift to maintain their high V_E.
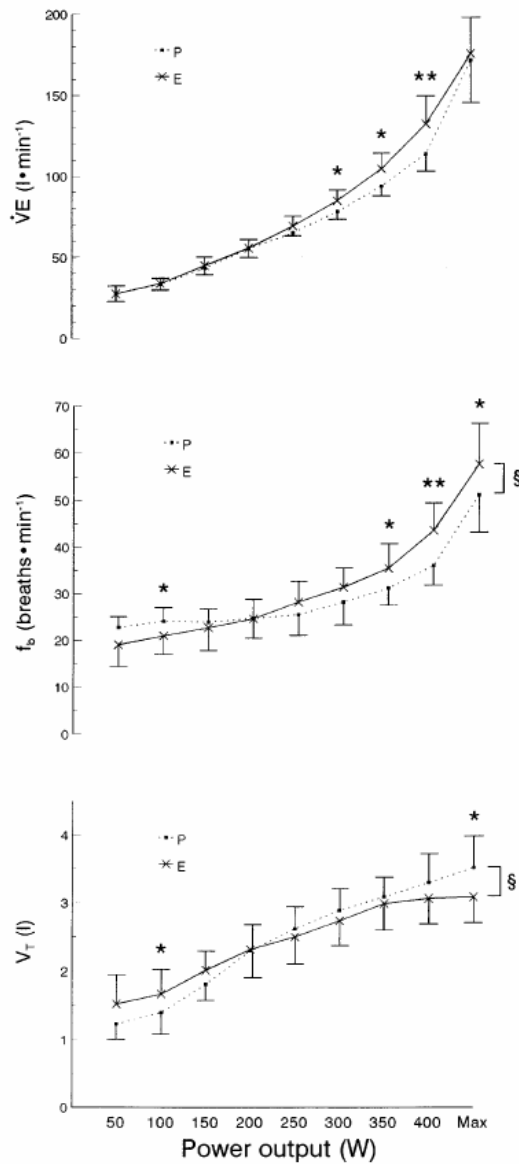
Figure 1. Ventilatory response of the elite (E) and professional (P) cyclists during the tests: pulmonary ventilation (V_E), breathing frequency (f_b) and) tidal volume (V_T). Max Maximal power output. * P < 0.05 ** P < 0.01, § significant (P < 0.05) interactive effect (group x exercise intensity) (From Lucia et al., 1999).
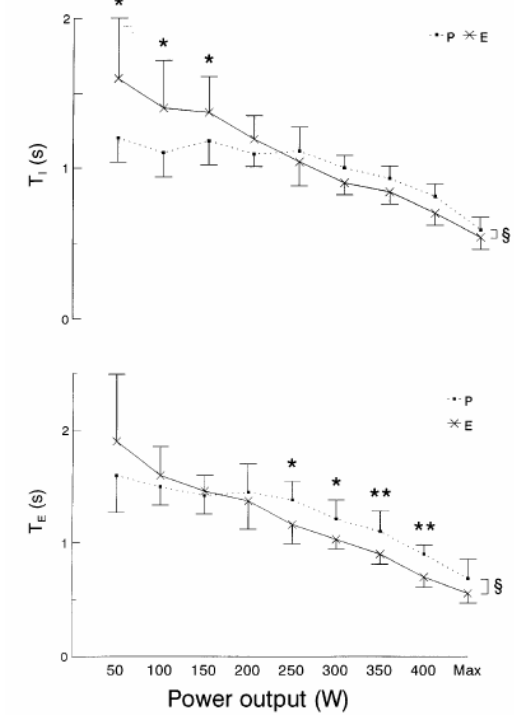
Figure 2. Inspiratory (t_I) and expiratory (t_E) times. Definitions as for Figure .1. * P < 0.05, ** P < 0.01, § significant (P < 0.05) interactive effect (group x exercise intensity. (From Lucia et al., 1999).

It should be acknowledged that the results of Lucia et al. (1999) were obtained in a laboratory environment. The subjects breathing was measured using a mouth piece and nose clip which have been shown to influence the breathing pattern in normal subjects by causing an increase in V_T while f_b is unchanged or decreased (Askanazi et al., 1980). Lucia et al. (1999) believed their

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subjects, having participated in previous studies using the same respiratory apparatus, were familiar with the equipment and it would have been unlikely to have affected their breathing. Additionally, both groups of cyclists were tested using the same equipment meaning both groups would have been affected in the same way. Lucia et al. (1999) set the cadence for their subjects, meaning exercise intensity and power could only be generated by increasing the force applied to each pedal stroke, whereas in field conditions cyclists increase power by increasing the cadence in addition to the force applied to each stroke (Conconi et al., 1996). Lucia et al. (1999) acknowledge that in real cycling conditions at high intensities, increasing cadences might alter breathing patterns through the sensory input from muscle mechanoreceptors to the respiratory centre, which could result in a higher f_b than they observed.

In sports with a rhythmic component such as rowing, respiration is coordinated with the action of the locomotor system (entrainment) to maximise respiratory efficiency, however entrainment has only been observed in high level rowers (Mahler, Shuhart, Brew, & Stukel, 1991; Vaughan, 1984). Mahler et al. (1991) compared the respiratory pattern of 16 novice and 18 experienced rowers and found entrainment at maximal effort in 78% of elite female rowers, but only in 30% of novices. Similar to the professional cyclists in Lucia et al.'s (1999) study, the elite rowers maintained a respiratory pattern that was void of a tachypnoeic shift, whilst the novice rowers increased ventilation by continuing to increase f_b and decrease V_T until exhaustion.

It is possible that maintaining optimal levels of effective ventilation is one factor that enables athletes to achieve levels of performance necessary to excel in their own sport. Lucia et al. (1999) speculated that a breathing pattern devoid of a tachypnoeic shift, and with a prolonged t_E, is required to perform at the highest level of cycling. Currently, there is insufficient research to

identify which physiological characteristics enable professional cyclists to breathe more efficiently, whether it is a genetic predisposition, or whether their ability to breathe efficiently is an independent factor that could be likened to a learned technique or skill that optimises performance. In an attempt to explain the observed differences in breathing patterns between professional and elite athletes, Lucia (1999) initially suggested that physiological factors related to the more demanding training carried out by the professional, as compared with the elite, cyclists was responsible. However, in a subsequent longitudinal study, Lucia et al. (2001) later demonstrated that a professional cyclist's breathing pattern did not vary during the three periods (rest, pre-competition and competition) of a competitive cycling season, despite the fact that a significant overall training effect on power output and $\dot{V}O_2$ was shown throughout the season. These findings suggest that training volume was not the causative factor, or if it was, it possibly occurred over years of training, and was not influenced by the differences occurring throughout a professional cyclist's season. Another suggestion is that the high f_b and decreased V_T often observed at high intensity exercise (Clark, Hagerman, & Gelfand, 1983; Folinsbee, Wallace, Bedi, & Horvath, 1983; Gallagher, Brown, & Younes, 1987) could simply be attributed to respiratory muscle fatigue, and that high level athletes develop a superior level of resistance to such fatigue due to their greater years of experience and volume of training (Syabbalo, Krishnan, Zintel, & Gallagher, 1994). In a group of competitive, but not professional, cyclists, respiratory muscle fatigue (measured by inspiratory pressure at zero flow) has been recorded at $17 \pm 4\%$ and $13 \pm 3\%$ following a 20 and 40km-TT respectively (Romer et al., 2002). The consequences of respiratory muscle fatigue will be discussed further in section 2.3.5.

In contrast, one theory that has been supported across decades (Hey, Lloyd, Cunningham, Jukes, & Bolton, 1966; Scheuermann & Kowalchuk, 1999) is that the integration of the depth and frequency of breathing required to achieve a given ventilatory demand appears to be independent of known respiratory stimuli, and may simply reflect a pattern of breathing that results in a minimal amount of work by the respiratory muscles. This theory may support the suggestion that breathing pattern may be a learned skill and could therefore be retrained. Clearly, experimental work is needed to determine whether an efficient breathing pattern can be taught, or whether it is an adaptive response secondary to changes in other physiological variables after training.

2.2.4. Abnormal Breathing Patterns in Athletes

Ventilation during exercise may be compromised in athletes for a number of reasons including, but not exclusively, mechanistic (ventilation-perfusion mismatching, diffusion limitations and increased intra- and post-pulmonary shunt (Dempsey & Wagner, 1999; Stickland & Lovering, 2006)), environmental and pathological (exercise-induced asthma, bronchospasm and vocal cord dysfunction), psychological (anxiety-induced hyperventilation) and biomechanical (cycling postures). Whilst further details of the mechanistic factors are beyond the scope of this review, the latter three will be explored in more detail below.

2.2.4.1 Environmental and Pathological Effects on Breathing

The prevalence of disordered breathing in athletes is unknown; however the literature suggests that athletes are just as much at risk as the general population, if not more, of developing breathing related problems, which will subsequently affect their breathing pattern during exercise (Rundell & Spiering, 2003). The incidence of asthma in athletes (11-50%; (Larsson, Hemmingsson, & Boethius, 1994; Rundell & Spiering, 2003)), is known to be greater than in the

general population (10-12%; (AIHW, 2005)) which could be due to increased exposure to cold, dry environments as well as pollen and pollution whilst training (Mendelli, Lounana, Messan, Menuet, & Petitjean, 2006). For example, Rundell and Spiering (2003) reported a 30% incidence of EIB in 370 developmental and elite athletes. In Olympic athletes, Fallon (2003) suggested that the incidence of EIB may be as high as 10-15%. Breathlessness (36.8%), exercise–induced phlegm production (14.9%), cough (7.3%), wheezing (6.2%) and chest tightness (4.9%) were amongst respiratory symptoms reported in a sample of 698 athletes despite only 15.3% having diagnosed asthma (Turcotte, Langdeau, Thibault & Boulet, 2003) suggesting that EIA is not the only cause of respiratory symptoms in athletes. Inspiratory stridor is another respiratory sign seen in athletes and is often associated with Vocal Cord Dysfunction (VCD) - the paradoxical closure of the vocal cords that occurs during inspiration (Rundell & Spiering, 2003). Mouth breathing, increased activation of the scalene muscles (accessory respiratory muscles), anxiety and air pollutants are all triggers of VCD. Symptoms usually present at a high V_E during both intense training and/or competition (Fallon, 2003), causing stridor, air hunger and chest tightness – all symptoms that closely mimic EIA (Brugman & Simons, 1998). Not surprisingly, VCD is often misdiagnosed in athletes as EIA and treated as such (Brugman & Simons, 1998; Fallon, 2004; Rundell & Spiering, 2003). VCD is unresponsive to traditional asthma medications, potentially meaning that athletes never receive the appropriate treatment and subsequent resolution of their symptoms (Fallon, 2004).

2.2.4.2 Psychological Effects on Breathing

In addition to the environmental and pathological factors presented above, psychological factors may also affect an athlete's breathing pattern (Taylor, Rehder, Hyatt, & Parker, 1989). Anxiety is known to increase respiratory rate through stimulation of the sympathetic nervous system (Ley &

Yelich, 1998; Bradley, 2002). Athletes competing in a sporting environment are subjected to stressful, anxiety-inducing environments on a regular basis (Baker, Cote, & Hawes, 2000), which may predispose them to BPDs. It is possible that the incidence of BPDs is higher in athletes than the general population due to the high levels of performance anxiety to which they are exposed.

2.2.4.3 Postural effects on breathing

Breathing may also be compromised in cyclists through biomechanical factors. More specifically, it is possible that the posture adopted by a cyclist when riding may compromise breathing efficiency. For example, time trial riders adopt an aerodynamic position (Figure 3) to minimize frontal area in order to maximize speed (Capelli, Rosa, & Butti, 1993; Faria, Parker, & Faria, 2005b).

Figure 3. A typical athlete's cycling position during a road time-trial (reprinted with the permission of Stephen Sheldrake, NZ Triathlete)

When cyclists adopt this position, the elbows are brought in close to the body and the trunk close to the thighs – a position that compromises the ability of the thoracic cavity to expand fully to enable breathing to maximal capacity (Hogg, 2005). This aerodynamic position may induce an increase in abdominal impedance, and therefore in diaphragmatic work to overcome it, leading to premature recruitment of the accessory respiratory muscles accelerating the onset of respiratory muscle fatigue (Boussana et al., 2007; Johnson, Babcock, Suman, & Dempsey, 1993).

While gains are to be made by adopting an aerodynamic position, some evidence exists to suggest that the aerodynamic position may not be metabolically efficient. Gnehn, Reichenbach, Altpeter, Widmer & Hoppeler (1997) observed a significant increase in $\dot{V}O_2$ (3%; P = 0.02), heart rate (3.3%; P = 0.02), and respiratory exchange ratio (1.3%; P = 0.02) when a cyclist's upper body configuration was changed from the upright to the time trial position at 70% $\dot{V}O_{2\,max}$. Although these are small percentage changes, they may be important if they carry over to the cyclist's performance. Conversely, in two sub-maximal conditions (70 and 80% $\dot{V}O_{2\,max}$), Johnson and Shultz (1990) found no evidence to suggest that riding in an aerodynamic position interfered with breathing mechanics in any way (70% VO_{2max}), nor did they show a significant difference in $\dot{V}O_2$ when cyclists rode at 80% of $\dot{V}O_{2\,max}$ during a 10 minute steady state ride.

Clearly, the apparent increased metabolic cost that may be associated with the time trial position, is offset by the gains made by decreasing the aerodynamic resistance (Gnehm, Reichenbach, Altpeter, Widmer, & Hoppeler, 1997). At speeds greater than 50kph (often achieved during time trials in elite competition) some authors argue that aerodynamic resistance is the most

performance-determining variable (Faria, Parker, & Faria, 2005). The effect of the time trial position on breathing has primarily been investigated at sub-maximal intensities (Gnehm et al., 1990; Johnson & Shultz, 1997). However, time trial competitions are raced at high intensities and it is unclear from the literature what effect the aerodynamic time trial position would have on breathing pattern at higher intensity domains (i.e. closer to $\dot{V}O_{2\ max}$). It is possible that adopting an efficient breathing pattern whilst riding in the time trial position may reduce the elevated metabolic cost previously reported in this position and, added to the gains of optimising aerodynamics, may further enhance performance.

A combination of environmental, pathological, physiological and psychological factors, in addition to the potential contribution that posture makes to altered respiratory mechanics, may affect an athlete's breathing. This may occur acutely, for example increased f_b immediately prior to a race or during training, but more detrimentally may affect it chronically through alteration of the subconscious breathing pattern and altered respiratory mechanics. The consequences of altered respiratory mechanics are multifactorial and, in addition to compromising breathing efficiency, may ultimately have a negative effect on performance. These consequences will be further discussed in the section below.

2.3. Consequences of Altered Respiratory Mechanics

Respiratory mechanics during exercise may be altered by the presence of BPDs at rest, the normal respiratory response to exercise, development of a tachypnoeic shift and postures adopted when riding. The consequences of altered respiratory mechanics (changes to respiratory timing and muscle recruitment patterns) may be detrimental to breathing efficiency and ultimately

exercise performance by initiating dynamic hyperinflation of the lungs and increasing the work of breathing. Dynamic hyperinflation and increased work of breathing may contribute to respiratory muscle fatigue, blood stealing and dyspnoea – all factors that have been associated with decreased exercise performance. This section will explore how altered respiratory mechanics increase the work of breathing and lead to such consequences, and in turn how exercise performance may be negatively affected.

2.3.1. Respiratory Timing

The "normal" timing of the inspiratory to expiratory ratio at rest is 1:1.5 - 2 (West, 1995). To achieve the increased f_b and decreased V_T associated with exercise, the ratio and length of inspiratory (t_I) and expiratory (t_E) time has to change. In normal breathing, the lengthened expiratory phase ensures the inspiratory muscles are returned to their optimal length prior to the next inspiration. Previous research with sedentary individuals and elite cyclists (Folinsbee, Wallace, Bedi, & Horvath, 1983) indicates that at high respiratory rates the reduction of expiratory time makes a significantly greater contribution to the rise in f_b, than the decrease in t_I, effectively resulting in a reverse inspiratory to expiratory ratio of 2:1. This breathing ratio leads to "breath stacking" – a phenomenon where a subsequent inspiration is taken prior to the full exhalation of the previous breath, resulting in tidal breathing occurring closer to total lung capacity (the volume of air in the lungs at the height of a maximal inspiration) (Chaitow & Bradley, 2002; McConnell, 2005). Increased end-inspiratory (Grazzini, Stendardi, Gigliotti, & Scano, 2005; Johnson, Aaron, Babcock, & Dempsey, 1996) and increased end-expiratory lung volumes have been observed in athletes (Sutton, 1992), suggesting that breathing at higher lung volumes or "dynamic hyperinflation" may be a consequence of the respiratory response to exercise (McConnell, 2005).

2.3.2. Dynamic Hyperinflation

Whilst the initial increase in V_T at the onset of exercise may cause end-expiratory lung volume (EELV) to decrease, increases to f_b and the subsequent changes to respiratory timing discussed above may increase EELV, resulting in dynamic hyperinflation (Johnson, Saupe, & Dempsey, 1992). Dynamic hyperinflation affects the mechanics of the respiratory muscles in three ways - by altering the length-tension, the pressure-volume and the force-velocity relationships. Muscles have an optimal length at which they can generate their maximal force. This is referred to as the length-tension relationship of muscles (Rack & Westbury, 1969). When a muscle is required to contract in a position that is shorter or longer than its optimal length, its force generating potential is compromised. Hyperinflation induces functional weakening of the inspiratory muscles by shortening the operating lengths of the diaphragm and accessory muscles. The inspiratory muscles therefore move to a weaker portion of the length tension relationship and their ability to generate a forceful contraction is compromised (Grazzini, Stendardi, Gigliotti, & Scano, 2005). Additionally, because the operating length of the muscles is shortened, the time available for contraction through range is less. The shorter time through which a muscle must contract, the lower its force generating potential (Grazzini, Stendardi, Gigliotti, & Scano, 2005) therefore the inspiratory muscles are also in a weaker portion of their force-velocity relationship.

Lung compliance refers to the capability of the lungs to distend under pressure as measured by pulmonary volume change per unit pressure change (West, 1995). As inspiration approaches total lung capacity the lungs are almost at maximal distension and the lungs become 'stiffer'. Therefore, for a given inspiratory pressure (effort), there is a smaller increase in lung volume during dynamic hyperinflation, than at normal lung volumes, resulting in decreased pulmonary ventilation. Additionally, the inward elastic recoil of the respiratory system is greater at higher

volumes, imposing an added load on the inspiratory muscles, which are already contracting inefficiently (ATS, 1998).

The alteration in the length-tension, force-velocity and pressure-volume relationships means that if dynamic hyperinflation occurs (through an inefficient breathing pattern) at a time when the demands on the inspiratory muscles are greatest (i.e. during intense exercise), then the muscle's force generation capacity will be significantly compromised (Johnson, Aaron, Babcock, & Dempsey, 1996). The capacity of the inspiratory muscles to generate dynamic pressure has been predicted to decrease by 17% for each 10% of the total lung capacity that is accounted for by an increased tidal volume above functional residual capacity (El-Manshawi, Killian, & Summers, 1986). Thus, even a small increase in end-expiratory lung volume will reduce the contractile properties of the inspiratory muscles, and add to the load presented to them such that the work required to sustain ventilation (work of breathing), especially during intense exercise, is significantly increased.

2.3.3. Expiratory Flow Limitation

Although traditionally the mechanics of inspiration have been the main subject of investigation in ventilation during exercise (Grazzini, Stendardi, Gigliotti, & Scano, 2005; Johnson, Saupe, & Dempsey, 1992), the mechanics of expiration may also be negatively affected in some athletes. At high levels of ventilation, an expiratory flow limitation has been identified in some athletes, specifically elite, endurance athletes (Johnson, Saupe, & Dempsey, 1992). As expiration becomes active to meet the increasing ventilatory demands experienced with high intensity exercise, the abdominal muscles contract to force the diaphragm up and the resulting pleural pressure can become positive. Positive pleural pressure may temporarily collapse the bronchi

and cause limitation of expiratory airflow. Thus, further increases in expiratory pressure limits expiratory flow, and requires additional metabolic work in an attempt to overcome the flow limitation (Kayser, Sliwinski, Yan, Tobiasz, & Macklem, 1997). It is suggested that the presence of expiratory flow limitation promotes dynamic pulmonary hyperinflation with the concomitant increase of work of breathing and impairment of inspiratory muscle function discussed above, which may contribute to dyspnoea and limitation to exercise (Koulouris & Kosmas, 2002).

2.3.4. Work of breathing

Breathing at rest in healthy individuals requires less than 5% of the body's total $\dot{V}O_2$ (Jones, Dean, & Chow, 2003). However, in highly trained individuals, the work of breathing during intense exercise (92-100% $\dot{V}O_{2\ max}$) may require up to 15% of the body's total $\dot{V}O_2$ (Aaron, Seow, Johnson, & Dempsey, 1992B), to enable the respiratory muscles to meet the metabolic demands of this level of exercise. It would be plausible to expect that to meet such an increase in metabolic demand, the respiratory muscles would also require a significant increase in the percentage of cardiac output (Q). In an attempt to demonstrate this, Harms et al (1998) manipulated the work of breathing using a proportional assist ventilator (PAV) to either decrease or increase the work of breathing. Using this technique during maximal intensity exercise, Harms et al. (1998) showed that a substantial portion of Q (14-16%) was indeed directed to the respiratory muscles to meet their metabolic requirements. Given the importance of a high V_E during maximal intensity exercise, and the consequent increase in the work of breathing, changes to breathing pattern, respiratory mechanics and work of breathing at maximal exercise could potentially play a part in limiting exercise performance.

2.3.5. *Respiratory Muscle Fatigue*

The diaphragm is the most fatigue resistant of all the skeletal muscles (Dempsey, 2006b). A healthy diaphragm is composed of approximately 45% type I and 55% type II muscle fibres (McConnell, 2005) and unsurprisingly, has a very high aerobic enzymatic capacity and blood supply (Dempsey, Romer, Rodman, Miller, & Smith, 2006). Despite these fatigue-resistant qualities, the diaphragm has been shown to fatigue during exercise to exhaustion at intensities greater than 85% of $\dot{V}O_{2\ max}$. Using bilateral phrenic nerve stimulation (BPNS) (1-20Hz) to measure transdiaphragmatic pressure before and after exercise to exhaustion at 80% $\dot{V}O_{2\ max}$ in healthy sedentary subjects, Mador et al. (1993) demonstrated a 17.2% decrease in transdiaphragmatic pressure 10 minutes after cycling exercise to exhaustion. After 60 minutes of rest, transdiaphragmatic pressure had recovered to 93 ± 7% of pre-exercise levels. Using similar methodology in active individuals exercising at 85 and 95% of $\dot{V}O_{2\ max}$, Johnson et al. (1996) demonstrated that transdiaphragmatic pressure immediately after exercise was reduced 25-50% below pre-exercise measures and took two hours to recover. Johnson et al. (1996) concluded that heavy, whole-body exercise to exhaustion in relatively fit, normal subjects resulted in significant diaphragmatic fatigue. Comparing these two studies, it appears that the degree to which the diaphragm fatigues, and the speed at which it recovers, is significantly affected by the intensity of exercise. Conversely, diaphragm fatigue has not been shown to occur below 80% $\dot{V}O_{2\ max}$ (Gallagher & Younes, 1989) or when the diaphragm is fatiguable with short term (less than eight minutes) progressive exercise to exhaustion unless a resistive load is added (Levine & Henson, 1988). This would suggest that diaphragm fatigue is determined by a combination of the work demanded of it, and the time that it is under load.

The actual work performed by the inspiratory muscles, specifically the diaphragm, per se is, however, not enough to explain the diaphragm fatigue observed by Mador et al. (1993) or Johnson et al. (1996). When resting subjects were asked to mimic (using auditory and visual feedback) the duration and magnitude of diaphragm work achieved during maximal intensity, endurance exercise, fatigue did not occur. Only by increasing diaphragm work voluntarily to twice that required during maximal exercise did diaphragm fatigue occur (Babcock, Pegelow, McClaran, Suman, & Dempsey, 1995). This would suggest that other changes that occur, perhaps elsewhere in the body, during intense exercise contribute to respiratory muscle fatigue, rather than just an inability of the respiratory muscles to meet the work demand.

Global inspiratory muscle fatigue has also been demonstrated to occur immediately following exercise both in untrained (Boutellier & Piwko, 1992) and trained individuals (Romer, McConnell, & Jones, 2002c; Volianitis et al., 2001). For example, Romer et al. (2002) demonstrated substantial decreases (compared to baseline measures) in P_o (pressure generated by the respiratory muscles at zero flow) of -17 ± 4% and -13 ± 3% following a simulated 20 and 40km-TT respectively in competitive cyclists. Furthermore, P_o had not returned to baseline levels at 30 minutes post-exercise. Similarly, but in competitive swimmers, Lomax and McConnell (2003) reported a 29% deficit from baseline measures in maximal inspiratory pressure (MIP) following a 200m freestyle swim at 90-95% race pace. In competitive rowers, Volianitis et al. (2001) demonstrated a 10% decline in MIP after six minutes of rowing ergometry at 100%. One explanation for the large differences in deficits observed in these three studies (apart from the obviously important postural differences) could be due to the nature of the sports and the ventilatory requirements of each. For example, entrainment has been observed in high level rowers and it is possible that the rowers used in the study subconsciously used entrainment to

optimise breathing and subsequently attenuate respiratory muscle fatigue. Conversely, in freestyle swimming the mouth is out of water for a very short time, during which an inspiratory effort must be taken. This short, fast inspiratory effort may predispose to dynamic hyperinflation and exaggerate the pressure generating deficit from respiratory muscle fatigue, due to alterations of the length – tension relationship (discussed earlier). Another factor that may exacerbate respiratory muscle fatigue in swimming, and contribute to the different degrees of inspiratory muscle fatigue observed by Romer et al. (2002) and Volianitis et al. (2001), is the hydrostatic compression around the chest wall when the inspiratory muscles are relaxed (Withers & Hamdorf, 1989). Hydrostatic pressure counteracts inspiratory muscle force (Frangolias & Rhodes, 1996) which may increase inspiratory muscle work in an attempt to overcome this.

Exercise duration, exercise intensity and inefficient respiratory mechanics can increase the work of breathing to the degree that respiratory muscle fatigue may occur, with negative consequences on exercise performance (Harms, Wetter, St Croix, Pegelow, & Dempsey, 2000). Harms et al. (2000) demonstrated this concept by unloading the respiratory muscles during high intensity exercise (90% $\dot{V}O_{2\ max}$) using a PAV. Subjects were able to cycle longer (14.4 ± 4.9%; p<0.05) than the control duration, whereas increasing respiratory muscle work significantly reduced (-15.1 ± 3.3%; p<0.05) exercise time from control values. It is possible that there were other factors contributing to the effect on exercise time in Harms et al.'s (2000) study in addition to respiratory muscle fatigue (for example blood stealing, which will be discussed later) however certainly when load was removed from the respiratory muscles their capability for sustained power generation was increased.

Respiratory muscle fatigue may negatively affect exercise performance by altering the way the respiratory muscles are recruited (Johnson, Aaron, Babcock, & Dempsey, 1996). Indirect evidence from pressure recordings suggest that the fatiguing diaphragm actually reduces its output as a force generator during the later stages of sustained endurance exercise and accessory inspiratory and expiratory muscles become the dominant effecter of time dependant hyperventilation (Dempsey, 2006b). Johnson et al. (1996) suggested that although total V_E and respiratory muscle pressure may continue to rise, the diaphragm is deactivated in an attempt to reserve function. Diaphragm fatigue may indirectly influence performance by a reduction in the relative contribution of the diaphragm; and a subsequent increase of accessory muscle use may negatively affect performance. This change in muscle recruitment pattern may lead to dynamic hyperinflation, inefficient respiratory mechanics, subsequent increased work of breathing and ultimately further respiratory muscle fatigue (Harms, Wetter, St Croix, Pegelow, & Dempsey, 2000) and blood stealing (Harms et al., 1997) – the latter being detrimental to limb function during strenuous exercise.

The ability of professional cyclists, unlike elite amateur and lower level cyclists, to maintain the diaphragmatic breathing pattern from low to high intensity (exhaustive) exercise (Lucia, Carvajal, Calderon, Alfonso, & Chicharro, 1999) demonstrates that a mechanism may exist that enables high intensity exercise to be maintained, without an increase in the work of breathing to the point that respiratory muscle fatigue, and the subsequent negative effect on performance, occur. It has been suggested that this controlled breathing pattern is one of the major factors that enables professional cyclists to perform better than the tier of cyclists below them (Faria, Parker, & Faria, 2005b). Lucia et al.'s (1999) findings suggest that either respiratory muscle fatigue did not occur in professional cyclists, or, if it did, it did not result in deactivation of the diaphragm with a

subsequent shift to an apical breathing pattern. This observation would suggest that the decreased diaphragm recruitment speculated by Johnson et al. (1996) is more an inefficient but common response during exercise, rather than an efficient physiological necessity. Johnson et al. (1996) commented that although the diaphragm appeared to be deactivated, total V_E and respiratory muscle output continued to rise. However, it is not known what, if any, greater increases in these variables would have occurred had the diaphragm continued to be the primary pressure generator. It is therefore possible that the presence of an altered breathing pattern is the instigator that then leads to respiratory muscle fatigue through the altered respiratory muscle mechanics discussed above, rather than the result of respiratory muscle fatigue itself.

Regardless of whether altered respiratory recruitment pattern is the cause or effect of respiratory muscle fatigue, the implications of it may not just be limited to the mechanical effects. Increased work of breathing and respiratory muscle fatigue may have a further effect of causing reflex vasoconstriction to exercising muscles, reducing blood flow to the muscles responsible for power generation during exercise, and subsequently decrease exercise performance.

2.3.6. Blood Stealing

A further consequence of altered respiratory mechanics, and the subsequent increased work of breathing, is the potential competition for blood supply between the peripheral locomotor and respiratory muscles. During exercise, an adequate blood supply is vital to ensure required nutrients are delivered to, and exercise metabolites are removed from muscle (Barclay, 1986; Frisbee, Murrant, Wilson, & Barclay, 1999). At maximal intensity exercise, competition exists between the respiratory and locomotor muscles for a supply of energy that is insufficient to meet the needs of both (Aliverti & Macklem, 2001). Subsequently blood flow may be diverted from the

exercising (locomotor) muscles to the respiratory muscles under certain respiratory conditions (blood stealing) to maintain the vital supply of respiratory muscle $\dot{V}O_2$ (Harms et al., 1997).

The concept of blood stealing was first investigated by Secher et al. (1977) who demonstrated that recruiting additional muscle mass during sub-maximal leg cycling ($\dot{V}O_2$ legs 67% $\dot{V}O_{2\ max}$) by simultaneously arm cranking ($\dot{V}O_2$ arms 44%; total body exercise 78% $\dot{V}O_{2\ max}$) decreased blood flow to the already exercising leg muscle by approximately 15% (12.4 to 10.5 L·min^{-1}). Since that pioneering work further studies have been undertaken that both refute (Kowalchuk, Rossitter, Ward, & Whipp, 2002; Richter, Kiens, Hargreaves, & Kjaer, 1992) and support (Harms et al., 1997; Harms et al., 1998; Sheel et al., 2001) the existence of the blood stealing phenomenon. In contrast to Secher et al.'s (1977) findings, Richter et al. (1992) was unable to demonstrate a decrease in Q_{legs} when arm cranking was added to cycling in untrained cyclists. Further, Kowalchuk et al. (2002) demonstrated that increasing the work of breathing by adding a load to both inspiratory and expiratory muscles during heavy intensity exercise (93 ± 3% $\dot{V}O_{2\ max}$), also in untrained cyclist, did not increase leg de-oxygenation (the variable these authors chose to indicate decreased Q_{legs}), implying that Q_{legs} was not reduced consequent to the increased work of breathing.

In contrast to the studies in untrained cyclists that refute blood stealing, two well designed studies that support its existence were completed by Harms et al. (1997; 1998) involving competitive male cyclists (N = 7; N = 8). These authors demonstrated that using a PAV to unload the respiratory muscles and decrease the work of breathing (by 63%, compared to control) during maximal incremental cycling to exhaustion, resulted in a decrease in Q (2.4 ± 0.7 l/min) and limb vascular

resistance (LVR) (0.5 ± 0.1 mmHg $\cdot l^{-1} \cdot$ min), whilst blood flow to the leg muscles (Q_{legs}) increased (0.8 ± 0.3 L $\cdot$ min^{-1}). Conversely, when the work of breathing was artificially increased (by 28%) by adding inspiratory resistance (3-5 cmH$_2$O $\cdot$ L$^{-1} \cdot$ s^{-1}), both LVR (1.5 ± 0.3 mmHg $\cdot l^{-1} \cdot$ min; p = 0.002) and Q_{legs} (1.3 ± 0.2 l/min; p = 0.002) decreased (Harms et al., 1997). These results clearly show that a relationship exists between increased work of breathing and Q_{legs} such that increasing the work of breathing has a detrimental effect on locomotor muscle blood flow. This could suggest that factors that increase the work of breathing in cyclists, for example posture and altered respiratory mechanics, may compromise leg blood flow – the consequences of which are discussed below. Conversely, factors that prevent the work of breathing from increasing may preserve Q_{legs}.

Blood stealing is thought to be due to sympathetically mediated vasoconstriction in the locomotor muscles induced in a reflex manner, possibly originating in fatigueing respiratory musculature (Harms et al., 1998). It is postulated that during maximal exercise, the work and / or aerobic status of the respiratory muscles may stimulate type III-IV afferents in the diaphragm and other respiratory muscles which then causes reflex vasoconstriction of limb vasculature during control or loaded conditions and local vasodilation with unloading of the respiratory muscles (Wetter, Harms, Nelson, Pegelow, & Dempsey, 1999). Increased sympathetic excitation of limb vasculature is measured by norepinephrine (NE) spillover (Harms et al., 1997). Harms et al. (1997) demonstrated a $78 \pm 5\%$ increase in NE with respiratory muscle loading (28% increased work of breathing) during maximal exercise, which was significantly related to LVR (r = 0.71, P < 0.05) suggesting a significant increase in limb vasoconstriction with increased work of breathing. To identify the specific role that the respiratory muscles had in the aetiology of blood stealing, Sheel et al. (2001) investigated the effect that respiratory muscle fatigue had on Q_{legs} at rest.

Using a combination of varying inspiratory resistance and respiratory patterns to fatigue the respiratory muscles in different ways, (i.e. changes to f_b and duty cycle) Sheel et al. (2001) demonstrated decreased Q_{legs} (-30%) and increased LVR (50-60%), when compared with control conditions, only at the point of diaphragm fatigue. Voluntary increases in inspiratory effort without respiratory muscle fatigue had no effect on either Q_{legs} or LVR, indicating that the work of breathing must be sufficient to cause respiratory muscle fatigue in order for blood stealing to occur.

The contrasting results above that support and refute the existence of blood stealing may be explained, however, by methodological factors. Wetter et al. (1999) observed that the effect of respiratory muscle unloading on Q_{legs} did not occur unless exercise intensity was greater than 75% $\dot{V}O_{2\ max}$. During sub-maximal (50% and 75% of $\dot{V}O_{2\ max}$) exercise, $\dot{V}O_{2\ total}$ increased with increasing work of breathing and decreased with decreasing work of breathing but Q_{legs} and LVR did not change with changing work of breathing. Even substantial increases in the work of breathing (50-70%), although raising whole body $\dot{V}O_2$, did not cause alterations in LVR or Q_{legs} during sub maximal exercise (Wetter, Harms, Nelson, Pegelow, & Dempsey, 1999). Therefore, it would appear that blood stealing only occurs during very high intensity exercise, and under conditions that fatigue the respiratory muscles. Although Kowalchuk et al. (2002) used a much greater inspiratory resistance than Harm et al. (1997; 1998) (7cmH$_2$O·L^{-1}·s^{-1} vs. 3-5cmH$_2$O·L^{-1}·s^{-1} respectively) at a work load that had previously been shown to induce decreased Q_{legs}, they controlled both f_b and V_T during all experimental protocols. It is possible that by doing this the respiratory muscles were not fatigued enough to invoke the metaboreflex and subsequent changes to Q_{legs} and LVR. Further, Richardson et al. (1995) and Richter et al. (1992) increased exercising muscle mass by adding another peripheral muscle group, rather than increasing the

work of breathing. The respiratory muscles were not specifically fatigued in these studies, and as the exercise intensity (<75% $\dot{V}O_{2\,max}$) was unlikely to be sufficient to induce respiratory muscle fatigue indirectly, it is unlikely that metaboreflex was invoked.

When methodological conditions are taken into consideration, the evidence presented above suggests that blood stealing does occur when the work of breathing is increased. The work of the respiratory muscles in normal physiological conditions at maximal exercise intensity appears to have two effects on the cardiovascular response – a) a substantial proportion of cardiac output is directed to the respiratory muscles to support their metabolic requirements as discussed above, and b) blood flow is reduced to (or 'stolen from') working locomotor muscles (Harms et al., 1997; Harms et al., 1998). Limb muscle force output and fatigue are highly responsive to changes in limb muscle blood flow under conditions of high intensities of muscle contraction (Romer & Dempsey, 2006). The effects of changing limb blood flow have been attributed to changes in O_2 transport and/or changes in the washout of local metabolites (Barclay, 1986). Given that blood flow to the legs during maximal intensity exercise may be in the order of 15.4 ± 0.3 L·min^{-1} (Harms et al., 1997), a decrease of 1.3 L·min^{-1} (8.4%) would likely have a large impact on performance. To investigate this effect, Harms et al. (2000) again used a PAV to offload, (and inspiratory resistance to load) the respiratory muscles during incremental tests to exhaustion at $\dot{V}O_{2\,max}$ in competitive cyclists. When inspiratory muscle work was decreased by 63%, subjects were able to exercise 1.3 ± 2 minutes longer (range -1.2 – 5.3 min; +14.4% ± 4.9%, p<0.05) than during control conditions, whereas with loading, subjects exercised 1.0 ± 0.8 minutes less (range -0.4-4.7 min; 15.1 ± 3.3%; p<0.05) (Harms, Wetter, St Croix, Pegelow, & Dempsey, 2000). Clearly this suggests that the decrease in Q_{legs} caused by increased work of breathing was sufficient to alter

cycling performance. To further investigate the effect of blood stealing on performance, and using similar methodology, Romer and Dempsey (2006) recently measured quadriceps power, as a measurement of peripheral fatigue in eight endurance trained male cyclists, before and after incremental cycling in a randomised series of control, inspiratory muscle loading and unloading. Exercise at 90% of maximum work rate (292W for 13.2 minutes) under control conditions reduced force output of the quadriceps by 28%, indicating an expected presence of quadriceps fatigue after exercise to exhaustion. This fatigue was gradually recovered with 70 minutes of rest following exercise. After completion of an identical amount and duration of exercise as in the control, but with unloading sufficient to reduce the work of breathing by 50-60%, limb fatigue was 8% less than the control, whereas respiratory muscle loading increased fatigue by 20% greater than the control and quadricep strength had not recovered by 70 minutes. This suggests that preventing the work of breathing from increasing to the point that the Q_{legs} is compromised, is an important consideration for cyclists in order to optimise power output and exercise performance.

The findings by Harms et al. (1997, 2000), Sheel et al. (2001) and Romer et al. (2006) clearly give strength to the suggestion that increased respiratory muscle work, particularly to the point of respiratory muscle fatigue, will decrease blood flow to the exercising peripheral muscles to the degree that physical exercise performance is impaired. If the altered respiratory muscle mechanics experienced by cyclists due to factors such as breathing pattern disorders, development of a tachypnoeic breathing pattern and the normal physiological response to exercise was such that respiratory muscle fatigue was invoked, theoretically performance at maximal intensity exercise would be compromised. Conversely, if strategies or interventions were employed to optimise respiratory mechanics and prevent unnecessary respiratory muscle fatigue, performance at maximal intensity exercise could be improved by decreasing blood stealing.

Clearly, maintaining optimal respiratory mechanics may be a simple way to provide the small improvement in performance that is so sought after by cyclists and sports scientists alike.

Maintaining optimal respiratory mechanics to prevent respiratory muscle fatigue may have a second benefit with regards to optimising cycling performance – quite separate to preventing blood stealing. Respiratory muscle fatigue is thought to be an important component in the development of dyspnoea, which is a major factor in limiting exercise in healthy humans.

2.3.7. Dyspnoea

Dyspnoea is one of the primary reasons given by healthy individuals for the termination of exercise to exhaustion (Hamilton, Killian, Summers, & Jones, 1996; Killian et al., 1992) indicating that some aspect of ventilation limits exercise. Dyspnoea is the subjective awareness of an increased work of breathing (Webber & Pryor, 1994, p. 5). It is a general term used to characterise a range of different descriptors that vary in intensity and are influenced by a wide variety of factors, such as cultural expectations and previous experience (Grazzini, Stendardi, Gigliotti, & Scano, 2005). In healthy humans, dyspnoea limits exercise when it reaches a level that is deemed no longer tolerable (Grazzini, Stendardi, Gigliotti, & Scano, 2005).

The pathophysiology underlying dyspnoea is both complex and uncertain and a detailed discussion of all potential contributing factors to dyspnoea during exercise (ATS, 1998; McConnell & Romer, 2004a) is beyond the scope of this review. Given the complexity of changes to respiratory mechanics during exercise it is difficult to be sure which alterations contribute most strongly to the sensation of dyspnoea. However, with regards to limiting exercise in athletes, it appears that the contractile properties of the respiratory muscles, combined with operational high

lung volumes and the conscious awareness of the outgoing respiratory motor command are likely contributing factors (Grazzini, Stendardi, Gigliotti, & Scano, 2005). Factors that impair the contractile properties of the respiratory muscles (for example functional weakening, pattern of recruitment and fatigue) have the potential to increase the intensity of dyspnoea, whereas factors that improve the contractile properties of these respiratory muscles have the potential to reduce the intensity of dyspnoea (McConnell & Romer, 2004a).

In the 1960's Moran Campbell coined the term length-tension inappropriateness (Campbell, 1966) and suggested that humans have a quantitative appreciation of the degree of effort associated with breathing. Further, dissociation or a mismatch between central respiratory motor activity and the mechanical response of the respiratory system may produce a sensation of respiratory discomfort (dyspnoea) (McConnell & Romer, 2004a). As such, the intensity of dyspnoea is increased when changes in respiratory muscle length (i.e. volume) or tension (i.e. pressure) are inappropriate for the outgoing motor command. During exercise, the central outgoing drive for ventilation is increased, however the decrease in respiratory muscle efficiency through altered respiratory mechanics (i.e. breathing occurring on a less compliant portion of the pressure-volume curve, increased elastic recoil of the respiratory system at end expiration, and respiratory muscle shortening) (ATS, 1998) does not enable appropriate volume and pressure changes to occur. In other words, although the drive to breathe has been increased in response to intensifying exercise, the capability of the respiratory muscles to respond appropriately is compromised. It is possible that this inappropriate response is perceived in the brain as 'not breathing enough' resulting in the sensation of dyspnoea. Theoretically, interventions that restore more efficient respiratory mechanics could enable the respiratory muscles to respond more appropriately to the increased central output and therefore decrease perceptions of dyspnoea (McConnell & Romer, 2004a).

The order in which the respiratory muscles are recruited in response to exercise may also contribute to the intensity of dyspnoea. Assuming a diaphragmatic breathing pattern is present, the diaphragm is recruited first, followed by increasing activation of the respiratory accessory muscles as breathing frequency increases. Animal studies show that the diaphragm is less well supplied with proprioceptors than the intercostal muscles (accessory muscles) (Corda, von Euler, & Lennerstrand, 1965). If the same is assumed in humans, more sensory input as to the work of the muscles would be received centrally from the accessory muscles, than from the diaphragm, potentially giving the impression that more work was being done, with a subsequent increase in perceived effort, when the accessory muscles were recruited.

The importance of the inspiratory accessory muscles in the aetiology of dyspnoea is further supported by evidence that the increase in respiratory effort during fatiguing inspiratory resistive loading correlated better with the level of activation of muscles of the rib cage and neck muscles than with the level of activation of the diaphragm (Ward, Eidelman, & Stubbing, 1988). Further, loaded breathing tasks preferentially fatigue the inspiratory accessory muscles rather than the diaphragm (Hershenson, Kikuchi, & Tzelepis, 1989). As the force generating capacity of the diaphragm exceeds that of the accessory muscles (Hershenson, Kikuchi, & Loring, 1988), a recruitment strategy that brings in the accessory muscles, especially sooner than necessary through a faulty breathing pattern, may necessitate a greater motor outflow since recruitment of weaker muscles to achieve a given intra-thoracic pressures change would require a higher level of motor outflow (McConnell & Romer, 2004a). Minimising, or at least delaying, the recruitment of the accessory muscles by encouraging maintenance of a diaphragmatic breathing pattern could

attenuate or delay the onset of dyspnoea by decreasing sensory input received from the accessory muscles regarding the work being done.

If dyspnoea was due solely to mechanical or physical changes to the respiratory pump, then one would expect patients with similar FEV_1 (forced expiratory volume in one second; a measure of lung function) values to report similar levels of dyspnoea. However, Cooper (2006) reported there was no correlation between these two variables, supporting the theory that dyspnoea may also be influenced by cerebral factors (ATS, 1998). It is acknowledged that the conscious awareness of the outgoing respiratory motor command has an important role in the perception of dyspnoea (McConnell & Romer, 2004a), and that sensory adaptation to intensity of dyspnoea may also occur (ATS, 1998). Whilst the magnitude of these topics puts them beyond the scope of this review, it is important to note that dyspnoea, by definition, is a person's *perception* of breathlessness (Grazzini, Stendardi, Gigliotti, & Scano, 2005). It is a personalised experience, likely affected by a raft of psychological and cerebral components, and any strategies used in an attempt to attenuate dyspnoea must recognise the subjectivity of the sensation and manage this appropriately (Grazzini, Stendardi, Gigliotti, & Scano, 2005).

2.4. Breathing as a Limiting Factor to Exercise and Strategies to Overcome it

2.4.1. Breathing as a Limiting Factor

Traditionally, the idea that breathing may be a limiting factor to exercise has been disregarded due to the apparent breathing reserve observed in athletes even at the highest level of exertion (Dempsey, 1986). This argument was based on the fact that even at exhaustion both trained and sedentary subjects breathed below their maximal voluntary ventilation (MVV; maximal voluntary

ventilation, usually measured in 15 seconds) and therefore still had a significant breathing reserve to increase ventilation further if needed (breathing reserve is calculated as MVV – V_{Emax}, (Markov et al., 2001)). For example, immediately after exercise to exhaustion at 85 and 95% of $\dot{V}O_{2\ max}$, Johnson et al. (1996) recorded values of 70 and 83% MVV respectively in 19 healthy individuals ($\dot{V}O_{2\ max}$ range 40-80ml·kg^{-1}·min^{-1}) after exercise to exhaustion. Similarly, elite cyclists reached 86.9% MVV and professional cyclists 90% MVV (Lucia, Carvajal, Calderon, Alfonso, & Chicharro, 1999) indicating that even highly trained athletes, performing at maximal levels of exhaustion, have an ability to increase their ventilation further.

However, in large studies (N= 578; N=417) comparing reasons for volitional termination in maximal intensity exercise tests in healthy individuals and those with respiratory pathology, both Hamilton et al. (1996) and Killian et al. (1992) identified that breathlessness (either alone or in combination with leg fatigue) contributed equally in both groups. Despite the healthy individuals having no underlying respiratory pathology and, based on the above studies, presumably ample breathing reserve (Johnson, Saupe, & Dempsey, 1992; Lucia, Carvajal, Calderon, Alfonso, & Chicharro, 1999), breathlessness still caused some healthy individuals to terminate exercise. Taking into consideration the apparent breathing reserve, this would suggest that although the physical capability exists to increase ventilation to meet the metabolic demands of maximal exercise, factors exist that prevent the person from maximizing their ventilatory effort.

The literature presented in the previous section indicates that breathing may be a factor that compromises maximal exercise performance if the work of breathing is such that respiratory muscle fatigue occurs. Increased work of breathing has been shown to increase respiratory

muscle fatigue (Johnson, Babcock, Suman, & Dempsey, 1993), decrease blood flow to the locomotor muscles (Harms et al., 1997) and increase dyspnoea (Grazzini, Stendardi, Gigliotti, & Scano, 2005) – a combination of which negatively affect exercise performance (Harms, Wetter, St Croix, Pegelow, & Dempsey, 2000). It would therefore be plausible to suggest that strategies that decrease the work of breathing would attenuate these negative consequences and have a positive effect on exercise tolerance. Certainly, Harms et al. (2000) demonstrated that decreased work of breathing increased exercise performance. More specifically, these authors demonstrated that decreasing the work of breathing using a PAV significantly decreased dyspnoea (0-10 Borg) at both isotonic (5 minutes, 8.5 ± 0.3 unloaded; 9.6 ± 0.2 control) and at end-exercise (approximately 9 minutes, 9.6 ± 0.1; 9.9 ± 0.1). Of particular relevance to athletes and sports performance was that the significant increase in time to exhaustion was most strongly correlated with the change in dyspnoea (r=-0.54, P ≤ 0.05) rather than changes to RPE_{leg} (r = -0.42, P ≤ 0.05), V_E (r = 0.29, P ≤ 0.05) or $\dot{V}O_2$ (r=-0.39, P ≤ 0.05).

2.4.2. *Inspiratory Muscle Training*

One strategy that appears to decrease the symptoms and exercise limiting consequences associated with poor respiratory mechanics and increased work of breathing is inspiratory muscle training (Markov et al., 2001; Romer, McConnell, & Jones, 2002c; Witt, Guenette, Rupert, McKenzie, & Sheel, 2007). Inspiratory muscle training (IMT) aims to increase the strength and endurance of the respiratory muscles and requires a person to breathe against a variable resistance, usually via a hand-held device. A variety of IMT methods exist including voluntary isocapnic hyperpnoea (Leddy et al., 2007; McMahon, Boutellier, Smith, & Spengler, 2002), flow resistive loading (Hanel & Secher, 1991) and pressure threshold loading (Romer, McConnell, &

Jones, 2002a; Volianitis et al., 2001); each with their own methodological strengths and weaknesses (*Table 3*).

	Voluntary Isocapnic Hyperpnoea (VIH)	Flow Resistive Loading (IFRL)	Pressure Threshold Loading (IPTL)
Table 3. Different Methods of IMT			
Procedure	Maintain high targets of ventilation for up to 30 minutes.	Inspire via variable diameter orifice whereby for a given flow the smaller the orifice, the greater the load. Up to 30 minutes	Individuals produce a negative pressure to overcome a threshold load and therefore initiate inspiration
Intensity Benefits	50-90% MVV ↑Time to exhaustion ↑ Maximum sustained ventilatory capacity ↑ V_E ↑ MVV	80% MIP ↑ Inspiratory muscle strength ↑ Total lung capacity (small)	50% P_0 ↑ Maximal force production ↑ Maximal velocity of muscle contraction ↑ Maximal rate of shortening ↑ Maximal power output ↑ Respiratory muscle endurance
Weaknesses	Time consuming Doesn't affect maximum pressure generating capacity. Possibly dries airways secondary to high ventilatory flow rates	Harder to ensure training load is reached secondary to changes to inspiratory pressure, which varies with flow.	Does not address breathing pattern. May exacerbate poor respiratory mechanics.
Effects on respiratory mechanics	Only affects velocity axis of force-velocity relationship	Only affects force axis of force-velocity relationship	Affects both force and velocity axis of force-velocity relationship.

An elegant discussion of the strengths, weaknesses and methodological considerations of IMT is provided by McConnell and Romer (2004b) and therefore will not be covered in depth in this review. In addition to different modes of IMT, a variety of training durations, intensities and protocols are typically used in studies investigating the effect of IMT upon exercise performance and are presented in *Table 4*.

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Table 4. A summary of Exercise Performance Studies using Inspiratory Muscle Training (IMT)

Study	Age (years)	Subject Data	Study Design	IMT Intervention	Measures Collected	Main findings of the effect of RMT intervention
Morgan, DW Kohrt, W M Bates, B J Skinner, JS (1987)		Trained male cyclists	RCT IMT N = 4 Control N = 5	3/52 VIH	VO_{2max} Cycling end. 95% VO_{2max} RME MVV	No change in Endurance time No change VO_{2max}
Fairbarn, M S Coutts, K C Pardy, R L McKenzie, D C (1991)		Experienced male cyclists	RCT IMT N = 5 Control N = 5	4/52 VIH (16 sessions)	VO_{2max} RME Cycling end. @ 90% Pmax	RME 12% No change VO_{2max} No change cycling endurance time
Hanel, B Secher, NH 1991		Healthy Humans	RCT IMT N = 10 Control N = 10	27.5 days, 30mins, 2 x daily IFRL	12 min run PI_{max} V_{Emax} VO_{2max} f_b	↑ MIP 18% ↑12 min run 8% (control ↑ 6%) No change V_{Emax} No change VO_{2max} ↓f_b 5%
Spenger, C M Roos, B Laube, S M Boutellier, U (1999)	26.3 ± 3.3	Athletic males	No control N = 20	4/52 VIH 30 mins, 5 x week	Cycling endurance Lung function VO_{2peak} P_{max} Lactate MVV f_b V_E	↑Cycling endurance 27% No change lung function ↑V_E 23% ↑f_b 37.5% ↓lactate after endurance 7% ↓lactate after incremental 15% ↑MVV 19% No change lung function
Chatham, K Baldwin, J Griffiths, H Sumers, L Enright, S 1999	26 ± 14	Healthy, active 10 Males (5 active) 12 females (6 active)	RCT IMT N = 11 Control N = 11	8/52 IFRL 80% MIP 3 x week	RRPE PRPE VO_{2max} Predicted Shuttle runs RMS RME	↑ VO_{2max} (Pred.) 3.78% ↑ shuttles ↑ RMS 31% ↑ RME 42% No change to RRPE No change to PRPE No change in control
Inbar, O Weiner, P Azgad, Y Rotstein, A Weinstein, Y 2000	28.9 ± 8.9	Well trained endurance track athletes	RCT SIMT N = 10 Placebo N= 10	10/52 IPTL using threshold inspiratory muscle trainer, 30min, 6x / week 30–80% WI_{max}	Pm_{Peak} PI_{max} V_{Emax} VO_{2max}	↑ PmPeak 10% ↑ PI_{max} 24.9% No change to V_{Emax} or VO_{2max}

Study	Age (years)	Subject Data	Study Design	IMT Intervention	Measures Collected	Main findings of the effect of RMT intervention
Markov, G Spengler, C Knopfi-Lenzin, C Steussi, C Boutellier, U 2001	RMT 43 ± 7 ET 40 ± 10 Placebo 37 ± 9	Healthy, sedentary subjects Males = 20 Females = 18	RCT IMT N = 15 ET N = 10 Placebo N = 15	40 training sessions over 14 weeks of: 1. VIH 30mins (60%MVV, continuous breathing) 2. ET 30 mins running or cycling 3. No training	W_{peak} VO_{2peak} Cycling Endurance Stroke Volume	↑Breathing endurance ↑Cycling endurance SV unchanged
Sonetti, D A Wetter, T J Pegelow, D F Dempsey, J A (2001)		Competitive male cyclists	RCT IMT N = 9 Control N = 8	4/52 IPTL AND VIH 3-5 mins IPTL and 30 mins VIH day, 5 x week	MIP Fixed work rate cycling 8km TT P_{max} Lactate HR V_E	↑MIP 8% ↑Fixed work rate 26% (control 16%) ↑1.8% ↑P_{max} 9% (control 6%) No significant difference between control and IMT in above variables No change lactate No change HR No change V_E
Steussi, C Spengler, C M Knopfi-Lenzin, C Markov, G Boutellier, U (2001)	43 ± 7	Sedentary Males = 16 Females = 12	RCT IMT N = 13 Control N = 15	40 x 30 min sessions VIH	Static lung function Cycling end @ 70% P_{max} P_{max} HR V_E	↑Cycling end 23% ↑RME 632% No change lung function No change HR No change V_E No change P_{max}
Volianitis, S McConnell, A K Koutedakis, Y McNaughton, L Backx, K Jones, D A (2001)	23.8 ± 3.8	Competitive female rowers	RCT IMT N = 7 Control N = 7	11/52 IMT 30 inspiratory efforts @ 50% MIP 2 x daily	6 min all out Sub max incremental load 5000m TT RRPE Lactate V_T f_b	4/52: ↑MIP 40.9 % 6 min 3.4% 5000m 3.1% RRPE no change 11/52 ↑MIP 45.3 % (from baseline) 6 min 3.5% (from baseline) 5000m N/A ↓Lactate 1.3% (not different from control) ↑V_T No change f_b

Study	Age (years)	Subject Data	Study Design	IMT Intervention	Measures Collected	Main findings of the effect of RMT intervention
Williams, J S Wongsathikun, J Boon, S M Acevedo, E O 2002	20.9 ± 1.2	Cross country runners. N = 7 Males = 5 Females = 2	No placebo	4/52 IMT with inspiratory resistive device. 25 mins 4-5 x week 50% MIP ↑ by 5% each week	PI_{max} breathing endurance time VO_{2peak} run time @ 80% VO_{2peak}	↑31% PI_{max} ↑128% breathing endurance time VO_{2peak} No change Run time no change
McMahon, M E Boutellier, U Smith, R M Spengler, C 2002	RMT = 26 ± 4 Control 28 ± 6	Experienced male cyclists	RCT RMT N = 10 Control N = 10	4-6/52 VIH IMT = 30 min sessions x 20, VE = 60% MVV Control = unclear	W_{peak} Cycling endurance time to exhaustion @ 85% W_{peak} Breathing endurance Peripheral chemoreceptor sensitivity	W_{peak} no change ↑ Cycling endurance time 3.26% ↑ Breathing endurance ↓ Peripheral chemoreceptor sensitivity
Romer, L M McConnell, A K Jones, D A 2002C	IMT 29.5 +/- 3.3 Placebo 30.3 +/- 2.6	Male competitive cyclists (5 triathletes)	DBRCT IMT N=8 Placebo N=8	6/52 IMT using POWERbreathe 30 dynamic inspiratory efforts, twice a day. Pressure threshold 50% of WI_{max}	Inspiratory muscle pressure pre and post 20km and 40kmTT.	↓P_o change after 20 & 40kmTT. Intervention group returned to baseline at 30 mins, placebo >30mins
Romer, L M McConnell, A K Jones, D A 2002A	IMT 29.5 +/- 3.3 Placebo 30.3 +/- 2.6	Male competitive cyclists (5 triathletes)	DBRCT IMT N=8 Placebo N=8	6/52 IMT using POWERbreathe 30 dynamic inspiratory efforts, twice a day. Pressure threshold 50% of WI_{max}	V_T Respiratory RPE Peripheral RPE 20kmTT & 40kmTT time VO_{2max} Power output	↑V_T= 0.16L (90%W_{max}) ↑V_T= 0.17L(100%W_{max}) ↑20kmTT = 65 ± 30s ↑40kmTT = 114 ± 38s ↓RRPE 16 ± 4% ↓PRPE 18 ± 4% No change to power or VO_{2max}
Romer, L M McConnell, A K Jones, D A 2002B	IMT 21.3 ± 1.1 Placebo 20.2 ± 0.7	Male sprint athletes	DBRCT IMT N=12 Placebo N=12	6/52 IMT using POWERbreathe 30 dynamic inspiratory efforts, twice a day. Pressure threshold 50% of WI_{max}	Recovery time RRPE PRPE	↓recovery time 6.9 ± 1.3% ↓RRPE 7.9 ± 0.6% ↓PRPE 7.2 ± 0.6%
Holm, P Sutter, A Fregosi, R F (2004)		Experienced cyclists or triathletes Males = 4 Females = 16	IMT N = 10 Placebo N = 4 Control N = 6	4/52 VIH 20 sessions 30 mins Gradually increasing f_b r V_T to achieve 18-19 or RPE scale	RME V_E F_b V_T HR Approx 40km-TT	↑ RME 12% ↑ V_E 20% ↑ F_b significantly" (P = 0.013) No change V_T No change HR ↑ TT 4.7%

Study	Age (years)	Subject Data	Study Design	IMT Intervention	Measures Collected	Main findings of the effect of RMT intervention
Edwards, A M Cooke, C B (2004)		Healthy, physically active males	IMT = 10 Control = 8	4/52 IMT using POWERbreathe (IPTL) 30 dynamic inspiratory efforts, twice a day. Pressure threshold 50% of MIP	MIP Run time to exhaustion (treadmill) Blood lactate Lung function VO₂	↑ significantly (P < 0.01) ↑Run time 4.3% (P < 0.05) No change lactate No change lung function No change VO₂
McConnell, A K Sharpe, G R 2005	IMT = 23.2 ± 2.64 Placebo = 23.8 ± 4.75	Recreationally active unclear whether male vs female?	RCT N = 12 IMT N = 6 Sham N = 6	6/52 IMT using POWERbreathe 30 dynamic inspiratory efforts, twice a day. Pressure threshold 50% of MIP	MIP Cycling Power at MLSS Lactate at MLSS	↑Increased 26% Cycling Power at MLSS no change ↓Lactate at MLSS
Leddy, J J Limprasertkul, A Patel, S Modlich, F Buyea, C Pendergast, D R Lundgren, C E G 2007	RMT 29 ± 8 Control 34 ± 6	Experienced male distance runners	RCT RMT N = 15 Control N = 7	4/52 VIH RMT = 30 mins, 5 x week Control = similar protocol with no rebreathing bag.	PI_max PE_max VO₂peak Run time @ 80% VO₂peak 4 mile time	PI_max no change PE_max no change ↑VO₂peak ↑Run time @ 80% VO₂peak ↑4 mile time

DBRCT = double blind, randomised controlled study; RCT = randomised controlled trial; VIH = Voluntary Isocapnic Hyperpnoea; IFRL = Inspiratory Flow Resistive Loading; IPRL = Inspiratory Pressure Resistive Loading; WI_max = Maximum Inspiratory Work; IMT = Inspiratory Muscle Training; RMT = Respiratory Muscle Training; TT = Time Trial; P_o = Pressure generated by inspiratory muscles at zero flow; VO₂max = Maximum oxygen uptake; V_max = maximum flow; RRPE =respiratory rate of perceived exertion; PRPE = peripheral rate of perceived exertion; W_max = Maximum power; SIMT = specific inspiratory muscle training; V_Emax = maximal minute ventilation; ET = endurance training; MVV = maximum voluntary ventilation; V_T = tidal volume; SV = stroke volume; W_peak = Peak power; VO₂peak = peak oxygen uptake; VIH = Voluntary Isocapnic Hyperpnea; MLSS = maximum lactate steady state; RME = respiratory muscle endurance

Whilst it is generally accepted that IMT is effective in increasing the strength and endurance of the respiratory muscles (8 - 45.3% and 12-632% respectively) regardless of mode (see *Table 4*), some studies have failed to report positive ergogenic (performance enhancing) effects as a result of these changes (Fairburn, Coutts, Pardy, & McKenzie, 1991; Hanel & Secher, 1991; Morgan, Kohrt, Bates, & Skinner, 1987). The lack of performance changes reported by these authors may be due to methodological factors, rather than inadequacy of the intervention itself. For example, Fairburn et al. (1991) reported no change to time to exhaustion in a fixed rate (90% $\dot{V}O_{2\,max}$) ride following RMT despite changes to inspiratory muscle strength and endurance in highly trained cyclists ($\dot{V}O_{2\,max}$ = 66.1± 4.7 ml·kg·min^{-1}). However on closer inspection McConnell & Romer (2005) identified large performance changes were in fact made – for example, a mean 25% increase in time compared with 4% in the control, but the study had insufficient power due to low subject numbers (n=10) and the results failed to reach significance.

Conversely, there are a number of studies that demonstrate IMT produces a range of positive performance benefits; for example, improved cycling endurance ((24%, P < 0.01) (Markov, Spengler, Knopfli-Lenzin, Stuessi, & Boutellier, 2001), (3.26 ± 4.29%) (McMahon, Boutellier, Smith, & Spengler, 2002) (23%; P < 0.05) (Steussi, Spengler, Knopfli-Lenzin, Markov, & Boutellier, 2001)) cycling time trial time (3.8 ± 1.7) (Romer, McConnell, & Jones, 2002a), recovery time following high intensity sprints (6.9 ± 1.3%; Romer, McConnell, & Jones, 2002b), rowing (3.1 ± 0.8%; Volianitis et al., 2001), running (4.3%; P < 0.05; Edwards & Cooke, 2004) (50%; P < 0.001; Leddy et al., 2007) and swimming performance (66%; P < 0.001; Wyelegala, Pendergrast, Gosselin, Warkander, & Lundgren, 2007) (*Table 4*).

A further benefit of IMT appears to be the ability to decrease dyspnoea – another consequence of altered respiratory mechanics that may compromise exercise performance. Just as factors that impair the contractile properties of the respiratory muscles are thought to contribute to dyspnoea, factors that improve the contractile properties are thought to attenuate it (McConnell & Romer, 2004a). Inspiratory muscle training appears to be effective in decreasing dyspnoea by increasing inspiratory muscle strength and therefore enabling the inspiratory muscles to create greater length and tension changes for each breath, in response to exercise (McConnell, 2005; McConnell & Romer, 2004a; Romer, McConnell, & Jones, 2002a).

It is not clearly understood how and/or why IMT improves performance. Inspiratory muscle training has consistently been shown to have no effect on $\dot{V}O_{2\,max}$ or traditional parameters of respiratory function i.e. PEF, FEV_1 (Inbar, Weiner, Azgad, Rotstein, & Weinstein, 2000; Romer, McConnell, & Jones, 2002c). One theory receiving increasing interest is that by delaying or decreasing respiratory muscle fatigue by increasing inspiratory muscle strength and endurance, the reflex activity from the type III/IV chemo-sensitive receptors in the respiratory muscles thought to trigger sympathetic vasoconstriction in the limbs, may be attenuated (McConnell & Romer, 2004b). Witt et al. (2007) supported this theory by demonstrating a significant decrease of sympathetically-mediated heart rate (83 ± 4 vs. 74 ± 2 beats·min^{-1}) and mean arterial pressure (99 ± 3 vs. 89 ± 2 mmHg) during a eucapnic resistive breathing task designed to invoke respiratory muscle fatigue after a five week intervention of IMT. In providing an explanation for why whole-body exercise improvements are seen following IMT, these authors suggested that the attenuated cardiovascular response they observed indicates IMT causes blunted sympatho-excitation to resistive inspiratory

work, which may subsequently protect locomotor muscle blood flow. McConnell and Sharpe (2005) demonstrated a significant decrease in blood lactate concentrations (B[La]) at MLSS power (power at maximal lactate steady state) following six weeks of IMT and suggested that the ergogenic effect of IMT may be also be related to reduced B[La].

Whether IMT is effective in eliciting improved exercise performance and decreased dyspnoea by decreasing the work of breathing (McConnell & Romer, 2004b; Witt, Guenette, Rupert, McKenzie, & Sheel, 2007) or by some other means, it appears from studies to date that increasing the strength and endurance of the respiratory muscles is a common and important finding (Inbar, Weiner, Azgad, Rotstein, & Weinstein, 2000; Markov et al., 2001; Romer, McConnell, & Jones, 2002a, , 2002b, , 2002c). Interestingly, it does not appear from the methodology of any of the studies that attention is paid to the pattern of breathing used by subjects when completing their IMT. In some instances, it might also exacerbate (or reinforce) an already poor breathing pattern. A recent case history presented by Dickinson, Whyte & McConnell (2007) is the only known paper that mentions the breathing pattern that is used during IMT. These authors specifically instructed the athlete to breathe to her diaphragm and to minimise cranial (upwards) shoulder movement. With exception of the case history of Dickinson et al. (2007), IMT does not appear to specifically address dynamic hyperinflation or the recruitment order of the inspiratory muscles – rather it looks to increase inspiratory muscle strength and endurance within the positional constraints of altered respiratory mechanics. However for athletes (who are presumably able to attain optimal respiratory mechanics in the absence of respiratory pathology) it would seem advantageous to specifically address factors that are known to contribute to respiratory muscle fatigue and dyspnoea, for example dynamic hyperinflation and the respiratory muscle recruitment order by

addressing the mechanics of breathing, to decrease the work of breathing and optimise exercise performance.

2.4.3. Breathing Pattern Retraining

A strategy that specifically addresses dynamic hyperinflation and respiratory muscle recruitment pattern, and pays specific attention to breathing pattern both at rest and under load, is breathing pattern retraining (Blager, 2000; Jones, Dean, & Chow, 2003). Physiotherapists have been using breathing exercises as a treatment modality for respiratory conditions for many years, with breathing pattern retraining being recommended for breathing pattern disorders as early as 1938 (Soley & Shock, 1938). Clinically, physiotherapists used diaphragmatic breathing pattern retraining and/or pursed lip breathing[1] to relieve the symptoms and exercise limitations associated with COPD (Jones, Dean, & Chow, 2003), VCD (Blager, 2000) and BPDs (Chaitow, Bradley, & Gilbert, 2002; Lum, 1977). Diaphragmatic breathing retraining involves educating the patient of its importance, discussion of potential feelings of discomfort that may arise during the retraining process, and demonstration and instruction of how to diaphragmatically breathe correctly (Chaitow, Bradley, & Gilbert, 2002; Lum, 1977) (refer to Appendix 2 and 3 for a further description of clinical breathing pattern retraining and BPR exercises). The beneficial effects of diaphragm breathing may be due to decreased work of breathing through improved respiratory mechanics, decreased dynamic hyperinflation and increased tolerance and normalisation of $PaCO_2$ through a slower, deeper respiratory pattern (Falling, 1995; Thoman, Stoker, & Ross, 1966; Tisp et al., 1986). Pursed Lip Breathing (PLB) is taught clinically to assist airflow in patients with COPD (Jones, Dean, & Chow, 2003) and VCD (Blager, 2000) and to help elongate the expiratory phase in athletes to maintain ventilation at appropriate lung volumes (Fallon, 2004). The emphasis is on expiring

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[1] Normal inhalation with expiration through slightly parted lips, exhaling air over the bottom lip

longer than the inspiratory phase to enhance lung emptying (Jones, Dean, & Chow, 2003), avoid a tachypnoeic shift as exercise intensity increases and to avoid breath stacking. The benefits of PLB may be due to decreased respiratory rate, changes in pattern of respiratory muscle recruitment, longer t_E and larger V_T (Ito, Kakizaki, Tsuzura, & Yamada, 1999; Sharp, Drutz, & Moisan, 1980). These changes may also lead to a decrease in dyspnoea and work of breathing (ATS, 1998).

Whilst BPR studies have been conducted with success in individuals with respiratory related pathologies for example asthma (Grossman, DeSwart, & Defares, 1985; Ram, Holloway, & Jones, 2003; Thomas et al., 2003), HVS (Grossman, DeSwart, & Defares, 1985) and VCD (Banez & Culbert, 2005) no study has been published detailing the potential efficacy of such techniques in athletic populations. This is surprising given the importance of optimal breathing in athletes, the numerous factors that can compromise breathing in athletes and the consequence of these on respiratory mechanics and, ultimately, exercise performance. In cyclists, breathing may be compromised by the presence of a BPD at rest, alterations to respiratory timing and pattern caused by the normal respiratory response to exercise, psychological and postural factors, resulting in an inefficient breathing pattern. It has been shown that an inefficient breathing pattern may increase the work of breathing (Jones, Dean, & Chow, 2003). Increased work of breathing may cause blood stealing (Harms, Wetter, St Croix, Pegelow, & Dempsey, 2000), decreased power output (Romer & Dempsey, 2006), increased dyspnoea (McConnell & Romer, 2004a) and decreased exercise time (Harms, Wetter, St Croix, Pegelow, & Dempsey, 2000). Optimising a cyclist's breathing pattern could potentially decrease the work of breathing such that blood stealing is delayed or prevented, power output is maintained for longer, and a decrease or delayed perception of dyspnoea. Dyspnoea has been identified as one of the limiting factors to exercise performance (Aliverti & Macklem, 2001). If dyspnoea could be delayed or prevented, this could enable cyclists to both

train and compete at a greater intensity. In elite cyclists the margins for improvement through training alone is thought to be 1-3% (Jeukendrup & Martin, 2001) and in a 40km-TT this small alteration could decrease race time by up to 1.33 minutes. If dyspnoea could be minimized, higher quality, more intense and longer training sessions might be possible and the physiological benefits of training could be maximised.

Although diaphragmatic breathing pattern retraining and PLB are used clinically, there are no studies reported in the scientific literature that investigate their use in the healthy, athletic population and the true effect of these breathing exercises in respiratory pathology is unclear. For example, Gosselink et al (1995) demonstrated that diaphragmatic breathing reduced rather than enhanced breathing efficiency in patients with severe COPD, concluding that diaphragmatic breathing contributed to inappropriate chest wall motion and decreased mechanical efficiency while increasing dyspnoea. Conversely, PLB when combined with diaphragmatic breathing has been shown to enhance pulmonary mechanics and breathing efficiency, relieve dyspnoea, slow the respiratory rate, increase tidal volume and help to restore diaphragmatic function (Falling, 1995; Thoman, Stoker, & Ross, 1966; Tisp et al., 1986). These are all changes that would theoretically assist athletes in containing the work of breathing, and the consequences associated with this, and potentially enhance exercise performance.

More recently, Jones, Dean and Chow (2003) investigated the differences in $\dot{V}O_2$ and respiratory rate during three commonly described breathing exercises (PLB, diaphragmatic breathing and a combination of the two) and spontaneous breathing at rest in patients with COPD. Subjects (N=30) were 'experienced' in all three breathing exercises and could perform them on command. Over a

ten minute recording period, respiratory rate was decreased from 17.3 ± 4.23 breaths/min in spontaneous breathing to 15 ± 4.32 in diaphragmatic breathing, 12.8 ± 3.53 during PLB and 11.2 ± 2.7 during combination. $\dot{V}O_2$ decreased significantly from 174.5 ± 25.2 mL·O_2·min^{-1} in spontaneous breathing to 165.8 ± 22.3 in diaphragmatic breathing, 164.8 ± 20.9 in PLB and 167.7 ± 20.7 in combination breathing indicating that people with COPD adopt a breathing pattern at rest that is not associated with the least $\dot{V}O_2$. Whilst significant, the differences observed by Jones et al. (2003) were recorded at rest, and were only small changes. Theoretically the differences between spontaneous breathing and the different breathing patterns could be larger at higher workloads, when more efficient breathing patterns would be desirable. The findings of Jones et al. (2003) may suggest that the components of respiratory mechanics and respiratory muscle efficiency may play a greater role in supporting spontaneous breathing than simply minimising $\dot{V}O_2$. Interestingly, despite the fact that all patients were "experienced" in the techniques and were able to adopt the required breathing patterns on command, none of the patterns were adopted by subjects as their automatic breathing pattern, despite the apparent improved efficiency. This could indicate that subjects had only learnt the patterns as a motor skill, not achieved the subconscious level of attainment necessary to reprogram the respiratory centres.

An explanation for the variable results in studies investigating breathing retraining in COPD may be due to lack of 'reprogramming' or 'resetting' of diaphragmatic breathing and PLB as the default or automatic breathing pattern. No indication was given in either study regarding the length of the intervention period. It is possible that subjects had 'learnt' the techniques and could voluntarily reproduce them at will, but were unable to maintain these patterns indefinitely therefore reverted back during spontaneous breathing. According to Grossman et al. (1985), unless the respiratory

centres are reprogrammed, patients can only tolerate small decreases in respiratory rate and increased CO_2 can be tolerated for approximately 10 minutes before the urge to revert to spontaneous breathing becomes overwhelming[2]. It is possible that neither Gosselink et al. (1995) or Jones, Dean and Chow (2003) incorporated a sufficient intervention period to allow central adaptation to occur. Maclennan et al. (1994) and Fabre et al. (2007) attempted to investigate the effect of entraining breathing with exercise during rowing and roller-ski skating respectively. Neither author provided an intervention period for participants to adjust to the new breathing pattern, testing time was greater than 10 minutes, and unsurprisingly neither author identified positive changes to breathing efficiency or perception of respiratory effort as a result of entrainment. It seems apparent that regardless of the mechanisms underpinning *how* breathing patterns are 'retrained', an unknown period of time is required for the new breathing pattern to become the automatic pattern and therefore become beneficial. This time frame may be longer in athletes who not only need to adjust to a new breathing pattern at rest, but also need to assimilate it to their exercise environment and associated increased ventilatory demand.

Another factor that needs to be considered when analysing studies of breathing pattern retraining in COPD is that often the altered breathing pattern is the effect of an impairment in respiratory mechanics due to the disease process, rather than the symptoms of COPD (for example dyspnoea and hyperinflation) being caused in the first instance by altered respiratory mechanics. Healthy humans who have no underlying respiratory pathology should theoretically have no need for altered respiratory mechanics and should therefore have the ability to achieve an optimal breathing pattern.

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[2] The concept of respiratory centres being 'reprogrammed' is only a theoretical notion. The precise mechanisms underpinning how breathing pattern is altered in a BPD, and subsequently 'retrained' to demonstrate optimal respiratory mechanics, is not known.

At this stage there is insufficient evidence, predominantly due to poor methodology of those studies that have been undertaken, to strongly support or reject the use of breathing pattern retraining in athletes or healthy humans. For example, Lum (1983) reported the effectiveness of breathing retraining offered by physiotherapists to patients with hyperventilation syndrome (a chronic breathing pattern disorder). In a group of more than 1000 patients, 80% became symptom free. However, the value of this study is limited as no details were given on the exact nature of the treatment or the instruments used for evaluating therapeutic outcome and no control was used. Small subject numbers, lack of control, unclear separation of treatment modalities and poor outcome measures are weaknesses of the six studies critiqued (*Table 5*) that have investigated the effect of breathing pattern retraining in individuals with breathing pattern disorders.

Table 5. Overview of Breathing Pattern Retraining Studies

Study	Treatment Group Intervention (N)	Control Group Intervention (N)	Outcome measure	Findings
Lum (1983)	N = 1350 Intervention unclear	Clinical judgement		80% symptom free
Grossman et al. (1985)	AR 7 sessions N = 5	BI 7 sessions N = 22	Self-report questionnaires e.g. STAI, EPI Respiratory measures	AR > BI, on psychological measures at 1 month post treatment
Van Doorn et al. (1982)	BF 7 sessions N = 10	BI 4 sessions N = 10	Self report HVS symptoms, $PetCO_2$	BF > BI on HVS symptoms at post-treatment BF = BI on HVS symptoms at 3 month follow up BF = BI on $PetCO_2$ at post treatment and follow up
Kraft & Hoogduin (1984)	1. Voluntary hyperventilation N = 6; 6 sessions 2. Relaxation breathing retraining N = 6; 6 sessions 3. Stress management N = 7; 6 sessions	No control	Self report HVS symptoms, HVS attacks, depression	1 = 2 = 3, on psychological measures, at post-treatment and one month follow up
Rapee (1985)	B 3 sessions N = 1	No control	Self report panic attacks, anxiety, physical symptoms	Reduction in panic attacks, symptoms and anxiety
De Ruiter et al. (1989)	B + E 8 sessions N = 13	1. E 8 sessions N = 13 2. B + C + E 8 sessions N = 13	Self report panic attacks, agoraphobia, PCO_2, breathing frequency	B + C = 1 = 2, on psychological and respiratory measures at post treatment

AR = auditory regulation; BI = breathing instruction; BF = biofeedback; R = relaxation; C = cognitive restructuring; B = breathing retraining; CT = cognitive therapy; E = exposure;

STAI = State-Trait Anxiety Inventory; AR > BI = auditory regulation scored significantly better than breathing instruction, $PetCO_2$ = End-tidal CO_2 pressure

Clearly there is a paucity of well-designed studies that investigate the effect of breathing pattern retraining in individuals with breathing pattern disorders, or on altered respiratory mechanics. In addition to the methodological issues discussed above, nothing can be found in the scientific literature to indicate how long each individual training session needs to be, the optimal frequency of sessions per day or per week, or total number of weeks an intervention should be followed in order to retrain a persons automatic breathing pattern. Further, and more specifically, no studies have been identified that investigate the effect of breathing pattern retraining on optimising respiratory mechanics in healthy athletes. Given the high demands placed on the respiratory system during racing and intense training, and the potential for maladaption in the person's mechanics of ventilation highlighted above, it has been previously suggested that optimising an athlete's breathing pattern would have beneficial effects on cycling performance (Lucia, Hoyos, Pardo, & Chicharro, 2001).

2.5. Summary

Competitive road cycling is a sport where very small improvements in physiology can have a significant affect on overall performance. Much attention is given to find ways that may enhance a rider's mean power during time trials and peak aerobic power during incremental endurance exercise.

An area that has not been explored to date is the mechanisms of breathing and the effect that optimising a cyclist's breathing pattern may have on performance. This is surprising given the significant increases in ventilatory demands observed during maximal intensity cycling. To meet these ventilatory demands both breathing frequency and tidal volume are altered. In most athletes this is achieved by shortening the expiratory time rather than the inspiratory time for each breath – a change that is associated with a tachypnoeic shift, breath stacking, dynamic hyperinflation and a subsequent increase in the work of breathing. Increased work of breathing, particularly to the point of respiratory

muscle fatigue, has been associated with decreased blood flow to the leg muscles during high intensity cycling, a consequence of which is a decreased power output and a drop in performance.

If increased work of breathing has a negative effect on cycling performance, it would be reasonable to argue that strategies that decrease the work of breathing may have a positive effect. Whilst respiratory muscle training has been shown to decrease the work of breathing and increase exercise performance, it fails to specifically address the pattern of breathing. Breathing pattern retraining is an intervention strategy used by physiotherapists to decrease the work of breathing by decreasing dynamic hyperinflation and delaying the onset of respiratory muscle fatigue. Theoretically, optimising an efficient, diaphragmatic breathing pattern in cyclists would serve to decrease the work of breathing, preserve blood flow to the legs, decreases perceptions of effort which collectively would enable a cyclist to maintain power output.

Despite the apparent benefit to cycling performance, no studies have investigated, to date, the effect that breathing pattern retraining could have on performance in cycling. Given the strength of theoretical argument presented in this review and the lack of existing research into the area of breathing pattern retraining in cyclists, an investigation into the effect of breathing pattern retraining on exercise performance in cyclists is clearly warranted.

Chapter Three: Methods

3.1. Participants

Twenty-four competitive male cyclists and triathletes from various cycling and triathlon clubs within the greater Auckland area were recruited for this study by flyer distribution and direct contact to clubs (Intervention Group N = 12, Control Group N = 12; match paired on pre-intervention 20km-TT time). Two participants withdrew from the intervention group during the study – one due to an acute respiratory illness, the other due to the inability to comply with standardising his training schedule. All participants were informed of the risks associated with the testing and the requirements of participation both verbally and in written form, and were given the opportunity to have any questions answered. Prior to participation, all participants gave their written informed consent in accordance with Auckland University of Technology's Ethics Committee guidelines and completed a medical questionnaire.

Since it is not possible to teach a person "sham breathing" without affecting their breathing pattern in some way, some deception as to the true aims of the study was applied. In order to have a true control group, full disclosure of the real purpose of the study was not provided to participants at its onset. Further written information about the true study purpose was provided and written consent was gained from the participants randomised into the intervention group. All participants were aware they could withdraw from the study at any time with no consequences.

Participants' characteristics are presented in *Table 6*. The majority of participants competed regularly in club level races or higher, in either road cycling or triathlon. Participants were excluded from the study if they were current smokers, had suffered an acute respiratory illness or injury that had affected training within the previous four weeks, or had previously received any formal breathing retraining or advice.

The study took place in the competitive phase of the athlete's season. Participants were training and competing regularly at the time of the testing, with an average riding volume of 246 ± 121.8 km per week (range 145-580 km). Training volume over the period of the study was not set by the investigator, however a requirement of inclusion in the study was that participants were in a stable phase of training and were requested to keep their training volume consistent over the duration of the study. To monitor the actual training load throughout the study, participants were given a heart rate monitor (PolarS625x; Polar Electro Oy, Kemplele, Finland) and a training diary (Appendix 4) to record training data.

Table 6. Baseline Participant Anthropometric Characteristics

	Intervention*	Control*
Age (yr)	38.6 ± 5.9	37 ± 10.6
Body Mass (kg)	78.3 ± 11.2	84 ± 10
Height (cm)	176.1 ± 6	182 ± 4.3
Years Competing	5.3 ± 2.8	3.9 ± 3
Hours per week training	8.8 ± 2.3	7.3 ± 4.1

*All values are mean ± SD

3.2. Equipment

3.2.1. Ergometer

An electro-magnetically braked cycle ergometer (Velotron, Racemate Inc, Seattle, USA) was used for all physiological assessments and sub-maximal rides during the intervention period. This model of ergometer utilises an electro-magnetic braking system to increase the amount of force applied to the rear wheel of the machine, where the assessor can predetermine the distance required to complete (as with the 20km-TT), or the level of resistance (i.e. power output) on the flywheel, independent of pedaling cadence (as with the incremental step test). This ergometer allowed for the design of different 'protocols' or 'courses', which in this study were the incremental step test and sub-maximal intervention period rides, and the 20km-TT respectively.

3.2.2. Pulmonary Gas Exchange and Spirometry

An automated breath-by-breath system (Metamax 3B, Cortex, Leipzig, Germany) was used to record pulmonary gas exchange measures during the pre- and post-intervention incremental test. The same system was used to perform spirometry assessment in the same sessions. This system has a proven reliability and validity when compared to the Douglas bag as the traditional gold standard measurement (Larsson, Wadell, Jakobsson, Burlin, & Henriksson-Larsen, 2004).

Calibration of the gas analysis system was performed prior to and immediately after all assessments, using a two-point calibration procedure. This involved calibrating the apparatus to ambient air, which was assumed to be 20.93% O_2 and 0.03% CO_2, and then to a known mixture of high-tolerance calibration gas (BOC, Auckland, NZ), composed of 14.82% O_2 and 4.80% CO_2. The two-point calibration and ambient air checking procedure was repeated until acceptable values (± 0.02%) were reached. Following the gas calibration, the flow-volume transducer was calibrated using a three litre syringe (Hans Rudolph, US). This calibration was verified with three different ventilation rates, according to the manufacturer's instructions.

3.2.3. Blood Lactate

Blood lactate concentration was measured using the Lactate Pro lactate analyser (Akray, Tokyo, Japan). Studies by Pyne, Boston, Martin, and Logan (2000) and Buckley, Bourdon and Woolford (2003) support the accuracy, reliability and versatility of this device when compared to other devices such as the Abl 700 Series Acid-Base Analyser 2, the Accusport Lactate Meter, the YSI 2300 Stat lactate analyser and the YSI 1500 Sport analysers.

Prior to each use, calibration of the lactate pro device was checked using the magnetic strip provided by the manufacturer of the lactate testing strips. Blood samples were taken from the right ear lobe after the

lobe had been cleaned with an alcohol swab to ensure an adequate blood sample was returned, and that this sample was not contaminated by sweat. The same Lactate Pro device was used for all participants.

3.2.4. Borg Scale

Rating of perceived effort for leg and breathing effort was recorded using the modified Borg Scale (MBS) (Appendix 5). This rating scale (0-10) was used in the incremental power test and the 20km time trial. Although the Borg scale is typically used for patients with respiratory disease, normal healthy people with exercise induced dyspnoea are also able to distinguish between different levels of breathlessness using the MBS (Wilson & Jones, 1989). The same scale, modified to reflect leg effort rather than breathlessness, was used so as not to confuse participants with different scales.

3.2.5. Nijmegen Questionnaire

The Nijmegen Questionnaire (Appendix 1) was used as a general screening tool to identify the presence of an underlying breathing pattern disorder (BPD) in the participants. The Nijmegen Questionnaire is a list of sixteen signs and symptoms that, grouped together, are used as a screening tool for anxiety-based BPDs. There are no specific objective measures to identify abnormal respiratory mechanics or BPDs in athletes. The sensitivity of the questionnaire in relation to the clinical diagnosis of anxiety-based BPDs is reported to be 91% and the specificity 95% (Van Dixhoorn & Duivenvoorden, 1984).

3.2.6. Breathing Harness

A newly developed breathing harness (Zephyrtech, Auckland, New Zealand) was used with the control group during the intervention period. The harness is worn as two straps, one around the upper chest and the other around the abdomen (Figure 4). Each strap has a sensor that detects expansion and

contraction of the underlying thoracic cavity. The upper chest sensor is positioned on the lateral aspect of the chest wall, and the lower sensor is positioned midline, on the upper abdomen.

Figure 4: Breathing Harness

The harness was used to differentiate between upper and lower chest breathing. Each of the sensors capture the amplitude of the expansion and contraction of the ribcage during inspiration and expiration respectively, and this data is presented in real time on a computer screen. The breathing harness is still in the developmental stages therefore there is no reliability or validity data to support its use. However, because the primary reason for using the harness was to create a "research purpose" for the control group (see Intervention Sessions - Control Group, 3.3.8.), this lack of data was not deemed important.

3.3. Experimental Procedures

3.3.1. General Protocol

All tests were performed at AUT University's Human Performance Laboratory located at the Millennium Institute of Sport and Health, Auckland, New Zealand. The laboratory was well ventilated and environmental conditions were standardised. The laboratory was temperature-controlled within the range 19-21°C, and humidity was consistently in the range of 60-75%.

Participants were required to attend the laboratory ten times over a period of seven weeks (Figure 5). During session one the medical prescreening questionnaires were analysed, height and body mass (in their riding clothes, without shoes) were recorded and participants underwent pulmonary function testing (spirometry), immediately followed by a familiarisation incremental power step test. During session two participants performed a 20km time trial (TT) familiarisation test. Sessions three and four were the formal baseline (pre-intervention) sessions for spirometry and the incremental power test, and the 20km-TT test respectively. Following baseline assessments, participants completed the intervention (sessions 5-8), then post-intervention assessments (sessions 9 and 10) in the order above.
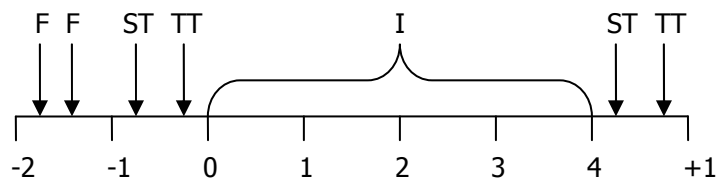
Figure 5. Time line (weeks) of familiarisation trials (F), formal incremental step test (ST) and 20km time trial (TT) and intervention period (I).

The incremental step test (including spirometry) and 20km-TT (for each of the familiarisation, pre- and post-intervention testing sessions) were completed at least 48 hours apart. To minimise sources of variation, the time between each of the tests, time of day and day of the week were kept consistent for each participant between their pre- and post-intervention test sessions.

Prior to the first assessment the participant's own bike dimensions were measured and recorded so that the cycle ergometer resembled the riders preferred bike setup as closely as possible. This included the height, reach and angle of the handle bars, as well as the height, fore and aft position, and angle of the seat. Additionally, participant's own pedals were attached to the cranks to allow them to ride in their own shoes. Prior to the first assessment participants were able to request changes to any of the bike's dimensions during their warm-up. Once confirmed, this setup was recorded and held constant for all future testing sessions.

To minimise the effect of nutrition on performance, food and fluid intake were kept consistent for pre- and post-testing. Participants were instructed to submit a food diary of the food and fluid consumed and at what time during the 24 hours preceding the formal incremental power and 20km-TT pre tests. This food diary was returned to participants for replication prior to their post-intervention tests. Participants were instructed not to consume any caffeinated beverages in the 12 hours preceding each test and to abstain from exhaustive or prolonged exercise for at least 48 hours prior to each test. All participants claimed to have adhered to these criteria.

3.3.2. *Spirometry*

Each participant's vital capacity (VC), forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), peak expiratory flow (PEF), maximum voluntary ventilation (MVV), inspiratory and expiratory lung volumes (IRV and ERV respectively) and tidal volume (V_T) were determined in accordance with American Thoracic Society guidelines (Miller et al., 2005). For all tests, participants sat in an upright position and wore a nose-clip to eliminate nose breathing. Participants completed three different procedures with a small rest (one to two minutes) between each test. A procedure was stopped and repeated if a participant bent forward at the waist in an attempt to maximise the expiratory phase, or if there were symptoms suggestive of upper airway occlusion during the maximal flow-volume loop procedure (such as pronounced inspiratory stridor, cough or expiratory wheeze).

The first procedure required participants to be quietly seated with the mouth piece and nose clip in situ, breathing normally. When they were ready a maximal inhalation was taken, followed by a controlled but maximal exhalation. Normal tidal breathing with the mouth piece and nose clip still in situ was commenced immediately after the maximal exhalation. After three or four tidal breaths the maneuver

was repeated. This procedure was repeated three times before the mouth piece was removed. The highest values for VC, IRV, ERV and V_T were recorded from this procedure.

The second procedure (maximal flow-volume loop) required a maximal inhalation to be taken with the nose clip on but *without* the mouth piece in situ. The participant then inserted the mouth piece at the peak of the inhalation, ensuring a firm seal was achieved, and exhaled as hard, fast and completely as possible. When the participant felt they could not exhale any further (approximately 6s) a forceful, fast and maximal inspiration was performed. When no further inhalation could be taken the participant removed the mouth piece and breathed normally until they were ready to repeat the procedure. This procedure was repeated until three consistently reproducible efforts were achieved (within 0.15L of each other; Miller et al., 2005)), as determined by computer analysis. The highest values for FVC, FEV_1 and PEF were recorded from this procedure.

The final spirometry measure was the MVV test. In the same testing position used above, participants were instructed to "breathe as hard, fast and deeply (in and out) as possible" for 12 seconds. Participants were instructed to sit quietly after completion of the 12 seconds and recover their breathing. They were advised prior to the test that they may feel dizzy or light headed during the last few seconds or on completion of this test and that this feeling would soon pass. Lucia et al (1999) in their study with competitive elite cyclists measured FVC and MVV in a standing position. Although the present study was also done with cyclists it was decided to use a seated position for two reasons: a) cycling is performed in a seated (albeit forward leaning) position so it seemed more appropriate to and b) since the MVV test is a maximal hyperventilatory test it is not uncommon for participants to feel faint or light headed during this test and it was simply deemed safer to have participants in a seated rather than standing position.

3.3.3. Incremental Power Step Test

Each participant's peak oxygen uptake ($\dot{V}O_{2\ peak}$), lactate threshold (LT), lactate turn point (LTP) and maximum aerobic power (P_{max}) were determined using an incremental step test to volitional exhaustion. Prior to this test participants had completed spirometry procedures, as described above. The face mask was then attached, ensuring a firm seal with no air leaks. The ergometer was calibrated in accordance with the manufacturer's instructions and when acceptable variations were achieved (<1%) participants commenced their 6 minute self selected warm-up. Participants did not have to use the whole 6 minutes and were able to perform any final stretches before commencing the test. The self selected warm up for the pre-intervention tests was recorded for each participant, and repeated prior to their post-intervention tests. Final calibration checks of the gas analysis system were performed during this warm-up period and recalibration was performed if required.

The incremental test for most participants started at 150W and consisted of a continuous step test protocol with three minute stages. However, to cater for a range of fitness levels, the starting power was adjusted to ensure that each participant completed at least seven but not more than nine stages. The starting power for each participant was the same for the pre- and post-test. Power output was progressively increased by 30W at the end of each three minute stage until exhaustion. The test was terminated when the participant stopped completely, or when the participant could no longer hold a cadence above 60 rev·min^{-1}. Participants were instructed to stay seated throughout the duration of the test, and to try to maintain a consistent cadence. A designated set cadence was not used for either the incremental test or the 20km-TT since each cyclist develops power at his own pedaling rate (Zavorsky et al., 2007) and a fixed cadence may not have resulted in an accurate reflection of a participant's maximum aerobic power.

Prior to commencement of the test participants were shown the modified (0-10) Borg scale (Kendrick, Baxi, & Smith, 2000), its use was explained to them and they had time to become familiar with it. Twenty seconds before the end of each stage participants were asked to rate their perceived leg effort, followed by their breathing effort. This order was maintained during all testing, to ensure participants were clear whether they were describing their leg or breathing effort. In addition, a blood lactate sample was taken from the participant's right ear lobe in the last 15 seconds of each stage. Heart rate and respiratory rate were also collected during this time frame.

Consistent feedback was given to each participant at the end of each power stage. Maximal verbal encouragement was given to each participant when it was obvious they were in the final stage of their test. There were always two assessors (primary investigator and research assistant) present for each test. To avoid bias in the feedback, and therefore to avoid the influence of externally motivating factors, the assistant assessor provided the maximal encouragement in the last stage and was blinded as to whether the participant had been in the control or intervention group.

3.3.4. Nijmegen Questionnaire

The Nijmegen questionnaire was completed prior to the pre-intervention 20km-TT. As no anxiety based BPDs were identified using this tool in either group, the questionnaire was not repeated post-intervention. The primary investigator read out each of the 16 signs and symptoms listed in Appendix 1 and participant was asked to rate them in accordance with the frequency chart provided (Appendix 1), over the previous six month period. Participants were only told that it was a breathing related questionnaire, not one that was specific to anxiety.

3.3.5. 20km Time Trial

Laboratory based time trials (TT) of varying distances have previously been shown to be both reliable and valid measures of cycling performance (Laursen, Shing, & Jenkins, 2003). More recently, Zavorsky et al (2007) has demonstrated that a familiarisation session improves the reliability of the formal pre- and post-testing measurements. A familiarisation TT followed by a second TT has a larger CV than that observed between the second and a third TT. Given that the smallest worthwhile meaningful change can be as little as 0.5-1.5% (Paton & Hopkins, 2001) it was considered important to include a familiarisation trial so as not to confuse typical error with performance improvement. Therefore, over the course of the study participants complete three 20km-TT's – a familiarisation trial, and both a pre- and post-intervention trial.

Participants presented to the laboratory at least 48 hours after their incremental power test, having followed the same guidelines as the incremental test with respect to exhaustive exercise, food and caffeinated beverages. The TT protocol allowed participants to change gears like they would on a road bike. Prior to the familiarisation trial participants had the opportunity to become familiar with the gear changing and to establish what gears they would like to use.

After calibration of the ergometer and a six minute self-selected warm-up (consistent for pre- and post-testing), participants were asked to bring the ergometer to a stop, so a "standing start" (seated, with no movement of the cranks) could be achieved. The ergometer had been programmed to run a 20km-TT protocol, such that when the program was started (five second count down for participants) the time clock ran until automatically stopping once the 20kms had been reached. Participants were able to select their own gearing and cadence and could alter either of these as needed throughout the test. Participants remained seated throughout the entire TT, with their hands on either the brake hoods or the top bar. These riding positions were chosen, as opposed to the aerodynamic position, to enable a

better comparison to the breathing patterns observed by Lucia et al. (1999) who also used these testing positions. Water was available as required.

At five minute intervals HR, power, speed, leg and lung RPE were recorded. Standardised verbal encouragement was given to participants at the 5, 10, 15, 19 and 19.5 km mark (for example "that's 5km, going well"). Standardised maximal verbal encouragement was given in the last 500m of the test.

3.3.6. Intervention Sessions - Randomisation

In an attempt to have two even groups based on performance measures, participants were match paired based on their pre-intervention 20km-TT time. Randomisation to either the intervention or control group was done by the toss of a coin, such that the individual in the pair whose christian name was alphabetically first was a "head", the other "tails". Whoever corresponded with the side of the coin that landed face up was put into the intervention group.

3.3.7. Intervention Sessions - Intervention Group

During session five, participants in the intervention group were told the true purpose of the study (i.e. that the investigators were interested in whether breathing pattern could be retrained in cyclists) and given a concise but comprehensive overview of efficient versus inefficient breathing, the consequences of each, the basic mechanics of breathing and how breathing can be retrained. In an attempt to minimise the placebo effect on the outcome of the study participants were not told that the *effect on performance* of changing respiratory pattern was of primary interest to the investigators. Participants were taught the two breathing exercises (static exercises) (Appendix 3) to be completed at home. The ways in which the static breathing exercises differed from the ideal breathing pattern for on the bike were discussed and demonstrated (dynamic exercise) but the participant did not perform the dynamic exercises during this session. Participants had the opportunity to have any questions answered and

were given the opportunity to withdraw from the study if they did not wish to continue with the new study protocol. Participants were provided with a CD with spoken instructions and a counted rhythm for the 10 minute static breathing exercise, 10 gold star stickers to be used as visual triggers, and written hand outs of the exercises (Appendix 3), potential symptoms they may experience during BPR (Appendix 6) and main points that were discussed during this session (Appendix 7).

During session six, participants demonstrated their 10 minute static breathing exercise technique and feedback was provided with appropriate changes made. Following this, participants completed a 10 minute sub-maximal ride (60% of P_{max}) during which they were taught to apply the diaphragmatic pattern with emphasis on the expiratory phase (dynamic breathing exercise). Sessions seven and eight each involved a 10 minute sub maximal (60% of P_{max}) ride with two 30 second maximal efforts. During each maximal effort the emphasis was on breathing control and on recovering one's respiratory rate when the effort was over.

Participants were encouraged to continue using their dynamic breathing skills during training in addition to their static breathing exercises.

3.3.8. Intervention Sessions - Control Group

Participants who were randomised into the control group were led to believe the purpose of the study was twofold:

1) to investigate the effect that breathing pattern had on performance in cyclists

2) to test the reliability and validity of a newly developed breathing harness.

Over the four weeks of the intervention period participants completed one 10 minute sub maximal (60% of P_{max}) ride each week wearing the breathing harness. Participants were asked to rate their leg and breathing RPE at five and ten minutes.

When participants wore the breathing harness for the first time they were shown how it worked and were able to view the computer screen to observe the different amplitudes of the apical and diaphragmatic breaths in real time. However, during the sub-maximal rides the screen was turned away from them. Participants were told that this was to prevent them from pacing or consciously altering their breathing. No formal data was collected from the breathing harness, or from the participants, during this intervention period. No feedback on adopted breathing pattern was given to the control group.

3.4. Data Analysis

3.4.1. P_{max} determination

As the stages of the peak power incremental test were three minutes in duration, final maximum power (P_{max}) was measured as the participants' last fully completed three minute stage, plus 0.16W for each additional second completed of the next power stage.

3.4.2. Lactate

The LT was defined as the first deflection point of 1 mmol·L^{-1} or greater above baseline (Yoshida, Chida, Ichioka, & Suda, 1987) in the Lactate (B[La]) vs. power output curve. LTP was defined as the point in the B[La] vs. power output curve before the observation of a second sudden and sustained increase in B[La] that coincided with a B[La] of between approximately 2-5 mmol·L^{-1} (Davis, Bassett, Hughes, & Gass, 1983). All LT and LTP analyses were performed by two independent examiners, and where discrepancies occurred, a third examiner was used.

3.4.3. Sub-maximal and maximal VO_2

$\dot{V}O_{2\,max}$ represents the maximum rate of oxygen that can be inhaled and delivered to and used by the working muscles during maximal exercise (Astrand & Rodahl, 1986; Saltin & Astrand, 1967). The $\dot{V}O_{2\,max}$ is often identified by a plateau in $\dot{V}O_2$ despite continued increases in exercise intensity.

However, a plateau in $\dot{V}O_2$ is not always observed (Doherty, Nobbs, & Noakes, 2003; Lucia et al., 2006) and along with the finding that different protocols (e.g. discontinuous step versus continuous ramp) and different exercise modes (e.g. cycling versus running) may yield different maximal results, (Lucia et al., 2006) it has been proposed that it may be more accurate to term this the peak, rather than the maximum, $\dot{V}O_2$ (Day, Rossiter, Coats, Skasick, & Whipp, 2003); i.e. the $\dot{V}O_{2\,peak}$. For the purpose of this study, $\dot{V}O_{2\,peak}$ was defined as the peak value achieved during the incremental assessment, averaged over a 30 second period. To quantify the O_2 cost of cycling (cycling economy) the mean $\dot{V}O_2$ in the last 30 seconds of each three minute stage was determined.

All $\dot{V}O_2$ and $\dot{V}CO_2$ data was recorded breath-by-breath, and to reduce the effect of breath-by-breath variability, outliers were removed using in-built functions in the Cortex software, thereby reducing the 'noise'. For determination of $\dot{V}O_{2\,peak}$ data during the incremental assessment was averaged on a 30 second basis. Each participant's absolute $\dot{V}O_{2\,peak}$ was recorded as the highest 30-second $\dot{V}O_2$ value obtained during the test.

3.4.4. Nijmegen Questionnaire

The Nijmegen Questionnaire is scored based on the frequency that a participant experiences the described signs and symptoms (Appendix 1). The maximum score is 64, and a score greater than or equal to 23/64 is considered indicative of an anxiety based breathing pattern disorder.

3.4.5. Statistics

Once individual values had been obtained they were entered into an MS Excel statistics spreadsheet designed by Hopkins (2003) and values were log-transformed. To make inferences about true (population) values of the effect of BPR on cycling performance, the uncertainty in the effect was expressed as 90% confidence limits and as likelihoods that the true value of the effect represents substantial change (Batterham & Hopkins, 2005). An effect was deemed unclear if its confidence interval overlapped the thresholds for substantiveness; that is, if the effect could be substantially positive and negative. An estimate of the smallest substantial change in power output was required to make these inferences. Paton and Hopkins (2001) estimated smallest effects of 0.5-1.5% in mean power, based on variability in competitive performance of elite cyclists in various time trials where drafting and group tactics did not contribute. A smallest worthwhile effect of 1.0% was assumed. Pearson correlations were performed to identify the strength of relationship between changes in RPE, f_b, P_{max}, 20km-TT speed$_{AVE}$ and 20km-TT time.

Chapter Four: Results

4.1. Participant Characteristics

The baseline performance and physiological characteristics of the 22 participants are presented in *Table 7.*

Table 7. Base Line Performance and Physiological Characteristics for Intervention and Control Groups*

Measure	Intervention (N=10)	Control (N=12)
20km-TT time (min:sec)	32:31 ± 1:31	32:36 ± 1:07
20km-TT P_{AVE} (W)	263 ± 5	269 ± 8
20km-TT $Speed_{AVE}$ (km·hr-1)	37.5 ± 0.8	37.8 ± 0.9
P_{max} (W)	331 ± 33	338 ± 31
P_{max} (W·kg -1)	4.2 ± 0.7	4.1 ± 0.6
VO_{2peak} (L·min-1)	4.3 ± 0.6	4.4 ± 0.5
$VO_{2\ peak}$ (ml·kg·min-1)	55.3 ± 8.5	53 ± 7.5
HR_{peak} (b·min-1)	181 ± 10	184 ± 11
$B[La]_{peak}$ (mmol·L)	10.2 ± 1.5	13.2 ± 1.8
Power at LT (W)	193 ± 26	180 ± 33
Power at LT (%W_{max})	60.1 ± 7.4	54.2 ± 6.4
VO_2 at LT (L·min-1)	2.9 ± 0.5	2.9 ± 0.4
VO_2 at LT (% VO_{2peak})	68.5 ± 6.4	65.5 ± 5.6
HR at LT (b·min-1)	140 ± 12	135 ± 12
HR at LT (% HR_{peak})	77 ± 4	74 ± 5
f_b at LT (breaths·min-1)	29 ± 6	30 ± 6
Power at LTP (W)	270 ± 40	257 ± 39
Power at LTP (%W_{max})	81.6 ± 6.5	75.5 ± 6.3
VO_2 at LTP (L·min-1)	3.7 ± 0.6	3.7 ± 0.4
VO_2 at LTP (% $VO2_{peak}$)	87.2 ± 5.5	83.6 ± 6.5
HR at LTP (b·min-1)	165 ± 13	158 ± 13
HR at LT (% HR_{peak})	91 ± 4	87 ± 3
f_b at LTP (breaths·min-1)	35 ± 5	38 ± 8

*All values are mean ± SD

The Nijmegan Questionnaire did not identify any anxiety-based breathing pattern disorders in either group, with the mean scores for both the intervention group (6.4 ± 5.06) and the control group (8.55 ± 3.75) being well below a score of 23 necessary to be indicative of an anxiety-based breathing pattern disorder.

Attendance to each intervention session was high for both the intervention (100%) and control (98.3%) groups. Compliance with the BPR was 89%, and all participants were able to accurately and appropriately perform their static and dynamic exercises, indicating they had practiced regularly.

4.2. Effects on cycling performance (Incremental power and 20km-TT performance)

Table 8 shows the effect of BPR on cycling performance. After 4 weeks of BPR there were substantial changes to P_{max} (3.2 ± 3%) in the incremental step test and to P_{ave} (3.1 ± 2.9%), average speed (1.3 ± 1.2%) and time (-1.5 ± 1.1%) in the 20km-TT.

Table 8. Mean Changes in Performance Measures post Breathing Pattern Retraining and Control and Chances That the True Difference in the Changes Is Substantial

| | Change in Measure* | | | | |
	Intervention (N=10)	Control (N=12)	Difference ± 90% CL	Cohen's ES ± 90% CL†	Practical Inference Descriptive
Incremental					
P_{max}	1.8 ± 4.5	-1.4 ± 3.5	3.2 ± 3.0	0.32 ± 0.3	Very likely +ve
20kmTT					
P_{Ave}	3.1 ± 4.5	0.0 ± 2.9	3.1 ± 2.9	0.3 ± 0.27	Very likely +ve
$Speed_{Ave}$	1.3 ± 2.0	0.0 ± 1.1	1.3 ± 1.2	0.34 ± 0.32	Very likely +ve
Time	-1.5 ± 1.8	0.0 ± 1.0	-1.5 ± 1.1	-0.38 ± 0.28	Very likely +ve

*Units of change are percentage means ± SD for all measures.
†± 90% CL: add and subtract this number to the difference to obtain the 90% confidence limits for the true difference.
‡Based on a smallest beneficial or harmful change in performance of 1%.

4.3. Effect on Physiological Measures

A comparison of the physiological measures determined during the incremental test to exhaustion in the two groups revealed no clear differences (*Table 9*). Although the effect of BPR on lactate and heart rate at LT and LTP was unclear, there was a trend for both of these variables to be decreased in the intervention group at LTP (-4.6 ± 16.6%; -2.3 ± 2.5% respectively). All other measures were unclear.

Table 9. Mean changes in Physiological Measures post Breathing Pattern Retraining and Control and Chances That the True Difference in the Changes is Substantial

	Change In Measure*				
	Intervention (N=10)	Control (N=12)	Difference mean ± SD	Cohen's ES ± 90% CL†	Practical Inference Descriptive‡
Power at LT	-2.7 ± 6.0	-0.5 ± 13.4	-2.2 ± 7.3	-0.13 ± 0.41	unclear
Power at LTP	1.7 ± 6.6	-1.4 ± 10.0	3.1 ± 6.1	0.21 ± 0.40	unclear
HR_{max}	0.1 ± 2.2	-0.1 ± 2.0	0.0 ± 1.6	-0.01 ± 2.80	unclear
B[La] at LT	13.8 ± 21.7	10.8 ± 22.8	2.7 ± 16.0	0.08 ± 0.46	unclear
B[La] at LTP	-0.9 ± 22.0	3.9 ± 24.2	-4.6 ± 16.6	-0.17 ± 0.54	unclear
HR at LT	0.9 ± 3.7	2.5 ± 3.9	-1.5 ± 2.8	-0.19 ± 0.34	unclear
HR at LTP	-0.7 ± 3.0	1.7 ± 3.5	-2.3 ± 2.5	-0.3 ± 0.31	unclear
HR_{ave}	2.5 ± 2.3	0.2 ± 4.9	2.3 ± 2.9	0.32 ± 0.40	unclear
VO_2 at LT (L·min^{-1})	-0.5 ± 7.1	0.0 ± 6.7	-0.4 ± 5.1	-0.03 ± 0.35	unclear
VO_2 at LTP (L·min^{-1})	-0.7 ± 6.2	0.3 ± 7.6	1.0 ± 5.1	0.08 ± 0.38	unclear
VCO_2 at LT (L·min^{-1})	0.7 ± 6.9	-1.5 ± 7.2	2.3 ± 5.2	0.14 ± 0.31	unclear
VCO_2 at LTP (L·min^{-1})	0.7 ± 7.0	0.7 ± 6.4	1.4 ± 4.9	0.09 ± 0.31	unclear
VO_2 at LT (% VO_{2peak})	-1.0 ± 5.9	4.3 ± 11.1	-5.1 ± 6.3	0.58 ± 0.68	unclear
VO_2 at LTP (% VO_{2peak})	-0.2 ± 5.0	0.9 ± 6.9	-1.2 ± 4.3	-0.15 ± 0.57	unclear
VO_{2max} (L·min^{-1})	0.5 ± 6.9	-0.4 ± 6.4	1.0 ± 4.9	0.09 ± 0.45	unclear
VO_{2max} (ml·kg·min^{-1})	0.7 ± 6.6	-0.5 ± 6.2	1.2 ± 4.7	0.08 ± 0.31	unclear

*Units of change are percentage means ± SD for all measures.
†± 90% CL: add and subtract this number to the difference to obtain the 90% confidence limits for the true difference.
‡Based on a smallest beneficial or harmful change in performance of 1%.

4.4. Effect on Respiratory Measures

The effect of BPR on static and dynamic lung function tests was unclear for all measures, except for FVC where the effect was deemed unlikely to be beneficial. Whilst there was a trend of both IRV (18 ± 36.4%) and ERV (9.8 ± 44.3%) to increase, and for FEV_1 and MVV to decrease, the 90% CI associated with these measures was large.

The effect of BPR on t_I (10.1 ± 8%; 9.4 ± 7.7%) and V_T (11.6 ± 14%; 12.6 ± 11.2%) at both LT and LTP respectively was substantial. There was a strong trend for t_E to be increased at both LT and LTP (5.0 ± 9%; 5.9 ± 7.1%). There was a clear trend for V_E at both LT (-5.6 ± 4%) and LTP (-5.2 ± 4%) to be lower post-BPR. Similarly, breathing frequency (f_b) was also substantially lower both at LT (-13.2 ± 8.9%) and LTP (-9.5 ± 8.4%). The effect on respiratory drive (V_T/t_I) and respiratory timing (t_I/t_{TOT}) at LT and LTP was unclear. *Table 10* shows the mean changes in respiratory measures for the BPR relative to the control condition and the statistics for the differences in the changes.

Table 10. Mean Changes in Respiratory Measures (non-exercise and exercise) post Breathing Pattern Retraining and Control and Chances That the True Difference in the Changes Is Substantial

	Change in Measure*				
	Intervention (N=10)	Control (N=12)	Difference ± 90% CL	Cohen's ES ± 90% CL	Practical Inference Descriptive‡
Lung Function Tests					
IRV	6.3 ± 35.3	-10 ± 62.7	18 ± 36.4	0.33 ± 0.61	unclear
ERV	8.7 ± 79.7	-1.1 ± 37.7	9.8 ± 44.3	0.2 ± 0.78	unclear
FVC	-2.8 ± 5.9	2.7 ± 8.1	-5.4 ± 5.1	0.44 ± 0.39	probably -ve
FEV_1	-1.7 ± 8.1	0.9 ± 6.5	-2.7 ± 5.4	-0.19 ± 0.37	unclear
PEF	-0.6 ± 5.9	-2.5 ± 16.3	1.98 ± 8.7	0.09 ± 0.39	unclear
MVV	-4.9 ± 16.7	5.5 ± 30.6	-9.9 ± 17.1	-0.71 ± 1.07	unclear
Incremental Test					
t_E at LT (sec^{-1})	12.1 ± 13.6	6.8 ± 10.3	5.0 ± 9	0.25 ± 0.43	unclear
t_E at LTP (sec^{-1})	10 ± 10.7	3.9 ± 8.2	5.9 ± 7.1	0.29 ± 0.35	unclear
t_I at LT (sec^{-1})	10.8 ± 13.4	0.7 ± 6.3	10.1 ± 8	0.42 ± 0.34	Very likely +ve
t_I at LTP (sec^{-1})	11.8 ± 12.4	2.1 ± 7.2	9.4 ± 7.7	0.43 ± 0.35	Very likely +ve
V_T at LT (L)	11.6 ± 14	0.9 ± 7.2	10.6 ± 8.5	0.53 ± 0.42	Very likely +ve
V_T at LTP (L)	12.6 ± 11.2	2.9 ± 9.4	9.4 ± 7.6	0.47 ± 0.39	Very likely +ve
VE at LT (L·min^{-1})	-5-5 ± 5.7	-0.1 ± 5	-5.6 ± 4	-0.33 ± 0.22	Very likely +ve
VE at LTP (L·min^{-1})	-4.1 ± 4	1.2 ± 6.7	-5.2 ± 4	-0.32 ± 0.23	Very likely +ve
VE_{max} (L·min^{-1})	-2.2 ± 11	-2.2 ± 11.5	0.0 ± 8.2	0.0 ± 0.52	unclear
V_T/t_I at LT (L·sec^{-1})	0.7 ± 16.8	0.2 ± 3.5	0.5 ± 9.6	0.03 ± 0.46	unclear
V_T/t_I at LTP (L·sec^{-1})	-2.9 ± 9.8	0.8 ± 9.4	-3.6 ± 7	-0.2 ± 0.37	unclear
f_b at LT (1·min^{-1})	-14.5 ± 14.4	-1.6 ± 8	-13.2 ± 8.9	-0.71 ± 0.43	Very likely +ve
f_b at LTP (1·min^{-1})	-11.3 ± 13.5	-2.0 ± 7.9	-9.5 ± 8.4	-0.51 ± 0.41	Very likely +ve
t_I/t_{TOT} at LT	-1.2 ± 4.1	-2.1 ± 4.1	0.9 ± 3.8	0.14 ± 0.57	unclear
t_I/t_{TOT} at LTP	-0.5 ± 5	-0.8 ± 3.2	0.3 ± 3.2	0.05 ± 3.2	unclear

*Units of change are percentage means ± SD for all measures.
†± 90% CL: add and subtract this number to the difference to obtain the 90% confidence limits for the true difference.
‡Based on a smallest beneficial or harmful change in performance of 1%.

4.4.1. *Comparison of measures at absolute workloads pre and post-BPR*

Several measures were compared over a range of absolute workloads pre- and post-intervention (Figures 6 to 10).

At each workload, f_b decreased (Figure 6) and V_T increased following BPR (Figure 6). There was a trend for V_E to slightly decrease following BPR (Figure 6).
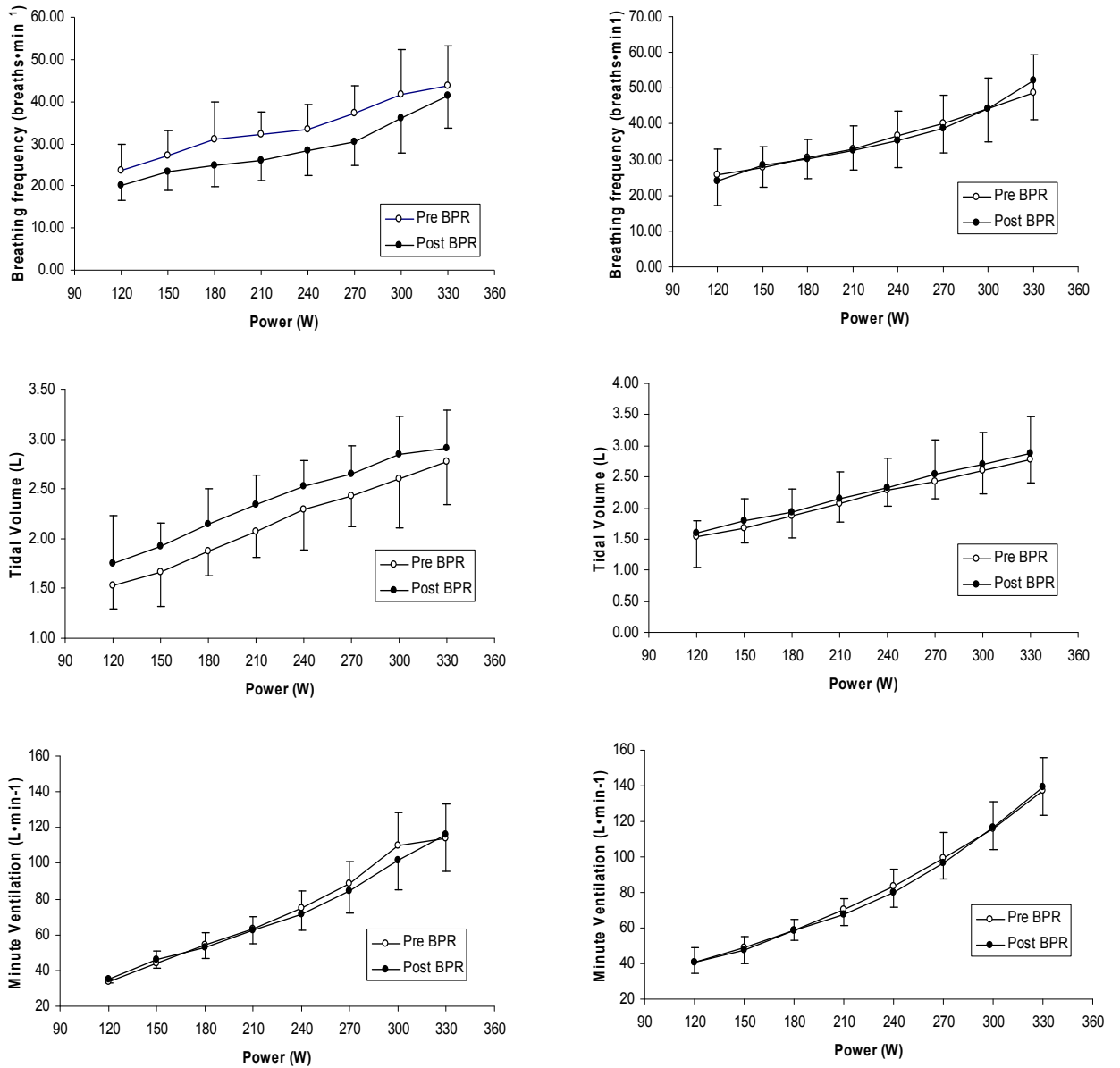
Figure 6. Effect of breathing pattern retraining on breathing frequency, tidal volume and minute ventilation in the intervention (left panel) and control groups (right panel).

As anticipated, the t_I and t_E decreased with the progressive increase in workload. After BPR, both t_I (13%) and t_E (12%) were longer (Figure 7).
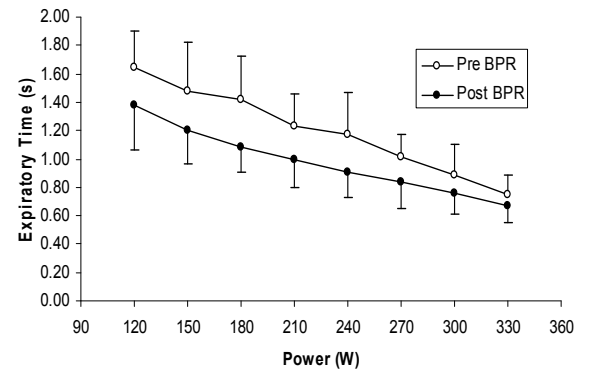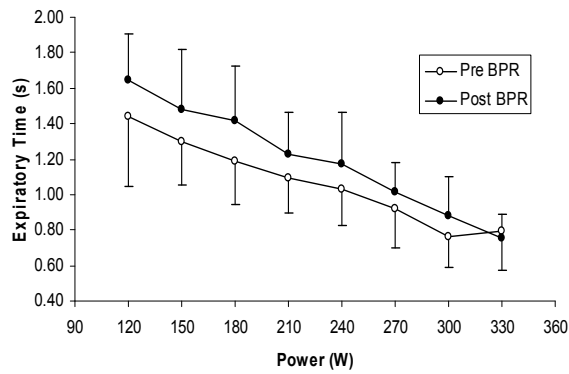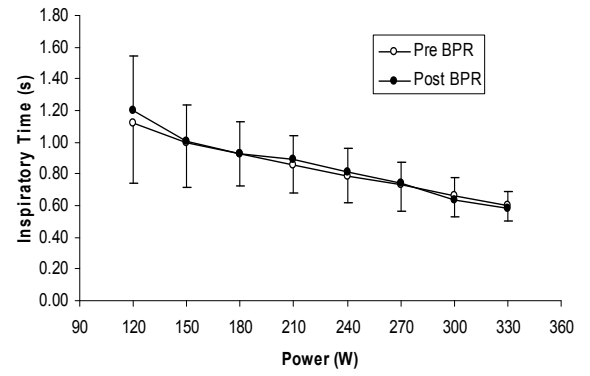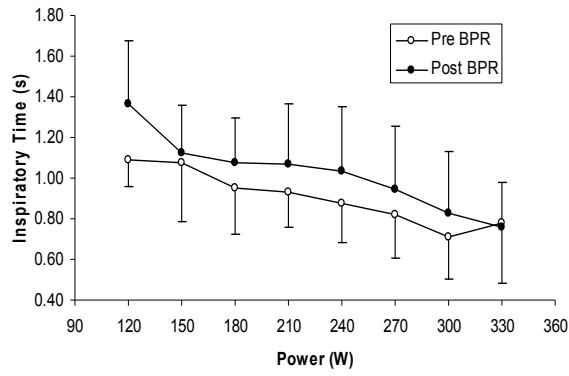
Figure 7. Effect of Breathing pattern retraining on inspiratory and expiratory time in the intervention (left panel) and control (right panel) groups.

Inspiratory drive (V_T/t_I) and inspiratory timing (t_I/t_{TOT}) did not change substantially throughout the incremental test as a result of BPR (Figure 8).
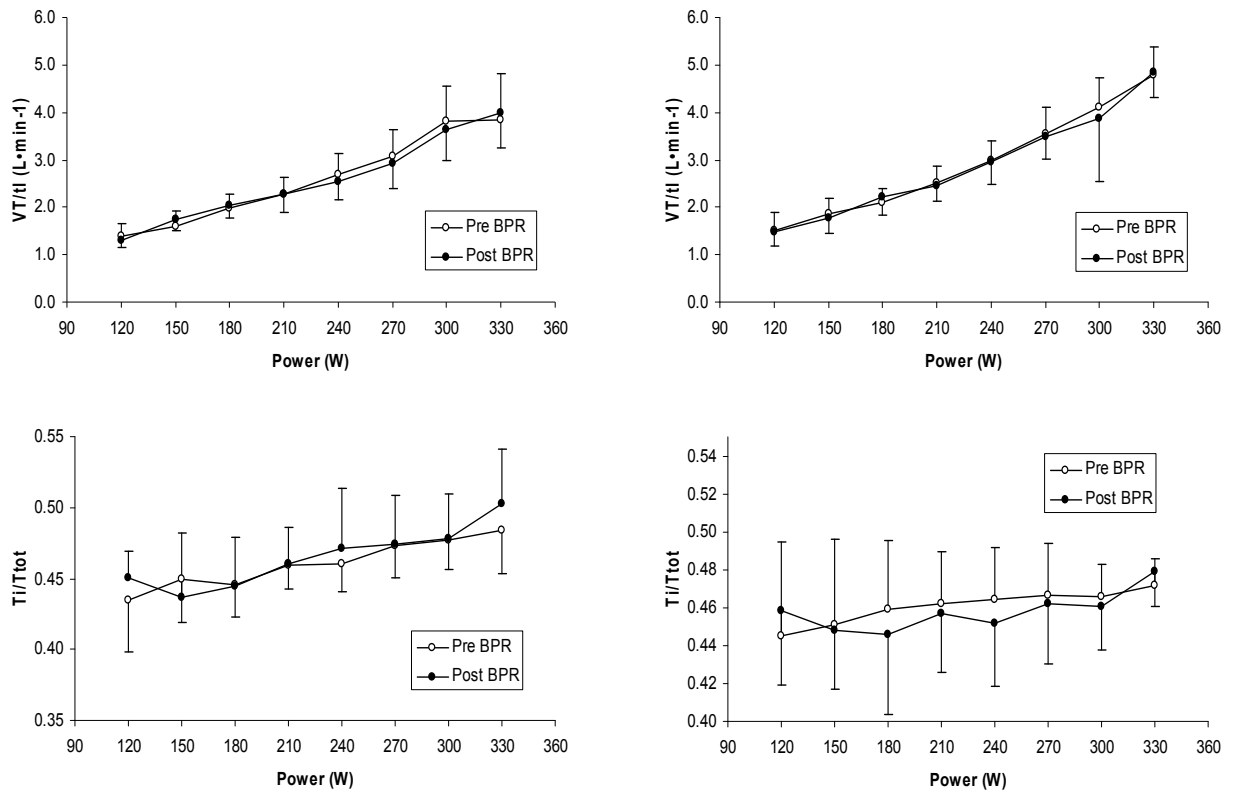
Figure 8. Effect of breathing pattern retraining on inspiratory drive (V_T/t_I) and inspiratory timing (t_I/t_{TOT}) in the intervention (left panel) and control (right panel) groups.

There was a slight trend for lactate levels to be decreased from 240 to 330W in the intervention group (Figure 9).
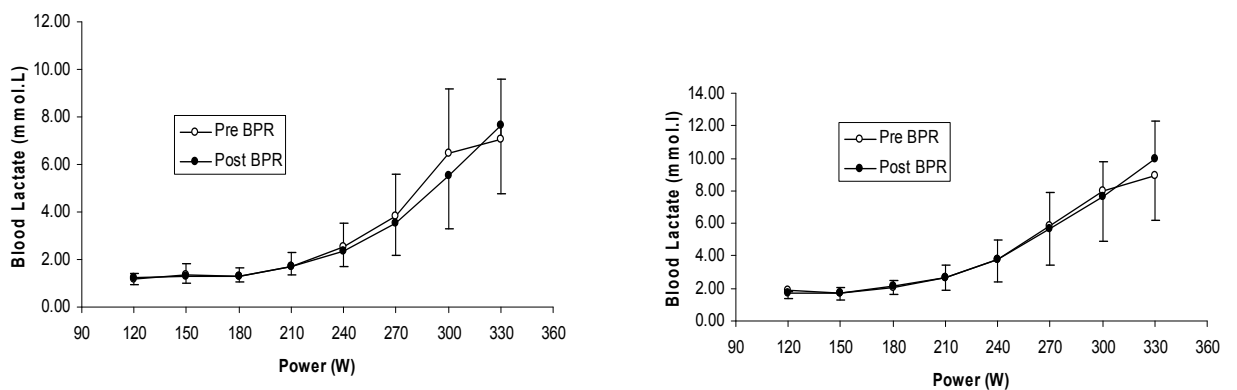
Figure 9. Effect of breathing pattern retraining on blood lactate in the intervention (left panel) and control (right panel) groups.

End-tidal partial pressures for oxygen ($P_{ET}O_2$) and carbon dioxide ($P_{ET}CO_2$) (data not presented), $\dot{V}O_2$ and $\dot{V}CO_2$ were not affected by BPR at any stage throughout the incremental test (Figure 10).
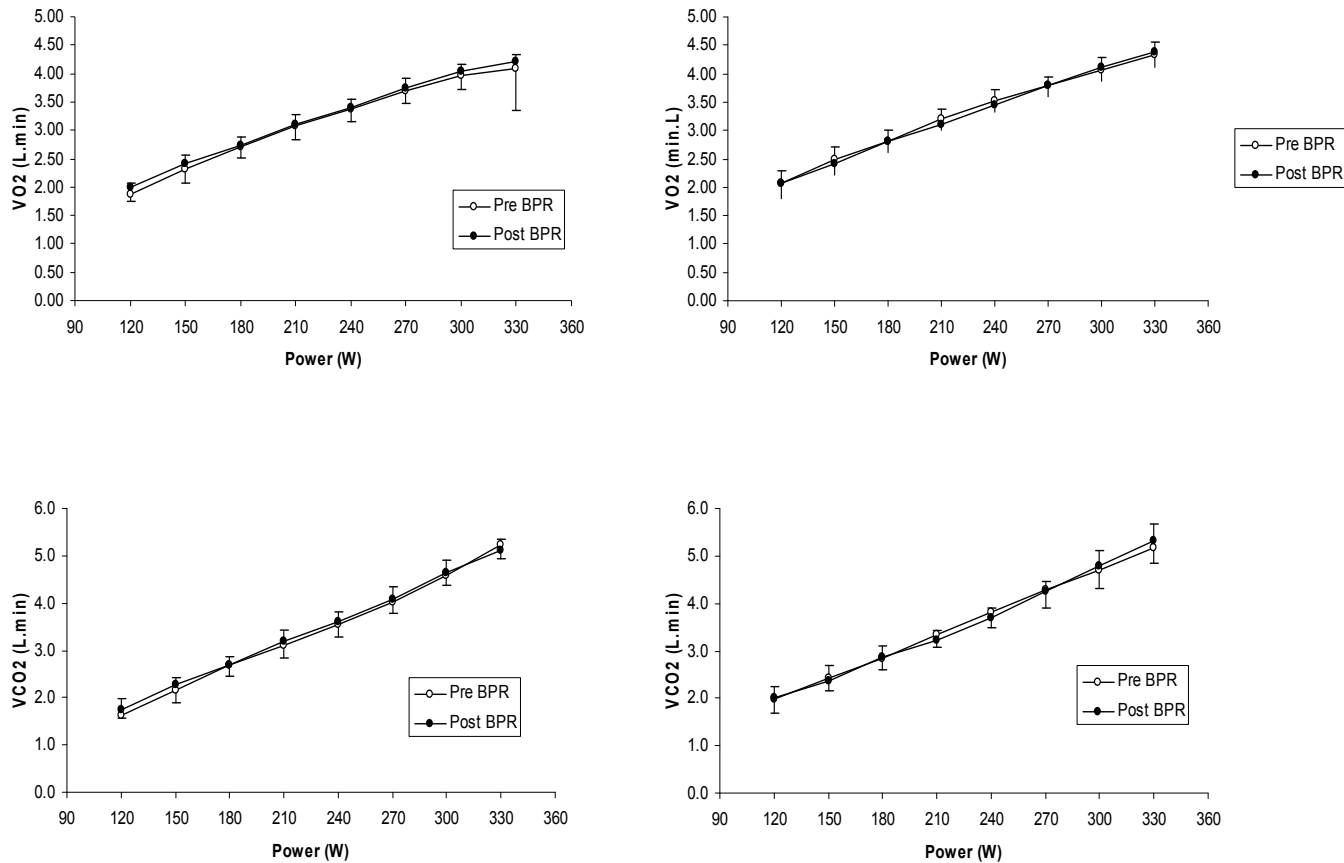
Figure 10. Effect of breathing pattern retraining on VO2 and VCO2 in the intervention (left panel) and control (right panel) groups.

4.5. Changes in RPE measures

4.5.1. RPE

Table 11 shows the effect of BPR on mean RPE (legs and the breathing) during both the incremental test and 20km-TT. The RPE at LT for both the legs (-27.9 ± 38.5%) and breathing (-30 ± 33.9%) were lower post-intervention. Similarly, leg and breathing RPE were also lowered at LTP (-24.7 ± 28.2%; -24.7 ± 28.1%). It was unclear whether BPR had a substantial effect on leg or breathing RPE at any point during the 20km-TT.

4.5.2. Reason for terminating exercise

Prior to BPR, 60% of the intervention group rated their lungs/breathing as the terminating factor in the incremental test, 30% rated their legs, and 10% both legs and lungs. In the control group, 42% rated their lungs, 42% rated their legs and 10% both legs and lungs as the terminating factor. Following BPR, only legs (90%) or both legs and lungs (10%) were rated as terminating factors in the intervention group whilst the control group continued to rate lungs (58%) or legs (42%) as the terminating factor.

Table 11. Mean Changes in Rate of Perceived Exertion Post Breathing Pattern Retraining and Control and Chances That the True Difference in the Change is Substantial

	Change in Measure*				
	Intervention (N=10)	Control (N=12)	Difference ± 90% CL	Cohen's ES ± 90% CL†	Practical Inference Descriptive‡
Incremental Test					
LT Leg$_{RPE}$	-18.2 ± 64	13.4 ± 39.6	-27.9 ± 38.5	-0.63 ± 0.62	Very likely +ve
LTP Leg$_{RPE}$	-9.8 ± 37.1	19.9 ± 43.2	-24.7 ± 28.2	-0.69 ± 0.60	Likely +ve
LT Breathing$_{RPE}$	-18.8 ± 47	16.1 ± 50.2	-30.0 ± 33.90	-0.77 ± 0.63	Likely +ve
LTP Breathing$_{RPE}$	-6.1 ± 37.1	24.8 ± 43	-24.7 ± 28.1	-0.69 ± 0.60	Likely +ve
20km TT					
15 min Leg$_{RPE}$	3.8 ± 22.2	1.4 ± 33.5	2.5 ± 19.9	0.08 ± 0.58	unclear
Final Leg$_{RPE}$	-0.1 ± 10.8	5.2 ± 31.2	-5.1 ± 16.1	-0.18 ± 0.52	unclear
15 min Breathing$_{RPE}$	-4.0 ± 28.4	6.7 ± 40.0	-10.0 ± 24.1	-0.39 ± 0.80	unclear
Final Breathing$_{RPE}$	-6.0 ± 14.4	1.6 ± 38.0	-7.4 ± 19.6	-0.34 ± 0.78	unclear

*Units of change are percentage means ± SD for all measures.
†± 90% CL: add and subtract this number to the difference to obtain the 90% confidence limits for the true difference.
‡Based on a smallest beneficial or harmful change in performance of 1%.

4.5.3. Correlations between RPE$_{breathing}$, RPE$_{legs}$, f_b and performance changes

There were no clear correlations between RPE$_{breathing}$, RPE$_{legs}$, or f_b and the performance changes observed in either the 20km-TT or the incremental step test.

Chapter Five: Discussion

The aim of this study was to investigate the effect of breathing pattern retraining (BPR) on cycling performance, and on respiratory, metabolic and perceptual responses during an incremental test in competitive cyclists. The main findings were that four weeks of specific BPR clearly enhanced endurance performance and incremental peak power and positively affected breathing pattern and perceived exertion.

To the best of the author's knowledge, this is the first study that has investigated the effect of BPR on endurance performance in athletes. Consequently, it is not possible to directly discuss the results from the present study in the context of those that have been previously reported. However, where appropriate, the findings of the present study have been compared to other cycling studies, or studies that have attempted to influence or modify ventilation in athletes using related methods such as IMT (Johnson, Sharpe, & Brown, 2007; Romer, McConnell, & Jones, 2002a) and VIH (Holm, Sattler, & Fregosi, 2004; Sonetti, Wetter, Pegelow, & Dempsey, 2001). Furthermore, in some instances reference has been made to studies that have acutely imposed a change to breathing pattern (i.e. entrainment), during a one off test (Fabre, Perrey, Arbez, & Rouillon, 2007; Maclennan, Silvestri, Ward, & Mahler, 1994) as opposed to specifically retraining breathing pattern.

The large standard deviations reported in tables 8, 9, 10 and 11 demonstrate a degree of individual response (intervention and control groups) to the intervention period. Whilst these responses are noted, the following discussion will focus on group changes and trends.

5.1. Effect of BPR on cycling performance

In the present study, cycling performance was measured in two-ways: i) a simulated (laboratory-based) 20km-TT and ii) an incremental step test to exhaustion. Both tests were performed on the same electromagnetically-braked cycle ergometer.

5.1.1. Effects on 20km-TT performance

The four week BPR intervention had a clear beneficial effect both on average 20km-TT power (3.1 ± 2.9%), average speed (1.3 ± 1.2%) and 20km-TT performance time (-1.5 ± 1.1%). These results meet the criteria for worthwhile effect size (0.5-1.5%) estimated by Paton and Hopkins (2001) in mean power, based on variability in competitive performance of elite cyclists. In terms of changes in performance time, the observed performance improvement is somewhat less than the cycling performance changes reported by Romer et al. (2002) (-3.8 ± 1.7%; 20km-TT) and Johnson et al. (2007) (-2.66 ± 2.5%; 25km-TT) after six weeks of IMT, or the improvement in approximate 40km-TT (-4.7 ± 1.6%) after four weeks of VIH (Holm, Sattler, & Fregosi, 2004). Conversely it is consistent with the findings of Sonnetti et al. (2001) who reported a 1.5% improvement in 8km-TT after four weeks of combined VIH and IPTL (although this latter study did not show a significant difference between the control and intervention groups). Similarly, but in rowing, the magnitude of performance change has been greater for IMT than the present study (-3.1 ± 0.8%, 5000m-TT after 11 weeks of IMT; Volianitis et al., 2001).

Whilst the substantial enhancement in cycling performance after BPR is encouraging, the smaller change compared to those reported should be considered. Differences could be explained by several methodological reasons including: i) the differences in intervention period and number of intervention sessions; ii) the time of competitive season that the intervention took place; and iii) physiological reasons including initial (pre-intervention) aerobic fitness. In terms of duration, the

present study involved a four week (28 sessions) intervention period. In contrast, the cycling studies that reported larger performance changes have typically used six week interventions both involving 84 sessions (Johnson, Sharpe, & Brown, 2007; Romer, McConnell, & Jones, 2002a). Holm et al. (1994) used a four week intervention which involved 40 sessions and recorded a larger change than the present study. The cycling study that reported a similar performance change (Sonetti, Wetter, Pegelow, & Dempsey, 2001) also used a four week intervention period with 20 sessions. The greater number of sessions could benefit athletes in two ways; first by enhancing the benefits seen during exercise (acute response to intervention), and second, by allowing athletes to train at a higher intensity, further enhancing performance (long term benefit). In support of the latter, Dickinson, Whyte and McConnell (2007) recently commented that improvements to breathing after IMT may enable athletes to train at higher intensities, with subsequent performance gains.

Adopting a new automatic breathing pattern is not achieved immediately upon commencement of BPR (Grossman, DeSwart, & Defares, 1985). Indeed, a period during which respiratory exertion is perceived as being greater is often experienced during the adaptation phase (Ley & Yelich, 1998). Interestingly, participants in the BPR group of the present study reported it took approximately three weeks (21 sessions) until they felt they were using their new breathing pattern comfortably and automatically during training. It is possible therefore that the performance improvements seen in the present study solely reflected a response to the participants' breathing pattern during exercise, but was probably not long enough for participants to benefit in terms of increasing their training effort. Thus additional weeks and/or sessions of BPR may have allowed participants to train at a higher intensity with their newly adopted breathing pattern, potentially resulting in physiological adaptation and further performance enhancement.

The time of the competitive season that the studies were performed may also have contributed to the variation observed in performance. As highlighted by Paton and Hopkins (2004), most intervention research involving competitive athletes is conducted in the non-competitive part of the season and that smaller performance gains are to be made by cyclists during their competition season due to the high level of intensity that they are training at. During this time of the season, the magnitude of change of some interventions may be inflated since athletes are further away from their peak condition. The present study was purposely undertaken during the competition season so that the true benefit of BPR could be determined. In comparison, Romer et al. (2002) conducted their study during the maintenance phase. In other studies, it is not stated what part of the season studies were performed (Holm, Sattler, & Fregosi, 2004; Johnson, Sharpe, & Brown, 2007).

It is unlikely that the difference (approximately 1-2%) in TT performance between the present study and those previously reported are due to the experience or ability of the participants. It would be reasonable to expect that higher level athletes may make smaller performance gains due to the smaller margins for improvement they possess (Hamlin & Hellemans, 2006). This is unlikely to be the case in this instance as the athletes recruited in the present study were of similar fitness level ($\dot{V}O_{2\ peak}$ 4.34 ± 0.47L·min^{-1}) to those recruited by Romer et al. (4.58 ± 0.17 L•min^{-1}, 2002a) and should theoretically have had the same potential to improve their aerobic performance. Further, despite recruiting cyclists with fitness levels lower than the present study Johnson et al. (3.65 ± 0.86 L•min^{-1}, 2007) and Holm et al. (3.78 L·min$^{-1,}$ 2004) demonstrated larger performance gains than the present study, again suggesting that pre-intervention fitness level was not a contributing factor to the variation in performance change observed between the present study and those reported above.

Finally, it is probable that the difference between the results of the present study and those previously reported may be due to different aspects of respiratory muscle function that IMT and BPR attempt to have an effect on. For example, IMT primarily aims to increase both the strength and endurance of the inspiratory muscles in an attempt to reduce inspiratory muscle fatigue (as measured by changes to MIP) (Romer, McConnell, & Jones, 2002b; Volianitis et al., 2001). Conversely, BPR does not specifically train the strength and endurance of the inspiratory muscles but instead aims to optimise the mechanics of breathing thus decreasing the work of breathing during exercise. Therefore BPR may affect TT performance by primarily altering the recruitment pattern of the accessory muscles and enhancing the mechanical advantage of the respiratory muscles (the benefits of which will be discussed later), with changes in B[La] being secondary. It is possible that if IMT was combined with BPR such that an optimal breathing pattern (automatic) was established *prior* to commencing and throughout the duration of IMT, there could be an accumulative effect on endurance performance.

5.1.2. Effects on Incremental Peak Power (P_{max})

In addition to enhanced 20km-TT performance, a substantial improvement in P_{max} (3.2 ± 3%, Table 8) was also observed following BPR. In contrast, neither IMT nor VIH have been shown to induce an ergogenic effect on incremental power in rowing (Volianitis et al., 2001) or cycling (Holm, Sattler, & Fregosi, 2004; McConnell & Sharpe, 2005; McMahon, Boutellier, Smith, & Spengler, 2002; Romer, McConnell, & Jones, 2002a) and there are conflicting results that both refute (Romer, McConnell, & Jones, 2002b) and support (Chatham, Baldwin, Griffiths, Summers, & Enright, 1999) its effect on incremental running. For example, in a well controlled, double blind study Romer et al. (2002b) were unable to demonstrate changes to incremental shuttle running performance after four weeks of IMT. Conversely, Chatham et al. (1999) demonstrated a 3.78%

improvement in shuttle running. However this latter study has been criticised because the IMT group was led to believe that IMT would enhance their performance, which inevitably may have motivated them to perform better, compared to the control group who had no expectation.

It is possible that, as with Chatham et al. (1999), participants in the BPR group of the present study had a higher expectation of improved performance and therefore their motivation to do better was elevated. To address this potential confounding variable from the outset, participants in the present study were at no stage led to believe that the aim of BPR was to improve performance. Rather, they were informed that the purpose was solely to investigate whether breathing pattern could indeed be altered during exercise. The combination of increased performance, yet decreased perceived exertion (see *Effects of BPR on Perceived Effort*) supports the assertion that increased motivation was not the primary influence on performance in the present study.

5.2. Effects of BPR on Respiratory Measures

An important finding in the present study was that BPR, as anticipated, resulted in a substantial change in breathing pattern during exercise. Several respiratory measures reflecting breathing pattern were determined during the incremental step test to exhaustion and for direct comparison were compared at similar absolute workloads pre- and post-BPR. These included V_E, f_b, V_T, t_I, t_E, inspiratory drive (V_T / t_I) and inspiratory timing (t_I/t_{TOT}). Dynamic measures of lung function were measured at rest.

As anticipated, no changes were observed in most dynamic lung function measures post-BPR (benefit unlikely, Table 10) – a finding that coheres well with IMT studies (Romer, McConnell, &

Jones, 2002a) (McMahon, Boutellier, Smith, & Spengler, 2002). However, respiratory measures during exercise were altered. BPR resulted in a substantial decrease in f_b and V_E, combined with an increase in V_T and t_I and it is possible that these changes explain the observed performance improvements.

5.2.1. Breathing frequency (f_b) and Tidal Volume (V_T)

Most healthy humans increase ventilation at high exercise intensities by initially increasing V_T, then f_b. As exercise intensity increases, the continued increase in f_b compromises V_T such that V_T plateaus or even slightly decreases, resulting in a tachypnoeic shift (Eastwood, Hillman, & Finucane, 2001; Gallagher, Brown, & Younes, 1987; Scheuermann & Kowalchuk, 1999). This ventilatory response has been demonstrated in untrained subjects with a wide range of fitness levels (Folinsbee, Wallace, Bedi, & Horvath, 1983; Gallagher, Brown, & Younes, 1987) as well as elite (but not professional) cyclists (Lucia, Carvajal, Calderon, Alfonso, & Chicharro, 1999). In the present study the BPR and control groups demonstrated similar breathing patterns pre-intervention, with similar f_b (51 vs. 56 breaths $\cdot$min^{-1} respectively) and V_T (2.7 vs. 2.7 L $\cdot$min^{-1}) being achieved at termination of exercise (330 vs. 340W) however substantial differences in breathing pattern were observed post-BPR. Whilst f_b increased and V_T decreased across workloads in the incremental test, the BPR group showed a significant decrease in f_b and an increase in V_T compared with the breathing pattern they adopted pre-intervention (Figure 6). Conversely, the breathing pattern of the control group did not change except for a decreased t_E post-intervention (which is not thought to be a desired change). The biggest difference between the two groups was the change in f_b, with the intervention group reaching a maximal f_b of 47 ± 7 breaths $\cdot$min^{-1} at exhaustion (335 ± 35W) whilst the control group demonstrated a maximal f_b of 60 ± 8 breaths $\cdot$min^{-1} (333 ± 30W). These f_b values are comparable to those observed by Lucia et al. (1999), who reported a f_b of approximately 51 and 58 breaths $\cdot$min^{-1} in professional and elite

cyclists respectively, though it should be acknowledged that a greater power output was reached (>400W) by Lucia et al.'s (1999) subjects (Figure 1). It is likely that the emphasis during BPR of maintaining a diaphragmatic breathing pattern, with forced exhalation, enabled the BPR group to keep their f_b substantially lower as exercise intensity increased.

5.2.2. Inspiratory and Expiratory Time

In an extensive investigation of the breathing pattern measures in elite and professional cyclists, Lucia et al. (1999) identified that t_I and t_E were the two parameters that differentiated elite and professional cyclists and that such difference may possibly contribute to the superior level of performance attained by the professionals. Inspiratory time reached a plateau in professionals at low intensities (300W) and then decreased with increasing exercise intensity, whereas it continuously decreased in elites from the lowest to the highest intensities (Figure 2). Expiratory time was significantly higher in professionals than elites at most moderate-to-high power outputs (Figure 2). Prior to the intervention of the present study, both BPR and control groups displayed a decrease in t_I from low to high intensities (Figure 7) consistent with the elite cyclists of Lucia et al. (1999). After intervention the control group, as expected, retained the same breathing pattern, however the BPR group's breathing pattern changed. Specifically, the t_I initially increased but then plateaued between 150 and 240W (early to mid workloads), before gradually decreasing (Figure 7) in a pattern similar to professional cyclists. It is likely that the BPR allowed subjects to maintain control of the length of t_I and to ultimately control their breathing pattern during incremental exercise.

Although the difference in t_E pre- versus post-intervention was unclear, there was a trend for t_E to be lengthened in the intervention group post-BPR at both LT and LTP (5.0 ± 9; 5.9 ± 7.1% respectively; Table 10). An increased t_E may also have been an important factor in preventing a

tachypnoeic breathing pattern developing, as evident by the increased V_T and decreased f_b discussed above. Lucia et al. (1999) suggested that the ability to maintain a breathing pattern during maximal intensity exercise void of a tachypnoeic shift is an important differential between professional and amateur elite cyclists, with the prolongation of t_E being an important adaptation made by professional cyclists during high exercise intensities. Most healthy humans increase f_b by decreasing t_E rather than t_I (Folinsbee, Wallace, Bedi, & Horvath, 1983) – a breathing pattern that reverses the normal inspiratory to expiratory ratio, and may lead to breath-stacking and dynamic hyperinflation (Chaitow & Bradley, 2002), the consequence of which may ultimately decrease exercise performance. Prolonging t_E in the BPR group may have aided in preventing a tachypnoeic breathing pattern from developing and subsequently contributed to enhanced exercise performance.

The change in t_I and t_E observed in the present study is exciting as it suggests that it is possible to change an athletes breathing pattern, and for them to maintain this during high intensity exercise, in a way that appears to be beneficial to endurance performance. Previously it had been suggested that this difference was an acquired physiological response that perhaps developed over years of training (Lucia, Carvajal, Calderon, Alfonso, & Chicharro, 1999), rather than something that could be learned in a relatively short period of time. It is believed that no other study has reported changes in t_I or t_E after a respiratory related intervention in athletes.

5.2.3. Minute Ventilation

The largest physiological change observed in the present study after BPR was the decrease in V_E at both LT and LTP (Table 10). Although V_{Emax} was unchanged in the present study (Benefit likely, Table 10), there was a trend for V_E to be decreased at workloads above LTP (240 – 300W; Figure 6). This finding is consistent with that of Lucia et al. (1999) who showed that V_{Emax} did not

differ between elite and professional cyclists (Figure 1), although it remained lower in professional than elite cyclists at higher workloads (300-400W). The change in f_b and V_T discussed above, coupled with an unchanged V_{Emax} suggest that cyclists in the present study were able to achieve the same V_E in a more 'controlled' way, which prevented the occurrence of a tachypnoeic shift. Lack of tachypnoeic shift is one of the major differences in breathing pattern highlighted by Lucia et al. (1999) and is thought to be beneficial to exercise performance by decreasing the work of breathing (Lucia, Carvajal, Calderon, Alfonso, & Chicharro, 1999), reducing respiratory muscle fatigue (Johnson, Aaron, Babcock, & Dempsey, 1996) and at high workloads reducing blood stealing (Sheel et al., 2001).

Considering V_E responds more closely to demands for CO_2 clearance than for O_2 uptake (Ward, 2007) it would be reasonable to expect that a change in B[La] could serve to decrease V_E. However, whilst significant changes were observed in V_E at LT and LTP, no clear change in B[La] at these points was observed, although there was a trend for both V_E and B[La] to be decreased in the higher workloads (240-300W; Figures 6 and 9). This would suggest that the changes in V_E were not solely in response to metabolic changes but that they were likely due to the change in f_b and V_T as a result of BPR.

The respiratory compensation point (RCP) marks the onset of hyperventilation during incremental exercise and is thought to be mediated by metabolic acidosis (Meyer, Faude, Urhausen, & Kindermann, 2004). It could be speculated that one of the mechanisms underlying the ergogenic effect of BPR is the ability to control ones breathing pattern at the RCP such that the work of breathing is decreased, rather than due to any real metabolic changes. This suggestion is supported by the results of the present study demonstrating a decrease in f_b and V_E, and increase in V_T, post-BPR at LTP (275 ± 26W; Figure 6). In contrast, other breathing interventions such as

IMT do not appear to consistently influence V_E, (Griffiths & McConnell, 2007; Romer, McConnell, & Jones, 2002a; Spengler, Roos, Laube, & Boutellier, 1999; Steussi, Spengler, Knopfli-Lenzin, Markov, & Boutellier, 2001; Volianitis et al., 2001; Williams, Wongsathikun, Boon, & Acevedo, 2002) despite an abatement in B[La] being a consistent finding (Griffiths & McConnell, 2007; McConnell & Sharpe, 2005; Romer, McConnell, & Jones, 2002b; Volianitis et al., 2001). This is surprising as theoretically decreased B[La] would result in decreased metabolites and as such a decreased demand to clear CO_2 (Ward, 2007).

Maintaining a low V_E may be beneficial for at least two reasons, the first by potentially preventing expiratory flow limitation. Expiratory flow limitation occurs when the breathing reserve (MVV - V_E) becomes small, or even negative, such that despite further increases in ventilatory drive in response to increasing exercise, ventilation can not increase further unless lung operating volumes increase. The ensuing CO_2 retention and arterial hypoxaemia predispose to decreased exercise tolerance (Ward, 2007). Although increasing lung operating volume in the presence of expiratory flow limitation can enable the necessary increased ventilatory demands to be met, the resulting increase in end-expiratory lung volume may result in dynamic hyperinflation which initiates a cascade of events (i.e. respiratory muscle fatigue, increased work of breathing, blood stealing; see section 2.3.6, pp33) that ultimately reduces exercise tolerance. In addition, decreased V_E may directly decrease the metabolic and therefore perfusion cost of the respiratory muscles which may also positively benefit exercise performance through attenuating the peripheral vasoconstriction metaboreflex.

The clear decrease in V_E that occurred during incremental exercise may reflect changes to the dead space-V_T ratio (see section 2.2.3, pp13). When a tachypnoeic shift occurs, the dead space-V_T ratio rises, and alveolar ventilation may be reduced, resulting in increased PCO_2 (Hanson,

Claremont, Dempsey, & Reddan, 1982). To preserve alveolar ventilation V_E may be increased by 10-30% (Hanson, Claremont, Dempsey, & Reddan, 1982). It has been demonstrated however, that such an increase in V_E is not required when V_T is maintained during high exercise intensities (Hanson, Claremont, Dempsey, & Reddan, 1982). It is possible that the intervention group did not need to increase their V_E to the extent they did pre-BPR as they were able to maintain a more effective V_T post-BPR.

The changes after BPR described above may have contributed to the improved 20km-TT performance and incremental power observed in the present study by attenuating the metaboreflex associated with increased work of breathing (Sheel et al., 2001), although no correlation was identified between the breathing pattern changes and exercise performance. Harms et al. (1997) demonstrated that increasing the work of breathing decreased blood flow to the legs in cyclists. Such decreased leg blood flow has been shown to decrease exercise tolerance and power output in cyclists (Harms, Wetter, St Croix, Pegelow, & Dempsey, 2000). A tachypnoeic breathing pattern is associated with increased dynamic hyperinflation which alters the mechanical contractile properties of the inspiratory muscles, increases the work of breathing and causes greater respiratory muscle fatigue (Scheuermann & Kowalchuk, 1999). Therefore, it is possible that BPR in the present study prevented the onset of a tachypnoeic breathing pattern and dynamic hyperinflation (in comparison to pre-intervention breathing pattern) thus containing the work of breathing and preserving leg muscle blood flow ultimately allowing subjects to increase both their 20km-TT and incremental step test performance. Direct measures of respiratory work and blood flow distribution during intense pre- and post-BPR would support (or refute) this suggestion.

It is thought that the sympathetically mediated reflex vasoconstriction in the limbs may be induced by reflex activity from chemo-sensitive type III / IV receptors of fatigueing respiratory muscle (Sheel et al., 2001). It is speculated that respiratory muscle fatigue may be caused by the respiratory muscles working in a mechanically disadvantaged position (i.e. under conditions of high elastic loads and velocity of muscle shortening and working at large percentages of their available capacity for muscle pressure generation (Johnson, Babcock, Suman, & Dempsey, 1993)), as occurs with dynamic hyperinflation, and that preventing or delaying dynamic hyperinflation may decrease respiratory muscle fatigue. In cyclists this would be an important change given the possibility of increased inspiratory muscle fatigue caused by the crouched cycling position (Boussana et al., 2007; Johnson, Babcock, Suman, & Dempsey, 1993). Unfortunately, respiratory muscle fatigue was not measured in the present study and therefore the ability of BPR to attenuate respiratory muscle fatigue is not known.

The exciting importance of the breathing pattern changes observed in the present study is that following BPR no tachypnoeic shift was observed, which reflects a breathing pattern usually observed only in very high level athletes (professional cyclists), and supposedly only achievable after years of training (Eastwood, Hillman, & Finucane, 2001; Lucia, Carvajal, Calderon, Alfonso, & Chicharro, 1999; Shephard, 1998; Steinacker, Both, & Whipp, 1993). Lucia et al. (Lucia, Hoyos, Pardo, & Chicharro, 2001) suggested that level of endurance training was not the causative factor for the difference in breathing pattern they observed in their 1999 study, unless it was a change that occurred over a number of years. Syabbalo et al. (1994) suggested that a tachypnoeic breathing pattern was caused by respiratory muscle fatigue and that high level athletes simply developed a superior level of resistance to such fatigue due to their greater years of training and therefore did not demonstrate such a pattern. This suggestion is unsupported by Romer et al. (2002c) or Griffiths and McConnell (2007) who did not identify changes to breathing pattern

despite identifying changes in maximal inspiratory effort of 28 ± 7% and 26% respectively – a measure used by these authors to identify respiratory muscle fatigue. In contrast, Hey et al. (1966) theorised that the integration of the depth and frequency of breathing required to achieve a given ventilatory demand is independent of known respiratory stimuli, and may simply reflect a pattern of breathing that results in a minimal amount of work by the respiratory muscles. The findings of the present study may give strength to this theory, and it is possible that the participants who underwent BPR were able to learn, or relearn, a breathing pattern that decreased the work of the respiratory muscles and which led to a positive effect on their exercise performance.

It is not known how breathing pattern is reprogrammed, and prior to this study whether in fact it is possible to reprogramme breathing pattern in trained athletes. However the results of this study may indicate that a change at some level of automatic ventilatory control did occur. Grossman et al. (1985) suggested that ones automatic breathing pattern can only be overridden for approximately ten minutes before one reverts back to a pattern that is comfortable and natural, yet the average incremental test in the present study lasted 21 minutes. The changes in breathing pattern discussed above were evident throughout the entire duration of each participant's test. This could suggest that the brainstem respiratory centres, or whatever factor(s) control the maintenance of automatic breathing pattern (currently unknown), were in some way altered. It could be argued that the participants in the BPR group simply altered their breathing pattern in response to verbal cues provided by the primary investigator. To address this issue, an assistant, blinded to which research group the participants belonged, was present for all post testing and provided the verbal encouragement. The assistant had no way of knowing which participants would benefit from verbal cueing. Further, studies that have altered breathing pattern, without giving an intervention period during which an athlete could acclimatise to, or automatically

adopt the new pattern, have failed to demonstrate changes to exercise performance (Fabre, Perrey, Arbez, & Rouillon, 2007; Maclennan, Silvestri, Ward, & Mahler, 1994) suggesting that a period of time is necessary (but currently unknown) for a new breathing pattern to become automatic. Based on the findings of this study it is suggested that if BPR is to be used in combination with IMT to further enhance the benefits of IMT, that a period of time is allowed for BPR and the (assumed) changes to automatic breathing pattern to occur *prior* to the introduction of IMT.

5.2.4. *Inspiratory Drive and Timing*

Despite observing changes in t_I and t_E, Lucia et al. (1999) found no difference in inspiratory drive or timing between the two groups. Similarly, no change was observed in the present study post-intervention (Figure 8) for either variable despite the changes to f_b and V_T. It should be borne in mind however that changes in the mechanical properties of the respiratory system can alter V_T/t_I for a given neural output of the respiratory centres (Milic-Emili & Grunstein, 1976). The changes in the present study to both components of inspiratory drive (i.e. V_T and t_I), combined with the possibility that BPR altered the mechanical properties of the respiratory system by decreasing hyperinflation may mask any changes to V_T/t_I that may have occurred. It is suggested that mouth occlusion pressure, diaphragm electromyography or phrenic nerve neurography are better indicators of inspiratory drive than V_T/t_I (Lucia, Carvajal, Calderon, Alfonso, & Chicharro, 1999). Mean values for inspiratory timing were consistently smaller than 0.5 in both groups pre and post-BPR (Figure 8) and similar to those observed by Lucia et al. (1999).

5.3. Effects of BPR on Perceived Effort

The results from the present study show that four weeks of BPR is effective in decreasing the perception of breathlessness and leg effort during maximal intensity cycling.

5.3.1. Perceived Breathing Effort

Following BPR $RPE_{breathing}$ during the incremental test was decreased (benefit likely, Table 11) at both LT (-30 ± 33.9%) and LTP (-24.7 ± 28.1%;) when compared with the control group. The changes to $RPE_{breathing}$ in the present study are larger than those reported previously. For example, $RPE_{breathing}$ was decreased following IMT by -16 ± 4% in cycling (Romer, McConnell, & Jones, 2002a), -7.9 ± 6% in repeated sprint activity (Romer, McConnell, & Jones, 2002b) and approximately -22%, (precise data not provided) during an incremental rowing test, however no changes were observed at the end of this test, or during an all-out six minute test (Volianitis et al., 2001). The effects of IMT on perceived respiratory effort has been strongly correlated (r = 0.64; P < 0.05) with improved performance (Romer, McConnell, & Jones, 2002b) however no correlation was identified in the present study between these two variables. Unlike the incremental test, $RPE_{breathing}$ was not substantially different during the time trial at either 15 minutes (isotime) or at completion of the time trial. This finding is not totally surprising as the incremental test is ridden at prescribed workloads whereas the TT has no set workload, enabling participants to ride at their highest tolerated intensity. It is more likely that changes in perceived exertion could be identified during the incremental test where effort during equal absolute workloads is compared, than in the TT where workloads are variable. It is interesting to note that $RPE_{breathing}$ for the control group was increased at LT and LTP (Table 11). This is likely due to a slight detraining effect as shown by a 1.5% decrease in power at LTP and incremental P_{max} (Table 8).

Perceived respiratory exertion, or dyspnoea, is one of the primary reasons given for the termination of exercise in healthy humans (Hamilton, Killian, Summers, & Jones, 1996; Killian et al., 1992). Theoretically if dyspnoea could be decreased, exercise tolerance may be increased. The degree to which $RPE_{breathing}$ was decreased at LT and LTP in the present study may have been a contributing factor in increased exercise performance. Just as factors that *impair* the contractile properties of the respiratory muscles are thought to increase dyspnoea (for example respiratory muscle fatigue, dynamic hyperinflation, increased accessory muscle recruitment) factors that *improve* the contractile properties have the potential to decrease the intensity of dyspnoea. BPR aims to decrease dynamic hyperinflation by maintaining an appropriate inspiratory:expiratory ratio, and delay recruitment of the accessory muscles by encouraging a diaphragmatic breathing pattern. It is speculated that BPR was effective in optimising respiratory muscle function to the degree that $RPE_{breathing}$ was significantly decreased, allowing participants to exercise longer with less discomfort. Because pacing strategies are thought to be affected by afferent (incoming central) information (Tucker et al., 2006) it is possible that the BPR group were able to ride at a higher intensity due to changes in afferent information that was reflecting the decreased work of breathing i.e. they rode harder because it felt easier. It is interesting to note that post-BPR only one participant in the intervention group terminated the incremental test due to his breathing (in combination with his legs) compared with seven pre-BPR, whilst seven in the control group terminated exercise due to their breathing and this was unchanged pre and post-intervention. This suggests that BPR was effective in decreasing dyspnoea to the point it was no longer perceived as the limiting factor during exercise. This could potentially mean cyclists could train at higher intensities without being restricted by their breathing.

Dyspnoea is a subjective experience that is partially contributed to by psychological and / or emotional influences (Grazzini, Stendardi, Gigliotti, & Scano, 2005). During the initial stages of

BPR participants were exposed to feelings of breathing discomfort as they attempted to alter (reprogramme) their V_T and f_b at rest and during exercise. In the early stages of retraining participants subjectively reported feelings of air-hunger and inability to inhale deeply enough. During this time they were educated and re-assured that these feelings were normal and just a perceived experience, not an indication of their body's requirement for oxygen. It is possible that through increased exposure to unpleasant respiratory sensations in a controlled environment the BPR group became more familiar and comfortable with the feeling of dyspnoea, knew they were in control of their breathing and no longer perceived respiratory exertion as such an intense experience.

Changes to the automatic breathing pattern do not occur immediately once BPR is undertaken (Grossman, DeSwart, & Defares, 1985). Indeed it is apparent from studies that have attempted to acutely manipulate breathing pattern that such short term changes do not affect perceived respiratory exertion. For example, paced breathing during roller-ski skating did not change $RPE_{breathing}$ (Fabre, Perrey, Arbez, & Rouillon, 2007), nor did entraining breathing on command during a rowing trial in rowers who were unfamiliar with entrainment (Maclennan, Silvestri, Ward, & Mahler, 1994), which is an interesting observation considering entrainment of breathing during upper limb exercise is thought to be a more efficient pattern (Maclennan, Silvestri, Ward, & Mahler, 1994; Steinacker, Both, & Whipp, 1993). As anticipated, the BPR group in the present study reported an *increase* in $RPE_{breathing}$ during the first 2-3 weeks of BPR, both in training and when doing their BPR exercises. This is possibly due to increased awareness of their breathing while they were consciously trying to change it (Ley & Yelich, 1998). The findings of Fabre et al. (2007) and Maclennan et al. (1994) in context with the subjective increased, prior to decreased $RPE_{breathing}$ in the present study suggests that an adjustment period is needed in order for conscious changes to breathing pattern to have a beneficial effect on $RPE_{breathing}$ during high

intensity exercise. It is not known how long this adjustment period needs to be in order to be effective although it would have been interesting to observe what changes may have occurred had participants in Fabre et al.'s (2007) and Maclennan et al.'s (1994) studies had time to adjust to the altered breathing pattern.

5.3.2. Perceived Leg Effort

In addition to decreasing $RPE_{breathing}$, four weeks of BPR also decreased (benefit likely, Table 11) perceptions of leg effort (RPE_{legs}) in the incremental test. The percent change in RPE_{legs} was similar at LT (-27.9 ± 38.5%) and LTP (-24.7 ± 28.3%). As observed for $RPE_{breathing}$, the decrease in RPE_{legs} is larger than that observed after IMT in cyclists (-18.4%) (Romer, McConnell, & Jones, 2002a) or repeated sprint activity (7.2 ± 0.6%) (Romer, McConnell, & Jones, 2002b). Similar to breathing RPE, the control group in the present study rated their RPE_{legs} as greater at LT (13.4 ± 39.6%) and LTP (19.9 ± 43.2%) during the post-intervention test, demonstrating a potential detraining effect.

The decrease in RPE_{legs} may have been simply due to the same mechanism that resulted in decreased $RPE_{breathing}$. It is possible that BPR prevented or decreased dynamic hyperinflation which subsequently decreased the work of breathing as discussed above. Harms et al. (1997) demonstrated that decreasing the work of breathing significantly increased blood flow to the legs in cyclists, which may aid the clearance of metabolites usually associated with peripheral fatigue (Cain, 1973). Blood pH is a mediator of perceptions of peripheral exertion, especially at exercise intensities that equal or exceed the lactate threshold as in the present study (Kostka & Cafarelli, 1982). By preventing blood stealing, blood flow to the legs may have been preserved allowing appropriate clearance of metabolites and affecting pH to a degree that peripheral exertion was perceived as less.

5.4. Effects of BPR on Metabolic Measures

Breathing pattern did not clearly influence any of the physiological variables measured in the present study, although trends suggest that BPR may positively influence B[La], power and heart rate at LTP (Table 9). This may be significant given the nature of physiological events that occur when exercising at and above LTP.

5.4.1. Lactate, Power and Heart Rate – LT and LTP

Ventilatory work during heavy endurance exercise may contribute to the accumulation of metabolites (Johnson, Sharpe, & Brown, 2007) that exacerbate respiratory and locomotor muscle fatigue. One of the physiological parameters that is most often associated with improved exercise performance following IMT is an attenuated B[La] response (Boutellier, Buchel, Kundert, & Spengler, 1992; Boutellier & Piwko, 1992; McConnell & Sharpe, 2005; Romer, McConnell, & Jones, 2002b; Spengler, Roos, Laube, & Boutellier, 1999) which suggests that metabolic responses can be modified by a breathing intervention. The present study failed to show a clear reduction in B[La] at absolute workloads pre- and post-BPR, however there was a trend for B[La] to be decreased at LTP (-4.8 ± 16.6%) but not LT (2.7 ± 16%) after BPR, though unclear because of the sizeable 90% confidence intervals. It is difficult to understand why IMT affected B[La] whilst the present study failed to show such a response. Spengler et al (1999) suggested that IMT was effective in decreasing B[La] by increasing the ability of the respiratory muscles to use more lactate as fuel to maintain work output. Because BPR does not specifically train the strength or endurance of the respiratory muscles it would be unlikely to induce an effect on lactate turnover, which may explain the difference. Rather, BPR served to decrease the work of breathing by optimising respiratory mechanics (non-physiological) which decreased the perception of dyspnoea and work of breathing and may lead to a reduction of blood stealing.

The present study measured B[La] during incremental exercise – a performance test that McConnell and Romer (2005) suggested would overwhelm any alterations to B[La] turnover that had been induced by IMT, which may explain why this parameter did not significantly change in the present study. Volianitis et al. (2001) also measured B[La] during incremental exercise and, like the present study, demonstrated a trend for B[La] to decrease, without reaching statistical significance. In contrast, previous studies that reported large changes in B[La] were measured during an endurance or sub maximal test – where it is thought the ability to limit the effect of gradually increasing blood lactate may be enhanced (McConnell, 2005). It is possible that a larger change, more consistent with previous findings, may have been observed if B[La] had been measured during the 20km-TT. Of course, it is possible that because BPR does not specifically increase the strength and endurance of the respiratory muscles, its ergogenic effect is not related to minimising the B[La] response regardless of the nature of the test protocol.

Whilst power at LTP showed a trend to increase (Table 9), this was not a clear difference – a finding consistent with McConnell and Sharpe (2005) who demonstrated no change to MLSS power after four weeks of IMT. Further, heart rate was unchanged in the present study (despite the trend to be decreased at LTP; Table 9) which is consistent with the change observed after IMT (Romer, McConnell, & Jones, 2002a). Collectively, this data suggests that the ergogenic effect of BPR is unlikely to be related to changes in these physiological (metabolic) variables.

5.4.2. Measures of Oxygen Uptake - sub-maximal (economy) and VO$_{2peak}$

The present study measured sub-maximal $\dot{V}O_2$ and $\dot{V}O_{2\,peak}$ in an attempt to determine whether BPR could reduce or affect the O_2 cost (work) of breathing associated with incremental exercise. Regardless of expression (relative or absolute), BPR had no effect on the O_2 of cycling (comparison of absolute $\dot{V}O_2$ at same power output pre- and post-intervention), $\dot{V}O_{2\,peak}$ (or

$\dot{V}CO_2$) in either the intervention or the control group, a finding that is consistent with the lack of effect reported by other authors who have demonstrated performance improvements after IMT (Griffiths & McConnell, 2007; Romer, McConnell, & Jones, 2002a), VIH (Holm, Sattler, & Fregosi, 2004; Leddy et al., 2007; McMahon, Boutellier, Smith, & Spengler, 2002) or acute breathing pacing strategies (Fabre, Perrey, Arbez, & Rouillon, 2007).

Potentially, changes in sub-maximal $\dot{V}O_2$ during cycling could be achieved by reducing the O_2 cost of breathing by optimizing the work of breathing. Wetter et al. (1999) demonstrated that decreasing the work of breathing by $40 \pm 6\%$ using a proportional assist ventilator at 75% $\dot{V}O_{2\,max}$ significantly decreased $\dot{V}O_2$ from 3.2 ± 0.1 to 3.1 ± 0.1 L·min^{-1}. At $\dot{V}O_{2\,max}$ intensity, Harms et al. (1997) demonstrated a significant decrease in whole-body $\dot{V}O_2$ (6.2%) when respiratory muscles were offloaded by 63%, which resulted in an 8% increase in $\dot{V}O_{2\,legs}$ showing that blood (and hence O_2) stealing occurred at maximal intensity. When respiratory muscle work at $\dot{V}O_{2\,max}$ intensity was increased by 28%, $\dot{V}O_{2\,legs}$ was significantly decreased (9.4%) but no change in whole-body $\dot{V}O_2$ was evident. In the present study, however, no changes in O_2 cost of exercise were observed after BPR (Table 9). To expect BPR to decrease the work of breathing by 63%, or even 40%, would be ambitious. It is likely that the effects of BPR on the work of breathing were much smaller and this could explain why no changes were detected in either $\dot{V}O_2$ or $\dot{V}O_{2\,peak}$ in the present study. Although no changes to $\dot{V}O_{2\,peak}$ were observed, the distribution of $\dot{V}O_2$ between the legs and respiratory muscles may have changed such that $\dot{V}O_{2\,legs}$ was enhanced even if only by a few percent (compared with the much greater 8% increase with PVA). To be able to detect such changes was beyond the scope of this study.

However, the ability to measure and differentiate Q_{legs} from $\dot{V}O_{2\ TOT}$ may enhance our understanding of the influence of BPR on blood and oxygen distribution and utilisation.

In summary, the minimal physiological changes observed after BPR are generally consistent with previous findings for other breathing interventions such as IMT and VIH. This could be the result of actual negligible change or a meaningful change but a lack of sensitivity of measures to detect such small changes in the physiological measures included as part of this study. The mechanisms underpinning how BPR contributed to improved time trial and incremental performance may be more related to changes in respiratory muscle work, than to any physiological adaptations given the unremarkable changes to physiological variables observed in the present study.

5.5. Limitations

The findings presented in this thesis are considered unique insofar as this is the first study to investigate the use of a physiotherapy intervention traditionally used with chronic respiratory conditions, to enhance performance in sport. Consequently, there were no methodological blueprints to guide the direction of this study. Perhaps understandably, there are a number of limitations in this study.

To further understand the effect of BPR on performance it would have been useful to measure respiratory muscle recruitment pattern, dynamic hyperinflation and locomotor muscle blood flow. Understanding whether changes to these variables occurred would provide a greater understanding of whether, and then how, BPR may affect exercise performance. Unfortunately, the resources were not available to measures these parameters, therefore it is not possible to objectively claim that BPR altered or delayed recruitment of the accessory muscles. Similarly, as

objective measures of dynamic hyperinflation during exercise were not utilised it is unclear whether dynamic hyperinflation was i) present pre-BPR or ii) altered post-BPR. Advanced physiological measures such as limb blood flow were not measured, adding to the difficulty ascertaining how BPR may have influenced performance from a mechanistic perspective.

The extent to which the respiratory muscles fatigued following endurance and incremental cycling was not established in the present study. In this thesis, it has been speculated that some of the decrease in MIP observed by others after exhaustive exercise may not be solely due to inspiratory muscle fatigue, but may be decreased due to the shift in mechanical advantage of the respiratory muscles caused by a tachypnoeic breathing pattern. To this end, MIP could have been measured pre- and post-BPR to determine whether BPR did indeed have an effect on this variable. In addition, it is possible that the four week intervention period did not allow the full effect of BPR to be realised (and observed/measured) for the reasons discussed in *section 5.1.1*. However this time frame was chosen to ensure compliance of the participants with i) their BPR exercises, and ii) was the maximum compliable time for standardisation of training during the subjects competitive season. Although training diaries were provided to all participants, compliance with accurately recorded heart rate and training volume was insufficient to accurately quantify consistency of training volume during the intervention period. However, analysis of the available data indicated there were no large variations in weekly training load in either group throughout the study.

Finally, it could be argued that failing to set the cadence in the present study may have introduced a confounding variable that affected breathing pattern. However, Prabhu et al. (1992) found no change in f_b, V_T or t_i/t_{TOT} between pedalling rates from 40-80rpm and participants in the

present study were required to replicate their self-selected cadence between pre- and post-intervention assessments.

5.6. Future Directions

The mechanisms underpinning *how* BPR resulted in enhanced endurance and incremental cycling performance remain unclear. It is speculated that BPR results in changes to the automatic breathing pattern, and that collectively these changes to respiratory mechanics may benefit an athlete in three ways. Firstly, prevention of a tachypnoeic shift after BPR may have a direct effect on inspiratory muscle fatigue by enhancing the mechanical advantage of the respiratory muscle during intense exercise. Secondly, improved breathing mechanics may have an indirect effect of improving blood flow distribution to the leg muscles during intense exercise thus optimising power output. Thirdly improved respiratory mechanics may have an effect on the intensity with which respiratory and peripheral efforts are perceived allowing the athlete to exert themselves more.

In light of the positive outcome of the present study the list of questions still deserving attention is long. For example, does BPR actually influence the inspiratory muscle recruitment pattern, dynamic hyperinflation and respiratory muscle fatigue? Does BPR actually affect locomotor muscle blood flow? What mechanisms underlie *how* the automatic breathing pattern is retrained, how long does it take and once changes are established are they lasting? What is the incidence of breathing pattern disorders in athletes in the absence of respiratory pathology?

To address some of these questions a number of suggestions for future studies can be made. Increasing the intervention period would allow further understanding of whether BPR had a greater effect on exercise performance once the new pattern was assimilated into training.

Maximal inspiratory pressure or some measure of respiratory muscle fatigue needs to be recorded to assess whether BPR influences respiratory muscle fatigue. Similarly, recording leg blood flow during cycling pre- and post-BPR would help the understanding as to whether BPR enhances exercise performance by preventing blood stealing. An objective tool to measure the incidence of breathing pattern disorders in athletes needs to be developed or the current practice of diagnosing breathing pattern disorders based on pattern observation needs to be validated.

5.7. Conclusion

The results of the present study suggest BPR has a positive influence on 20km-TT performance and incremental power during cycling in trained athletes. In the absence of any strong physiological changes, it appears the observed changes in performance were due to altered respiratory mechanics and a reduced perception of exertion. Specifically, BPR substantially decreased f_b and V_E, and increased V_T and t_I compared with pre-intervention values, resulting in a more controlled breathing pattern at high exercise intensities. The mechanisms underpinning the ergogenic effect of BPR remain unclear. Further research to enhance our understanding of the role that BPR has in sport performance is clearly warranted.

Appendices

Appendix 1 Nijmegen Questionnaire

Nijmegen Questionnaire

	Never		Rare		Sometimes		Often		Very Often	
	1	2	1	2	1	2	1	2	1	2
Chest Pain										
Feeling Tense										
Blurred Vision										
Dizzy spells / light headed										
Confusion										
Faster / deeper breathing										
Shortness of Breath										
Tight feelings in the chest										
Bloated feeling in the stomach										
Tingling Fingers										
Unable to breathe deeply										
Stiff fingers or arms										
Tight feelings around the mouth, jaw or throat										
Cold hands or feet										
Heart racing (palpitations)										
Feelings of Anxiety										

Never = 0   Rare = 1   Sometimes = 2   Often = 3   Very Often = 4

Total 1   / 64   date……………………..

Total 2   / 64   date……………………..

Nijmegen Descriptors[3]

Never: Never.

Rare: Once a month.

Sometimes: Once a week.

Often: More than once a week but not every day.

Very often: Daily or more.

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[3] Participants have a copy of these descriptors in front of them when they complete the Nijmegen Questionnaire

Appendix 2 Clinical Breathing Retraining

Despite the fact that breathing pattern retraining has been used by physiotherapists since the 1930's (Soley & Shock, 1938) there are no "gold standard" treatment methods. Instead the basic method described by Lum (1983) has been modified and integrated into clinical practice by clinicians working in the area of breathing pattern disorders and retraining. The description provided below is a general overview, summarizing current clinical practice. The reader is reminded that, to date, no studies have investigated time frames, appropriate intervention strategies or physiological reasons for how and/or why breathing pattern retraining may be an effective tool for optimising respiratory mechanics. Therefore much of this description is provided without scientific rational for its existence, instead it is based on the experience of the author and other physiotherapists working in the field of BPDs (Bradley, 1992; Chaitow, Bradley, & Gilbert, 2002).

There are two components of breathing pattern retraining. The first involves learning a new motor pattern, to train the inspiratory muscles to be recruited in the optimal way. Just like learning a new golf swing or swimming technique, the initial phase of learning and retraining requires intense concentration. With further practice the new muscle firing pattern will become more efficient and eventually automatic (Wilmore & Costill, 1999) and based on studies investigating neural plasticity and motor skill acquisition this may occur anytime in the first four to six weeks of retraining (Enorka, 1997). There are two main techniques involved in breathing pattern retraining – diaphragmatic breathing and pursed lip breathing. Diaphragmatic breathing retraining requires a conscious appreciation of inspiring air to the lung bases with a slight forward abdominal displacement and passive, relaxed expiration (Jones, Dean, & Chow, 2003) and is used to delay recruitment of the accessory breathing muscles and to encourage breathing at optimal lung volumes. Pursed lip breathing is taught to help elongate the expiratory phase of

breathing to maintain an optimal inspiratory to expiratory ration, prevent breath stacking and to avoid a tachypnoeic shift in breathing pattern (Jones, Dean, & Chow, 2003).

A patient is often able to demonstrate a diaphragmatic breathing pattern within the first treatment session, albeit not comfortably, and to continue to demonstrate it correctly when requested. At this point the diaphragm pattern is a learned motor skill that can be used to voluntarily override the automatic pattern.

The second part of breathing pattern retraining is more difficult as it involves re-programming the brainstem respiratory centres and based on Grossman et al.'s theory (1985) involves retraining the body to respond to a higher level of CO_2. The normal reaction of the body to a rising CO_2 level is to increase the respiratory rate to bring the CO_2 level back to appropriate parameters (Wilmore & Costill, 1999). When a breathing pattern disorder is well established, the CO_2 in the blood stream may be lower than normal (Garssen, de Ruiter, & van Dyke, 1992). One aim of breathing pattern retraining is to decrease the respiratory rate to 12-14 breaths per minute. As a result, $PaCO_2$ starts to rise relative to the brainstems incorrect perception of "normal" and the appropriate but undesired response of trying to increase respiration is initiated. Clinicians find that this part of breathing pattern retraining takes patients the most time, and is most difficult to overcome as it feels uncomfortable and unnatural to the patient. It has been suggested that decreased respiratory rate can only be tolerated for up to ten minutes at a time, and only small increases in CO_2 can be withstood before the urge to breathe is overwhelming (Grossman, DeSwart, & Defares, 1985). A session of breathing pattern retraining is usually broken into small groups of "correct" breathing, interspersed with the patient's comfortable pattern, to build tolerance to the new CO_2 levels. The length of time required to adjust to the appropriate $PaCO_2$ is unknown. However, it is likely that a breathing pattern disorder will not be overcome, despite

that ability to demonstrate a diaphragmatic breathing pattern on demand, until the $PaCO_2$ level has been "reset".

It is common, and almost expected, that patients will feel significant discomfort when trying to correct their breathing pattern (Innocenti, 1998). These symptoms are explained to patients at the outset of retraining and further discussion can take place if they do arise. If a breathing pattern disorder is well established it will feel comfortable and even normal to the patient, despite the symptoms generated. Correction of breathing pattern will be opposed by the decreased respiratory rate causing increased CO_2 which may initiate feelings of 'air hunger'. Further, a patient may be used to breathing at high lung volumes secondary to dynamic hyperinflation. A full, relaxed exhalation will alter operating lung volumes and give the impression that no enough air is being taken into the lungs. Additionally, if mouth breathing is a habit, the increased (but appropriate) resistance of nose breathing will feel difficult at first (Garssen, de Ruiter, & van Dyke, 1992). All of these symptoms will decrease with time and practice. If retraining is consistent and regular the diaphragmatic breathing pattern appears to be relearned and re-programmed. It is possible that once automatic breathing has been reprogrammed, and causative factors have been addressed, (for example, anxiety or postural factors) no ongoing breathing intervention would be necessary. This is unlike strength training, including respiratory muscle training, which requires an ongoing maintenance program to preserve strength gains (Wilmore & Costill, 1999).

Appendix 3 Breathing Retraining

10 minutes Daily

- Lie on your back, both hands firmly on the top of your chest.
- **First breath** - breathe in through your nose, out through your mouth, pressing down on your upper chest as you breathe out. Feel the top of your chest deflate.
- All the **rest of your breaths** MUST be in and out of your nose.

- Keep the firm pressure on the top of your chest so that when you breathe, you cannot move your upper chest. Your stomach will move up as you breathe in and fall as you breathe out. This is not a push of your stomach, it just moves as your lungs expand and deflate.
- Try to breathe to the following 1,2,3,4 rhythm:
  > BREATHE IN for the count of "1",
  > BREATHE OUT for the count of "2 and 3",
  > PAUSE for "4".
- Aim for your out breath to be twice as long as your in breath with a slight pause after you breathe out.
- Initially start with 5 breaths in this rhythm, then revert to your comfortable breathing. Spend 10 minutes alternating between the correct pattern and your comfortable breathing.
- As you get used to it, try to breathe with the correct pattern for more breaths in a row until you can comfortably breathe for 10 minutes with the correct rhythm.
- Continue with 10 minutes every day. The correct pattern WILL get easier the more you practice.

Gold Stars
- These are to remind you to correct your breathing through out the day.
- Put them in various places in your environment e.g. on your head stem, on your computer monitor, dashboard, phone etc.
- When you see one do the following:
  - Stop mentally what you are thinking about,
  - Drop your shoulders, upper chest and breathe out,
  - Take three normal size breaths into your stomach.
- It is very important that you do this to "reprogram" your breathing.

When Exercising
- Try to keep breathing into your stomach for as long as possible. At some point you may revert to breathing 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.

Appendix 4 Training Diary

TRAINING DIARY
AUT University
Contact: Rachel Vickery, 0211779309, rachinaus@hotmail.com

- Fill in the date, the duration, the distance in kms, heart rate (average and max) and average speed.
- Then mark the intensity of the session, and how breathless you felt when you did it (using the 0-10 scale at the front of your diary).
- If you do sessions other than on the bike please indicate what type of session you did.
- Record training as soon as possible after each session.
- Bring this diary with you each week you come to the Millennium Institute

Date	Duration	Distance	HR (Average & Max)	Average Speed	Intensity 0-10	Breathlessness: 0-10	Any Comment? (Breathing Exercises)*
Monday							
Tuesday							
Wednesday							
Thursday							
Friday							
Saturday							
Sunday							

* The comment on breathing exercises was only included in the diaries given to the Intervention Group participants

Total

Appendix 5 Modified Borg Scale

<u>Modified BORG scale</u>

SCALE	SEVERITY
0	No Breathlessness At All
0.5	Very, Very Slight (Just Noticeable)
1	Very Slight
2	Slight Breathlessness
3	Moderate
4	Some What Severe
5	Severe Breathlessness
6	
7	Very Severe Breathlessness
8	
9	Very, Very Severe (Almost Maximum)
10	Maximum

Appendix 6 Patient Handout – Potential Symptoms associated with BPR

<u>What You May Feel</u>

When you start to change your breathing pattern it is normal for it to feel uncomfortable. Some people describe feelings like:

- Air hunger
- "not getting enough air in"
- "not breathing in long enough"
- Light headed
- Dizziness
- Needing to take a deep breath in after finishing the "correct" breaths.
- General discomfort

All these feelings are normal. When you breathe slower (correctly) your body is retaining more carbon dioxide than what it is used to. Usually when our carbon dioxide level gets too high we feel the need to breathe more (think about how you feel when you have held your breath as long as possible). It takes time for your body to adjust to the new carbon dioxide level. Over time these feelings will decrease and the new way of breathing will feel much easier. The only thing that will speed the change over time is spending your 10 minutes daily breathing with the 1,2,3,4 pattern, using your gold stars and trying to breathe to your stomach when you are riding.

Initially you may feel more out of breath as you exercise, or you may feel that it is harder to breathe when you ride. Again this is normal, as you are breathing through a smaller hole, however this will also improve and the more you practice the quicker you will adjust.

Appendix 7 Participant Handout – Rationale for Exercises

Why do I have to do my 10 minutes practice every day?

There are two parts to changing your breathing pattern. The first is the easy part and is as simple as learning to stride out more when you run. It is what we call a motor skill or technique. The gold stars are used as a reminder for you to be aware of how you are breathing and to correct your technique if necessary. It is important to break the habit of how you are breathing, and use the correct technique. This will be achieved through awareness of how you are breathing, and consciously changing to the new technique of breathing to your belly. It is important to use the visual triggers (gold stars). From experience you will not correct your breathing technique as often if you don't use them, and it will then be harder to breathe properly when you ride.

The second part is the hardest part as you need to reprogram the part of your brain that controls breathing. When you breathe incorrectly over a period of time your brain gets used to a certain level of carbon dioxide in your blood stream (usually a lower level) and bases future breathing rates and depths on this. Although you can override your brain's program (for example, holding your breath under water, or breathing really quickly on command), over-riding your brain's program can not be tolerated for longer than about 10 minutes. The only way you can achieve a correct breathing pattern long term is to reset the level of carbon dioxide that your brain responds to. This is why you do your 10 minutes of correct breathing! By consciously slowing your breathing rate, your carbon dioxide level will slowly normalise and you will be able to tolerate the correct breathing pattern longer.

Combining the awareness and correction of how you are breathing (gold stars), with the tolerance to maintain this correct pattern, will help you breathe more efficiently on the bike.

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