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THE EFFECTS OF RESPIRATORY MUSCLE TRAINING ON AEROBIC PERFORMANCE IN MODERATELY TRAINED CYCLISTS

by

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies for acceptance, a thesis entitled "The effects of respiratory muscle training on aerobic performance in moderately trained cyclists" submitted by Sidd Thakore in partial fulfillment of the requirements for the degree of Master of Science.

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Abstract

This study examined the effects of 4 weeks of pressure resistant respiratory muscle training (RMT) on 8km bicycle time trial performance in 23 moderately trained cyclists (13 RMT group and 10 placebo group). The experimental design included RMT with inspiratory resistance equivalent to 50% of maximum inspiratory pressure (MIP) (3 sets of 30breaths/day, 6 days/week) and a breathing frequency of 35 breaths per minute. The placebo group followed a similar protocol but used a resistance equivalent to 10% of MIP. The RMT group showed a significant improvement in global respiratory muscle strength and endurance ($P<0.05$) but not in diaphragm strength ($P>0.05$). The 8km time trial performance decreased significantly in the placebo group by 2.4 +/-2.0% ($P = 0.004$) and showed a variable non-significant change in the RMT group (-0.4 +/-2.1%) ($P = 0.05$). It is concluded that RMT has no effect on aerobic performance in moderately trained cyclists..

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Dedication

My family for their utmost support in all my endeavors, as crazy as they might be.

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CHAPTER ONE

INTRODUCTION

Background

Traditionally, diffusion capacity, cardiac output, hemoglobin concentration, blood volume, and locomotor muscle oxygen extraction ability have been identified as factors that limit aerobic performance (Bassett and Howley 1997). More recently, it has been suggested that components of the respiratory system particularly the respiratory muscles (RM) may also limit aerobic performance (Boutellier, Buchel et al. 1992; Boutellier and Piwko 1992; Johnson, Aaron et al. 1996; Boutellier 1998; Sonetti, Wetter et al. 2001; Volianitis, McConnell et al. 2001; Romer, McConnell et al. 2002; Romer, McConnell et al. 2002).

Following exhaustive exercise, the RM may demonstrate signs of fatigue (Coast, Clifford et al. 1990; Johnson, Babcock et al. 1993; McConnell, Caine et al. 1997; Volianitis, McConnell et al. 2001; Romer, McConnell et al. 2002). It has been suggested that such fatigue could impair athletic aerobic performance by decreasing blood flow to the locomotor muscles, increasing motor unit recruitment and disrupting metabolic homeostasis (Loke, Mahler et al. 1982; Bye, Esau et al. 1984; Johnson, Aaron et al. 1996; Harms, Wetter et al. 2000; Sonetti, Wetter et al. 2001).

Numerous investigations have used respiratory muscle training (RMT) to increase respiratory muscle strength and endurance (Fairbarn, Coutts et al. 1991; Boutellier, Buchel et al. 1992; Boutellier and Piwko 1992; Suzuki, Yoshiike et al. 1993; Tzelepis, Vega et al. 1994; Tzelepis, Vega et al. 1994; Spengler, Laube et al. 1996; Spengler, Roos et al. 1999; Tzelepis, Kasas et al. 1999; Harms 2000; Inbar, Weiner et al. 2000; Spengler,

Knopfli-Lenzin et al. 2000; Markov, Spengler et al. 2001; Sonetti, Wetter et al. 2001; Stuessi, Spengler et al. 2001; Volianitis, McConnell et al. 2001). Whether these increases result in improved athletic performance is uncertain because of conflicting study results.

Statement of the Problem

The concept of RMT as a performance-enhancing training method has received limited study until recently. The term RMT refers to a training overload on the RM's caused by an increased ventilatory effort. This increased ventilatory effort is either from increased ventilation rates or increased resistance to breathing. Typical RMT methods utilize isocapnic hyperpnoea, flow resistive training or pressure resistive training. Although RMT has consistently demonstrated the ability to strengthen the respiratory muscles in humans, the results of studies examining its effect on aerobic performance are conflicting. Possible reasons for these conflicting results include:

1. Small sample sizes
2. Varying subject characteristics (with respect to gender and training status)
3. Poorly selected performance outcome measures
4. Non-validated training devices (with respect to accuracy and reliability of breathing resistance).
5. Poor study design
6. Different RMT regimes (with respect to the training load, duration, type of device and breathing pattern)
7. RMT does not improve performance

Some ways of addressing the limitations of previous studies include:

1. Using a randomized clinical trial study design (one experimental and one placebo group)
2. Appropriate sample size supporting a small effect size
3. Using a validated and reliable RMT device
4. Using a RMT regime previously demonstrated to elicit performance improvements
5. Control of breathing frequency (*f*) and tidal volume (V_T) during RMT.

Statement of Purpose

The primary purpose of this study was to determine the effects of RMT on 8km cycling time trial performance.

The secondary purpose of this study was to investigate how RMT affects various physiological parameters that may be influenced by RMT and subsequently improve performance. These parameters are: maximum oxygen uptake (VO_{2max}); maximum aerobic power (MAP); respiratory muscle strength and endurance; blood lactate concentration; perception of breathlessness; perception of muscular effort; V_T; *f*.

Statement of Hypothesis

The primary study hypothesis is:

- The 8km cycle time trial time will be significantly shorter in the RMT group compared to the placebo group.

The secondary study hypotheses are:

- Global RM strength and endurance and diaphragm muscle strength will improve significantly in the RMT group compared to the placebo group.
- The work of breathing will decrease significantly in the RMT group compared to the control group. This will be expressed as a significantly lower *f*, VO_2, V_E, and increased V_T in the RMT group compared to the control group for a given constant high intensity power output.
- The blood lactate concentration will be significantly lower in the RMT group compared to the control group for a constant high intensity power output.
- The perception of breathlessness and the perception of muscular effort by the legs will be significantly lower in the RMT group compared to the control group for a constant high intensity power output.

Significance of Study

It is important that every new method of training undergoes adequate scientific scrutiny before it is accepted in practice. Currently, the benefits of RMT on aerobic performance are uncertain. The results of this study will help determine whether RMT improves performance in moderately trained cyclists. Additionally, it will contribute new knowledge to the literature regarding the effects of RMT with a controlled breathing pattern on RM function.

CHAPTER TWO
REVIEW OF LITERATURE

Introduction

The inspiratory process of respiration is accomplished by the upper rib cage and sternum moving cephalad and ventrally, the lateral part of the lower rib cage moving outward and the diaphragm moving downward (Epstein 1994). All these actions result in a subatmospheric interthoracic pressure that facilitates the movement of air into the lung. The expiratory process of respiration during resting breathing is passive and utilizes the elastic energy stored in the lungs and inspiratory muscles (Celli 1998). During exercise or forced expiration, this process becomes active with the compression of the rib cage and abdominal muscles, all which act to increase the interthoracic pressure (Celli 1998).

Functional Anatomy

Diaphragm

The diaphragm is the primary inspiratory muscle (Loring and DeTroyer 1986). Traditionally, the diaphragm was though of as a single dome shaped muscle that inserted into the lower ribcage (Loring and DeTroyer 1986). However, there is evidence that the diaphragm consists of two independently operating musculotendinous segments, a costal segment and crural segment that are connected by the non contractile central tendon (De Troyer, Sampson et al. 1981).

The identification of the crural and costal portions of the diaphragm as separate muscles arises from their different embryological origin, innervations, and muscle fiber content. The human costal segment arises from the myoblasts originating in the lateral body walls whereas the crural segment of the diaphragm develops in the dorsal mesentery

of the esophagus (Loring and DeTroyer 1986). Anatomically, the costal segment originates from the central tendon and inserts on the xiphoid process anteriorly, the lower costal margin anterolaterally, and the tenth to twelfth ribs laterally while the crural segment originates from the anterior surface of the lumbar spine and surrounds the esophagus hiatus (Rochester 1992; Abe, Kusuhara et al. 1993). The crural segment has a higher proportion of slow twitch oxidative fibers and a lower proportion of fast twitch glycolytic fibers than the costal segment (Riley and Berger 1979). EMG studies have confirmed that the crural diaphragm is innervated by C6 and C7 while the costal diaphragm is innervated by C5 and C6 (De Troyer, Sampson et al. 1982). These muscles also differ in their mechanical action on each segment of the ribcage. DeTroyer et al (1982) demonstrated that the costal segment expands the lower rib cage and increases both lung volume and abdominal pressure. The crural segment also increases lung volume, abdominal pressure and abdominal dimension but has no effect on the lower rib cage. When the abdomen is open, the crural segment has an expiratory action on the lower rib cage, due to the fall in pleural pressure when it contracts.

Based on the innervations and action of the two segments of the diaphragm, it has been proposed that costal diaphragm is in series while the crural segment is in parallel with the intercostals and accessory muscle (De Troyer, Sampson et al. 1981). However, a subsequent study by DeCramer et al (1984) has suggested that the costal and crural portions of the diaphragm behave as if they are mechanically arranged partly in parallel and partly in series at functional residual capacity (FRC) but move into a pure mechanical series arrangement as lung volume increases.

The diaphragm accounts for approximately 60% of the total volume displacement between residual volume (RV) to total lung capacity (TLC), with its relative contribution being approximately 25% greater between RV and FRC than between FRC and TLC (Rochester 1992). It is the axial portion of the diaphragm that shortens the most during inspiration and represents 1/4 to 1/3 of the total surface area of the rib cage during quiet breathing (Loring and DeTroyer 1986). The inspiratory action of the diaphragm can be explained in three ways. First, the piston like axial displacement of the diaphragm forces the abdominal contents downward and forward thus increasing the vertical dimension of the chest cavity and the transverse diameter of the thorax (Mead 1979; Troyer and Loring 1986). Second, the increased intra abdominal pressure from the descent of the diaphragm dome is transmitted across the zone of apposition, pushing the lower ribs outward and resulting in rib cage expansion (De Troyer and Estenne 1988). Finally, contraction of the abdominal contents during inspiration opposes the descent of the diaphragm dome and the force exerted on the lower ribs is maintained cranially resulting in an upward and outward movement of the lower ribs (De Troyer and Estenne 1988).

The Intercostal Muscles

The intercostal muscles are composed of the external and internal fibers, both innervated by T1-T12 (Epstein 1994). According to the original theory proposed by Hamberger (1749), contraction of external intercostal muscle will tend to raise the ribs and inflate the lungs because of their orientation and insertion points. This is because the muscle fibers slope caudal and ventrally from the rib above to the rib below and their lower insertion is further from the center of rotation of the ribs than their upper insertion.

In contrast, the internal intercostals muscles are oriented in an opposite fashion, sloping obliquely caudal and dorsally form the rib above to the one below so that their lower insertion is closer to the center of rotation of the ribs than the upper one. Consequently, this muscle will lower the ribs and deflate the lungs (DeTroyer et al, 1999).

Recently, De Troyer et al (1999) identified a neural gradient between the dorsal and ventral parts of the rib cage and between the rostral and caudal interspaces and this gradient mirrors the mechanical advantage gradient of both intercostals muscles. The external intercostals in the dorsal third of the rostral interspaces have a large inspiratory mechanical advantage which decreases and reverses to an expiratory mechanical advantages as one moves toward the costochondral junctions and toward the base of the rib cage (De Troyer, Legrand et al. 1999). On the other hand, the internal intercostals in the dorsal portions of the caudal interspaces have a large expiratory mechanical advantage which decreases and reverses to an inspiratory mechanical advantage as one moves ventrally and cranially toward the rostral interspaces (De Troyer, Legrand et al. 1999). As one moves in the cranial and ventral direction, internal intercostal expiratory activity decreases until it is absent in the middle and ventral portions of the most rostral segments. A similar effect is observed with external intercostal inspiratory muscle activity as one moves toward the costochondral junctions and toward the base of the rib cage (De Troyer, Legrand et al. 1999).

Other studies have reported efferent discharge to the external intercostal muscles in the caudal section during expiration and to the internal intercostal muscles in the second interspace during inspiration (Le Bars and Duron 1984). It has also been reported

that the external and internal intercostals in a given interspace often change their length in the same direction during breathing (Decramer, Kelly et al. 1986).

The classical classification of the parasternal as an external intercostal muscle should also be addressed. Anatomically, this muscle should be classified as an internal intercostal muscle because there is only one layer in this region (De Troyer, Legrand et al. 1999). Since this muscle plays a predominant role in inspiration, it also refutes Hamberger's theory that internal and external intercostal muscles are responsible only for expiration and inspiration respectively. Needle EMG studies demonstrate the parasternal muscle is always activated during primary breathing and is considered a primary inspiratory muscle (De Troyer and Sampson 1982).

Scalenes

The scalenes muscles (anterior, medius, and posterior) run from the transverse processes of the lower five cervical vertebrae to the superior aspect of the first and second ribs and are innervated by C4-8 (Epstein 1994). Needle EMG studies show these muscles to be the primary muscles of respiration as activity begins at the onset of inspiration, in concert with the diaphragm and parasternal muscles, and peaks at the end of inspiration (De Troyer and Estenne 1984). The action of this muscle is to lift the sternum and the first two ribs, leading to an upward and outward expansion of the upper rib cage (De Troyer and Estenne 1984).

Abdominal Muscles

The abdominal muscles are considered the most powerful expiratory muscles and include the external oblique, rectus abdominus, internal oblique and transverses abdominus muscles (Cambell, Agostoni et al. 1970). The external oblique is the most superficial abdominal muscle and originates by fleshy digitations from the external surfaces of the lower eight ribs and inserts into both the iliac crest inferiorly and the linea alba medially (Loring and DeTroyer 1986). The internal oblique originates form the inguinal ligament, iliac crest and lower portion of the lumbar aponeurosis and inserts into the anterolateral surfaces of the cartilages of the last three ribs (Loring and DeTroyer 1986). The transverses abdominus lies deeper to these muscles and originates from the inner surface of the cartilages of the last six ribs, lumbar fascia, iliac crest and the inguinal ligament and inserts into the aponeurosis that form the posterior lamina of the rectus sheath (Loring and DeTroyer 1986). The rectus abdominus extends axially along the whole length of the ventral part of the abdomen, external surfaces of the 5^{th}, 6^{th} and 7^{th} costal cartilages and inserts to the pubis (Loring and DeTroyer 1986). The rectus abdominus is innervated by T7-12 while the other abdominal muscles are innervated by T7-L1 (Epstein 1994).

During resting respiration, expiration is predominately a passive process that utilizes the energy stored in the elastic properties of the lungs and inspiratory muscles (Epstein 1994). However, during exercise and voluntary hyperventilation there is an increased recruitment of the expiratory muscles which act to deflate the rib cage by pulling the lower ribs and sternum down (De Troyer, Sampson et al. 1983). This action

will raise the intra-abdominal pressure and decrease the end expiratory volume (West 1995; Aliverti, Cala et al. 1997).. These muscles also prevent bulging of the intercostals spaces by stiffening during contraction. Although the abdominal muscles are generally regarded as expiratory in nature, their role during inspiration should not be underestimated. The increase in abdominal pressure during active expiration places the diaphragm at a more advantageous position on its length tension curve by displacing it further into the thorax and increases the passive elastic energy of these muscles (Troyer and Loring 1986; Aliverti, Cala et al. 1997). This energy is released during the beginning of inspiration, thus contributing to the inspiratory process (Troyer and Loring 1986). In addition to their respiratory, the abdominal muscles are responsible for postural activity (Loring and DeTroyer 1986).

Sternomastoids Muscle

The sternomastoids runs from the mastoid process and the occipital bone to the manubrium and the medial aspect of the clavicle and is innervated by cranial nerve X1 and C1-2 (Epstein 1994). This muscle is quiet during resting respiration but is activated during high minute ventilation and lung volume and contributes to inspiration by increasing tidal volume (V_T) (Danon; Druz et al. 1979). Its primary action is lifting the sternum and expanding the upper rib cage during inspiration (De Troyer, Estenne et al. 1986).

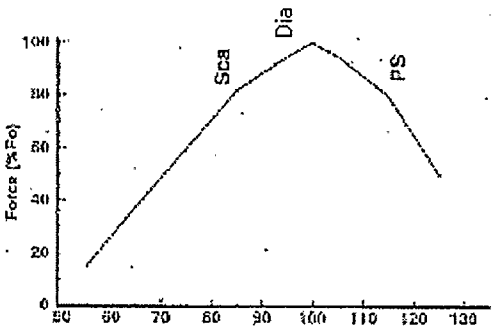
Triangularis Sterni Muscle

The triangularis sterni muscle, a primary expiratory muscle, originates from the lower half of the sternum and inserts on the costal cartilages of ribs 3-7 (Epstein 1994). During forced expiration or high minute ventilation, the action of this muscle is coupled with that of the abdominal muscles and acts to pull ribs caudally, deflating the rib cage (De Troyer, Ninane et al. 1987). This muscle is innervated by T1-6 (Epstein 1994).

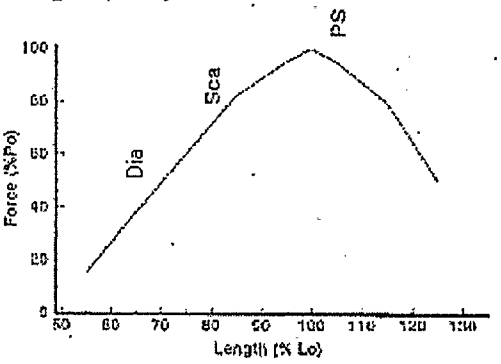
Contractile properties of the RM

The respiratory muscles behave in a similar manner as skeletal muscle with respect to the modulation of their force output. The length-tension relationship in skeletal muscle has been explained in detail elsewhere and is beyond the scope of this thesis but briefly, the force output of these muscles is dependent on their precontractile length or resting length (length-tension relationship) and the velocity of shortening (force velocity relationship) (Farkas, Cerny et al. 1996) Figure 1 (A,B,C). The maximal force generating capacity of the diaphragm occurs at a lung volume that is between FRC and RV when measured with twitch transdiaphragmatic pressure (Pdi,twitch) (Smith and Bellemare 1987). The Pdi,twitch amplitude decreases by about 5% at 40% of TLC or supine FRC, decreases a further 15% at 50% TLC or upright FRC and falls by about 60% from maximal at TLC. In contrast to the diaphragm, the neck inspiratory and parasternal muscles shorten to a length that is closer to their optimal force generating capacity length at high lung volumes (Jiang, Deschepper et al. 1989).

A. Functional Residual Capacity



B. Total Lung Capacity



C.

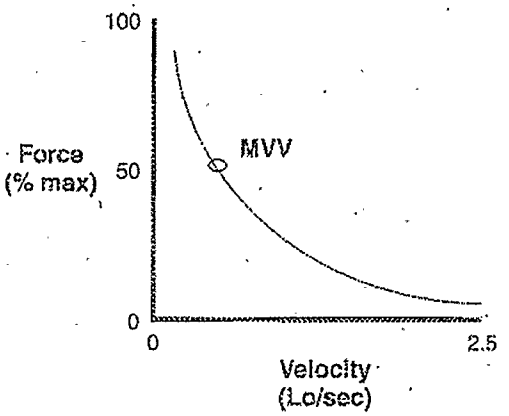


Figure 1: Maximal length-tension curve of the primary respiratory muscles and operating length of muscle at FRC (A) and the effect of increasing lung volume to total lung capacity on operating length (B). Figure 1 (C) represents the idealized relationship between contractile force and velocity of shortening of the human diaphragm. The point identified as "MVV" is an estimate of the shortening velocity during unencumbered MVV. Dia – diaphragm; Sca – scalene; PS – parasternal intercostals. Reprinted from Celli (1998) and Farkas et al (1996).

The measure of maximum static inspiratory pressure (MIP) is greatest at RV, falls off slightly at FRC and declines to zero a TLC (Farkas, Cerny et al. 1996). Since the diaphragm has a larger shortening capacity than the other inspiratory muscles, the relationship between MIP and lung volume results primarily from the force-length properties of the diaphragm (Farkas, Cerny et al. 1996). MEP is maximal at TLC and declines progressively as lung volume is decreased to RV (Farkas, Cerny et al. 1996).

The force velocity relationship of the respiratory muscles is currently an area of uncertainty (Farkas, Cerny et al. 1996). The in situ relationship between trandiaphragmatic pressure (Pdi) and inspiratory flow rates have been described as linear by some and hyperbolic by others (Kikuchi, Sasaki et al. 1982; Topulos, Reid et al. 1987; Tzelepis, Vega et al. 1994). Regardless, overall pressure development by the either the diaphragm or the combination of the respiratory muscles follows some type of inverse relationship with the velocity at which the muscle is shortened (Farkas, Cerny et al. 1996).

Ventilation and exercise

To facilitate the increased ventilatory demands during exercise, there is an increased motor unit recruitment of the diaphragm and accessory inspiratory muscles. This recruitment acts to pull the ribcage upward and forward creating an increase in the lateral and anteroposterior diameters of the thorax during inspiration and the abdominal muscles and triangularis sterni are recruited to depress the rib cage and compress the abdominal contents upwards during expiration (Whipp and Pardy 1986). The pectoralis major and minor, latissimus dorsi, serratus anterior and trapezius muscle may also play

an inspiratory role on the thorax because of their anatomical location (Celli 1998). Although their respiratory role has not been fully studied, according to their mechanical arrangement, if the extrathoracic portion is fixed, their contraction should help expand the rib cage by pulling the superior rib cage outwards and upwards (Celli, Rassulo et al. 1986; Celli 1998). During normal resting breathing, the diaphragm accounts for approximately 50% of the inspiratory volume change with the rib cage muscles (primarily the parasternal muscle) providing the rest (Sheel 2002). Exercise markedly increases diaphragm recruitment but measurements of Pdi and surface electromyogram (EMG) have shown that its contribution to total respiratory muscle pressure decreases as exercise progresses. This is evident by a plateau in diaphragm activity at around 400% of resting values while the total respiratory muscle pressure output is increased by over 500-700% of resting values (Bye, Esau et al. 1984; Johnson, Aaron et al. 1996).

Fiber Types

Similar to skeletal muscle, the RM's also differ in their fiber composition. The diaphragm and intercostals have a higher composition of slow twitch fibers (55% and 65% respectively) than fast twitch fibers (45% and 35% respectively) while the scalenes muscles have a higher composition of fast twitch than slow twitch fibers (65% fast twitch and 35% slow twitch) (Sharp and Hyatt 1986). The increased fast twitch percentage in the scalenes enables a faster contraction time than the diaphragm and may play an important role in achieving the higher ventilation rates observed during exercise (Epstein 1994). With regards to the expiratory muscles, the rectus abdominus is almost equally

composed of fast twitch and slow twitch fibers (46% slow twitch and 54% fast twitch) (Hards, Reid et al. 1990).

Breathing Pattern during Exercise

The breathing pattern exhibited during exercise is a consequence of a highly coordinated effort between the respiratory muscles. During resting breathing, the mean ventilation rate (V_E) is between 6-8L/min and during intensive exercise in the highly trained individual can exceed 200 L/min (Whipp and Pardy 1986; Sheel 2002). The increase in V_E during exercise is caused by increases in f and V_T. At lower V_E, increases in both V_T and f are responsible for the rise in V_E. Increases in V_T are achieved by decreasing the end-expiratory volume and increasing the end inspiratory volume while increases in f is achieved by decreasing both inspiratory (T_I) and expiratory time (T_E) although there is a greater decrease in T_E than in T_I (Sheel 2002). The increase in V_T will generally plateau at about 50% of the individuals vital capacity (even in highly trained individuals) and is followed by a continued increase in f to facilitate additional increases in V_E. This latter increase in V_E is achieved by further decreasing both inspiratory (T_I) and expiratory time (T_E) (Whipp and Pardy 1986). f will increase by 1-3 times the resting value and can reach values in excess of 50 breaths per minute (bpm) during high intensity exercise (Spengler, Roos et al. 1999). At lower V_E, such as those observed at the beginning of exercise, the flow resistance of the respiratory system is low, and the output of the system is limited by the contraction of the respiratory muscles, therefore, it is more efficient to increase V_T than increase f (Bartlett, Brubach et al. 1958b). At higher V_E, such as those observed near maximal exercise, the impedance of the respiratory system

increases and the respiratory system is mostly limited by the force of the contraction (Bartlett, Brubach et al. 1958b). It is likely that an increase in *f* is the most efficient method (lowest respiratory work rate) of making subsequent increases in V_E (Mallios and Hodgson 1994).

Although it is clear that V_E does increase with exercise, the mode of exercise has shown to affect the type of breathing pattern developed. Termed entrainment, Mahler et al (1991) reported that elite rowers favored a 1:1 (breath every stroke) or 1:2 (2 breaths every stroke) breathing pattern which developed through training (Mahler, Hunter et al. 1991; Mahler, Shuhart et al. 1991). Similarly, Paterson et al (1986) reported that *f* showed a clear, but intermittent tendency to entrain with limb frequency during moderate intensity cycling (Paterson, Wood et al. 1986). It has been suggested that the V_E pattern exhibited during exercise develops as a result of locomotion rather than the converse and may require only a few months to develop depending on the individual (Hill, Adams et al. 1988; Mahler, Hunter et al. 1991). The reason for this is because the locomotion pattern is more consistent than the breathing pattern. The advantage of entraining the breathing pattern to exercise is a possible decrease in the overall oxygen requirement (Garlando, Kohl et al. 1985).

Assessment of Respiratory Muscle Strength and Endurance

RM strength and endurance measures are important for determining the effectiveness of a RMT program but unlike the limb muscles, the RM's are difficult to evaluate directly because of their inaccessibility (Clanton and Diaz 1995). Nevertheless, a variety of different methods are commonly used to assess respiratory muscle strength

and endurance in both the clinical and research setting. These measures include: maximum static inspiratory and expiratory pressures measured at the mouth (MIP and MEP respectively), sniff esophageal pressure (sniff Pes), maximal transdiaphragmatic pressure (Pdimax), constant load resistive breathing (CLRB), incremental load resistive breathing (ILRB) and maximum sustainable ventilatory capacity (MVSC) (Laporta and Grassino 1985; Clanton and Diaz 1995; Johnson, Aaron et al. 1996; Perret, Pfeiffer et al. 1999; Inbar, Weiner et al. 2000). These methods will now be individually described.

Maximal Inspiratory Pressure (MIP)

MIP is the maximum inspiratory pressure that is produced at the mouth and is measured as a peak value or as a one second average value that is averaged between the 3 highest values. A highly significant correlation between MIP peak and MIP 1-sec average has been reported ($r > 0.97$, $P<0.0001$) (McConnell and Copestake 1999). Since there is little or no airflow during the effort, it is assumed that the change in pressure in the mouthpiece is equal to the change in pressure at the alveoli which is equal to change in pleural pressure (Pacia and Aldrich 1998). Although the method of measuring MIP varies slightly between studies, basically it requires the subject to perform a maximal inspiratory effort against an occluded mouthpiece (Muller maneuver)(Figure 2). The mouthpiece has a small air leak to prevent the facial muscles from contributing to the pressure development and is attached to a manometer (Koulouris, Mulvey et al. 1988; Rochester 1988; Reid and Dechman 1995).

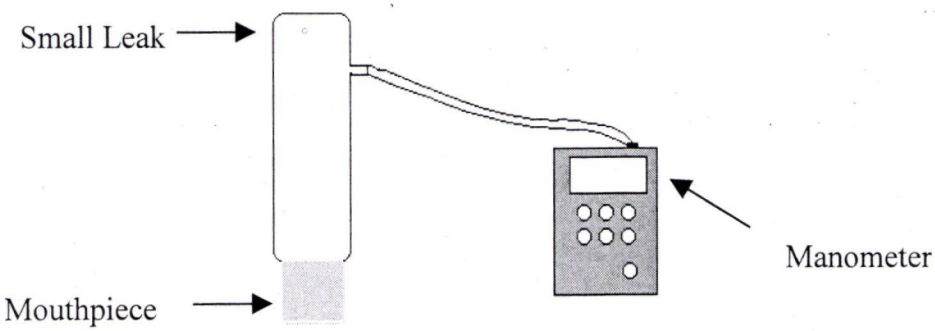


Figure 2: A commonly used setup for measuring maximum inspiratory and expiratory pressures. A small leak is provided to ensure the patient does not generate the pressure with the muscles attached to the buccal cavity

Measurements are usually made from around RV, where the diaphragm is at an optimal position on its length tension curve, with the subject in the sitting position (Reid and Dechman 1995; Farkas, Cerny et al. 1996). It is important to clarify that MIP (Muller) provides an estimate of the maximal inspiratory pressure produced at the mouth from the combined activation of all the respiratory muscles. This does not mean that all the muscles are activated maximally. It has been suggested that the maximal inspiratory pressure is limited by the weaker muscle group to prevent unacceptable distortions of the chest wall (Hershenson, Kikuchi et al. 1988). Therefore, during a MIP measurement the stronger diaphragm is expected to be submaximally activated while the weaker rib cages muscle are expected to be maximally activated. This is supported by EMG activity and estimation of pleural and abdominal pressure although this literature has been questioned (Hershenson, Kikuchi et al. 1988; McKenzie, Plassman et al. 1988; Gandevia, McKenzie et al. 1990; Nava, Ambrosino et al. 1993).

Reported normal values for MIP in athletes range from 104-171cmH20 and in normal individuals range from 127-138cmH20 (Table 1). MIP has been positively correlated with standing height, body weight and physical activity (Harik-Khan, Wise et al. 1998; Carpenter, Tockman et al. 1999). The day-to-day reliability of MIP measurements has not been well evaluated. Previous studies have measured MIP using between 3 – 20 consecutive measurements (Wen, Woo et al. 1997; Maillard, Burdet et al. 1998; McConnell and Copestake 1999; Volianitis, McConnell et al. 1999). Using 18-20 measurements for MIP tend to elicit values that are between 7-12% higher than using 3 measurements for MIP (Fiz, Montserrat et al. 1989; Wen, Woo et al. 1997). Reasons for the varying results reported between studies may be attributed to subject motivation and their familiarization with the maneuver. Since the MIP maneuver is a maximal effort, subjects not familiar with maximal exertion or have poor motivation will underestimate their true values (Decramer and Macklem 1995; Pacia and Aldrich 1998; Sheel 2002). This maneuver also requires coordination of the RM's in a manner than many individuals may be unfamiliar with (Laroche, Mier et al. 1988). Although MIP provides a relatively simple and economical method to measure overall RM strength, it may underestimate the actual value when fewer than 18 maneuvers are used. This may be problematic in the elderly or diseased population where motivation and fatigue may prevent the measure of an individual's true capacity and in the research setting where small changes in MIP is expected (Miller, Moxham et al. 1985; Laroche, Mier et al. 1988).

Table 1: Normal values for various measures of respiratory muscle strength

Technique	Lung Volume @ measurement	Subject Characteristics	Value Mean cmH_20 (SD)	Reference
MIP				
MIP	RV	Healthy adults	127 (8)	(Hart, Sylvester et al. 2001)
MIP	RV	Club rowers	171 (9)	(Volianitis, McConnell et al. 1999)
MIP	RV	Endurance athletes	142 (24)	(Inbar, Weiner et al. 2000)
MIP	RV	Moderate trained cyclists	168 (40)	(Sonetti, Wetter et al. 2001)
MIP	RV	Trained cyclists	104 (8)	(Romer, McConnell et al. 2002)
MIP	RV	Female rowers	104-130 (12)	(Volianitis, McConnell et al. 2001)
MIP	RV	Healthy Adults	138 (24)	(Volianitis, McConnell et al. 2001)
MEP				
MEP	TLC	Aerobic athletes	165 (30)	(Amonette and Dupler 2001)
MEP	FRC	Healthy adults	161 (37)	(Rubinstein, Slutsky et al. 1988)
MEP	TLC	Normal males	233 (84)	Black and Hyatt
MEP	TLC	Normal males	216(44)	(Rochester and Arora 1983)
Pdimax				
Pdi (Sniff)	FRC	Normal males	148 (24)	(Miller, Moxham et al. 1985)
Pdi (sniff)	FRC	Normal male	114(32)	(Chan, Cheong et al. 1996)
Pdi (combined)	FRC	9 normal males, 1normal female	145 (37)	(Laporta and Grassino 1985)
Pdi (feedback)	FRC	9 normal males, 1normal female	180 (14)	(Laporta and Grassino 1985)
Pdi (Muller)	RV	Normal male	97(34)	(Chan, Cheong et al. 1996)

Pdi (Muller)	RV	Normal males	108 (30)	(Miller, Moxham et al. 1985)
Pdi (Muller)	RV	Normal males	190 (26)	(Bye, Esau et al. 1984)
Pdi (Muller)	FRC	9 normal males, 1normal female	150(33)	(Laporta and Grassino 1985)

RV: residual volume, FRC: functional residual capacity, TLC: total lung capacity, MIP: maximum inspiratory pressure, Pdi: transdiaphragmatic pressure

Maximum Expiratory Pressure (MEP)

MEP is a simple non-invasive method of assessing global expiratory muscle strength (Rubinstein, Slutsky et al. 1988). Normal values for MEP range between 161-233cmH20 (Table 1). Briefly, subjects are instructed to inspire to TLC before forcefully exhaling through an occluded mouthpiece (similar in design to the one used for MIP) that is connected to a manometer (Figure 2). Similar to MIP, measurements are taken as a peak or 1sec value and generally averaged over the three highest measurements (Clanton and Diaz 1995). The highest measurements of MEP are obtained at lung volumes greater than 70% of TLC because the major expiratory muscles are at their optimal force generating length (Black and Hyatt 1968; Rochester 1988). Since few studies have investigated the MEP maneuver, little information is available on its intrasubject variability. However, it is known that the type of mouthpiece will significantly affect the measurement. Rubinstein et al (1988) reported that a large circular or scuba diving mouthpiece with the cheeks supported by the hands resulted in higher values of MEP than the no hand method. From experience in our lab, subjects can find it difficult to exhale forcefully from TLC while maintaining a tight seal around the mouthpiece, even when the hands support the cheeks. MEP shares many of the same limitations as MIP

such as being volitional in nature and requiring a learning period. Currently MEP is the primary method used to measure global expiratory muscle strength.

Maximal Transdiaphragmatic Pressure (Pdimax)

The diaphragm is the major inspiratory muscle, and thus the assessment of its function is important in both the research and the clinical setting. Duomarco and Rimini (1947) were the first to theorize on the pressure changes in the abdomen and pleura that would occur during inspiration (Macklem 1998). They hypothesized that during inspiration, the chest wall would expand and the pleural pressure (Ppl) would always decrease, while the change in abdominal pressure (Pab) depended on diaphragm and abdominal muscle activity. With diaphragm contraction, abdominal pressure would increase and the abdominal wall would be displaced outward, but abdominal muscle relaxation and non-diaphragmatic inspiratory muscle contraction would lead to a decrease in abdominal pressure and an inward displacement of the abdominal wall. Agostoni and Rahn (1960) were the first to measure transdiaphragmatic pressure and quantify the diaphragm's contribution to respiratory pressure swings by using balloon catheters inserted transnassaly into the esophagus and stomach (Figure 3). The pressure in the esophageal balloon (Pes) provides an estimate of Ppl as long as the pressure difference across all intervening structures (balloon wall, esophageal wall and mediastinal structures) is zero (Milic-Emili, Mead et al. 1964). In order to minimize this pressure difference, a volume of about 0.2-0.4ml of air should be inserted into the balloon, although this has been shown to vary with balloon characteristics (wall thickness and cross sectional area) (Milic-Emili, Mead et al. 1964; Lemen, Benson et al. 1974). Ideally,

the amount of air that should be inserted into the balloon should be on the lower linear portion of its pressure volume curve (Figure 4).

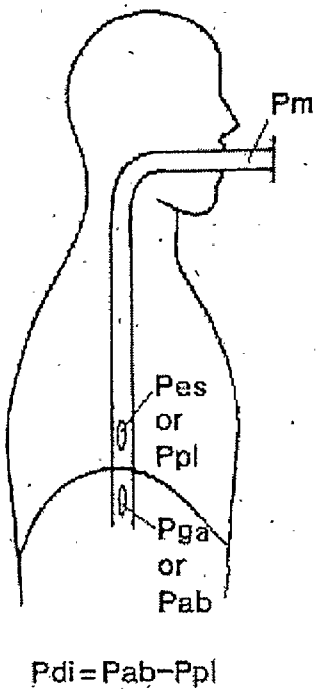


Figure 3: Schematic representation of mouth pressure (Pm), esophageal pressure (Pes) or pleural pressure (Ppl) and gastric pressure (Pga) or abdominal pressure (Pab). Trandiaphragmatic pressure is the difference between Pg and Pes. Reprinted from Reid and Dechman, 1995.

If the balloon is filled with too much air, the Ppl will be overestimated because of the elastic recoil of the balloon and distension of the esophagus and surrounding structures (Mead, McIlroy et al. 1955; Milic-Emili, Mead et al. 1964; Milic-Emili 1984). Similarly, if the balloon is not filled adequately, it will not be on the linear part of its pressure volume curve because of balloon recoil and pleural pressure will be underestimated (figure 4) (Milic-Emili, Mead et al. 1964; Milic-Emili 1984). Abdominal pressure is estimated from the pressure in the gastric balloon (Pga). Distension of the

stomach is not an issue as in the esophagus but rather it is important that the gastric balloon doesn't empty when gastric pressure becomes highly positive such as during forced expirations (Macklem 2002). Therefore enough air should be inserted into the balloon to prevent emptying (~ 200cmH_20 in athletes) while remaining on the flat part of its pressure volume curve. The pressure volume curves for the esophageal balloon should be determined in water as apposed to air because its characteristics in water closely parallel its characteristics in the esophagus particularly when large volumes are required (Lemen, Benson et al. 1974). The stomach is also considered a liquid container, and thus its pressure volume curve should also be determined in water (Agostoni and Mead 1964; Milic-Emili 1984).

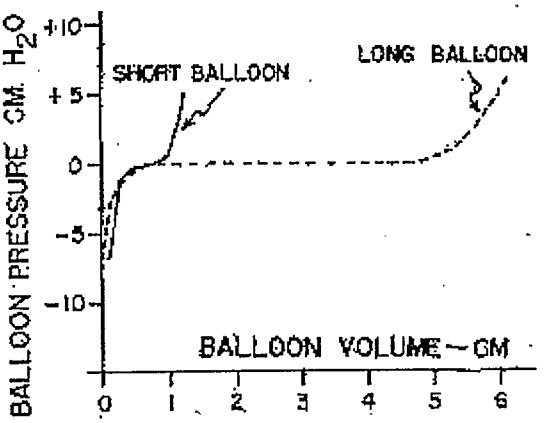


Figure 4: Plot of balloon pressure plotted against balloon volume for short and long esophageal balloons.

Maximal diaphragm pressure (Pdimax) is an estimate of the maximal force produced by the diaphragm (Laporta and Grassino 1985). There are a variety of maneuvers that are used to measure Pdimax. The 3 most common are a sniff maneuver (short sharp maximal sniff with the nostril unoccluded), a Muller maneuver (similar to the MIP maneuver) and a combined Muller and expulsive maneuver (forceful contraction

of the abdominal muscles and a simultaneous maximum inspiratory effort with feedback) (Laporta and Grassino 1985; Miller, Moxham et al. 1985). The combined Muller maneuver provides a more accurate estimate of the maximum force generating capacity of the diaphragm compared to the Muller or sniff maneuver as shown by a considerably higher pressure measurement of Pdi (Laporta and Grassino 1985; Hershenson, Kikuchi et al. 1988; Nava, Ambrosino et al. 1993). With a combined maneuver, the diaphragm will first match the pressure exerted by the rib cage muscles and subsequently shorten with any additional force. To maintain the length of the diaphragm at FRC, the abdominal muscles must contract and increase Pab. Again, this will require the diaphragm to increase its activation to prevent it from being pushed into the thorax (Laporta and Grassino 1985). In the end, an estimate of the actual pressure that the diaphragm can hold is provided (Laporta and Grassino 1985). The primary limitation of this maneuver is the difficulty of coordinating it in naïve subjects (Chan, Cheong et al. 1996).

In contrast to the combined maneuver, the Muller maneuver and the sniff maneuver will elicit submaximal measurements of Pdi because the weaker rib cage muscles limit diaphragm activation (Laporta and Grassino 1985; Hershenson, Kikuchi et al. 1988). Furthermore, since the sniff maneuver is dynamic in nature, the force generating capacity of the diaphragm will be further reduced (Miller, Moxham et al. 1985). Rather than providing a measure of the maximal force generating capacity of the diaphragm, the sniff and Muller maneuvers provide an estimate of the pressure across the diaphragm during a maximal inspiratory maneuver. Also, the sniff and Muller maneuver are influenced by the amount of abdominal contraction. Depending on how the

maneuver is performed, substantial variability in Pga and Pdi may be observed (Laporta and Grassino 1985; Miller, Moxham et al. 1985; Verin, Delafosse et al. 2001).

Of the three maneuvers, the sniff is the easiest to perform, requires the least learning and has low intra subject variability (coefficient of variation (CV) = 7.2%). It may be expected that the Muller maneuver would elicit higher Pdimax values than the sniff maneuver. This is because the former is a quasi-static maneuver as compared to the latter being dynamic maneuver where there may be a presumed loss of force due to diaphragm shortening (Miller, Moxham et al. 1985; Chan, Cheong et al. 1996). However, the opposite is generally true. This may occur because there is greater diaphragm activation during the sniff maneuver than during the Muller maneuver and/or because there is an antagonistic action of other respiratory muscle groups during the Mueller maneuver that is not found during the sniff maneuver (Miller, Moxham et al. 1985). The sniff is also a more familiar maneuver that does not involve the use of a nose clip and is repeatable without tiring (Miller, Moxham et al. 1985). The sniff maneuver seems to be the most practical method to assess the pressure across the diaphragm during a maximal inspiratory maneuver in naïve subjects.

Sniff esophageal pressure (sniffPes)

Although MIP is a relatively simple non-invasive method of determining global inspiratory muscle strength, it is both demanding and difficult to perform in certain individuals (Laroche, Mier et al. 1988). Therefore, low MIP values may represent a lack of motivation or coordination rather than muscle weakness (Heritier, Rahm et al. 1994). An alternative method used to measure maximal inspiratory pressure is sniff Pes

(Laroche, Mier et al. 1988). This maneuver is more natural and probably easier to perform than the MIP maneuver and helps discriminate patients that have a low MIP caused by poor technique or lack of motivation from those with true inspiratory muscle weakness (Laroche, Mier et al. 1988). Furthermore, MIP can underestimate alveolar pressure (Palv) because of tissue compliance in the upper airway or in patients with severe airway obstruction where there is often a substantial lag of transmission of Palv to the mouth (Jaeger 1982; Murciano, Aubier et al. 1982; Milic-Emili 1984). The within day and between day coefficient of variation (CV) for Sniff Ppl is 5.2% and 4.1% respectively (Koulouris, Mulvey et al. 1989). Unlike sniff Pdi, sniff Pes is not affected by the type of maneuver performed whether it be mainly diaphragm or mainly ribcage (Verin, Delafosse et al. 2001).

Constant load resistive breathing (CLRB)/ Increment threshold loading (ITL)

CLRB and ITL are two methods used to assess RM endurance. The CLRB method requires the subject to breath against a set resistance until task failure while ITL requires the subject to breath against a resistance that is increased every 2 minutes until task failure. The outcome measure used for CLRB is the time the resistance can be sustained while the outcome measure for ITL is the maximum resistance that can be sustained for 2 min. The advantage of the ITL method is that breathing pattern has a smaller effect on the intrasubject reproducibility of this measure as compared to CLRB (CV of 20-65% for CLRB vs. CV of 0-14% for ITL) (McElvaney, Fairbarn et al. 1989). However if f is controlled, both methods produce results that do not differ significantly (Fiz, Romero et al. 1998). Perret and Pfieffer et al (1999) reported the CLRB method to

be the most sensitive non-invasive measure of respiratory muscle performance although others have questioned the reproducibility of this measure. Both methods provide an estimate of RM endurance when the breathing pattern is controlled. The advantage provided by the CLRB method is not having to pause every 2 minutes to increase the load making it easier to use with commercially available pressure resistance RMT devices.

Maximum Sustainable Ventilatory Capacity (MSVC)

This breathing endurance test is an extension of the 15-second MVV test. Although specific methods vary between studies, this test measures the time a percentage of MVV can be sustained. This percentage generally ranges between 65% - 90% of the 15 second MVV (Spengler, Laube et al. 1996; Markov, Spengler et al. 2001; Sonetti, Wetter et al. 2001; Stuessi, Spengler et al. 2001). The greatest limitation of this test is that it is difficult to determine whether the change in MVSC is due to changes in ventilatory impedance or to changes in muscle function (Clanton and Diaz 1995). Additionally the effect of learning or familiarization may have a significant influence on the variability of this test as with other volitional respiratory muscle tests (Eastwood, Hillman et al. 1998; Sonetti, Wetter et al. 2001).

Evidence against the respiratory muscles limiting aerobic performance

There are several reasons why the respiratory system may not be a limiting factor for aerobic performance (Leith and Bradley 1976; Fairbarn, Coutts et al. 1991). First, indirect evidence is obtained from observations that maximum ventilation (V_{Emax}) during exercise never meets the V_E achieved during 15-second MVV (Boutellier 1998).

Second, even sedentary subjects are able to increase their V_E when they near the end of an incremental exercise test suggesting that the respiratory muscles are not fatigued (Boutellier 1998). Lastly, a number of studies have indicated that the oxygen saturation of the blood is rarely compromised during maximal exercise in healthy humans (Dempsey 1986; Morgan, Kohrt et al. 1987; Inbar, Weiner et al. 2000). However, all these reason have shortcomings. First, MVV testing usually lasts only 12-15 seconds in duration and the V_E achieved in this short period is not representative of the V_E attainable for longer durations (Boutellier 1998). Second, hyperventilation during high intensity exercise only lasts for a short duration. It is possible that the recruitment of higher force producing, anaerobically dominant fast twitch fibers are responsible for this short-lived increased force production. This is analogous to an individual's ability to perform a final sprint well above their usual pace at the end of a marathon. Lastly, oxygen saturation is only one determinant of aerobic performance. Even though V_E appears to be high enough at maximal workloads to maintain oxygen saturation of the blood, it is possible that V_E may limit performance in ways not related oxygen saturation (Wasserman, Van Kessel et al. 1967; Bassett and Howley 1997).

Support for the respiratory system limiting aerobic performance

The respiratory system may play a limiting role on athletic performance because of evidence suggesting RM fatigue following exhaustive exercise and its possible influence on blood flow distribution (Boutellier, Buchel et al. 1992; Boutellier and Piwko 1992; Johnson, Aaron et al. 1996; Boutellier 1998).

RM Fatigue

The term fatigue has several meanings.. For the context of this paper, fatigue refers to a condition in which there is a reduction in the force-generating capacity of the muscle resulting from muscle activity under load that is reversible by rest (American Thoracic Society1990). Fatigue of the RM's can occur at any one of the many steps involved in voluntary muscle activity and has been separated into central and peripheral components (Roussos and Macklem 1986). Peripheral fatigue refers to a reduced force generating ability at the periphery and can involve impairments in neuromuscular transmission and its propagation down the sarcolemma, dysfunction within the sarcoplasmic reticulum involving calcium release and uptake, availability of metabolic substrates and accumulation of metabolites and actin-myosin crossbridge interaction (Roussos and Macklem 1986; Coggan and Coyle 1991; Enoka and Stuart 1992; Balog, Thompson et al. 1994; Davis and Bailey 1997; Powers and Howley 2001). Central fatigue refers to a reduced central motor output and can include a reduction in the number of motor units involved in the activity or reduction in motor unit firing frequency as well as the influence of psychological factors such a motivation and perception (Ikai and Yabe 1969; Asmussen and Mazin 1978; Asmussen and Mazin 1978; Davis and Bailey 1997). An excellent review on respiratory muscle fatigue is available by McKenzie and Bellemare (1995) .

Measures of RM fatigue

The various methods used to estimate RM strength (discussed earlier) have also been used to estimate RM fatigue. These methods include MIP, MEP and Pdimax. Although these methods are relatively simple to perform, the maneuvers are volitional in nature, the test conditions themselves are difficult to control and are not sufficiently objective or independent of total body fatigue and the significant role of motivation on pressure development (Johnson, Babcock et al. 1993). Two other methods used to estimate RM fatigue are EMG spectral shift and bilateral phrenic nerve stimulation (BPNS). With an EMG spectral shift, analysis of the EMG in its frequency domain delineates its power spectrum (Roussos and Macklem 1986). Fatigue is identified as a shift of the spectrum to lower frequencies which occurs before there is failure to develop adequate force and is quantified either as a reduction in centroid (mean) frequency or as a reduction in the ratio of power at high frequencies to power at lower frequencies (H/L ratio) (Gross, Grassino et al. 1979; Roussos and Macklem 1986; Pacia and Aldrich 1998). Limitations of this technique include interference from other muscles, location of electrode placement and substantial cardiac artifact when using esophageal electrodes (Yan, Lichros et al. 1993; Pacia and Aldrich 1998). Sieck and Fournier (1990) also challenged the validity of EMG as an index of fatigue because changes in EMG did not necessarily reflect the extent of diaphragm fatigue. BPNS involves a stimulation of the phrenic nerve bilaterally behind the sternomastoid muscles in the neck and the response is measured as pressure changes in the esophageal and gastric balloons (Johnson, Aaron et al. 1996; Pacia and Aldrich 1998). This method appears to provide the most objective

estimate of diaphragm fatigue because of its effort independence. However, the reliability of this measure depends on muscle length, abdominal compliance, quasi isometric condition and supramaximal stimulation (Johnson, Babcock et al. 1993; Mador, Rodis et al. 1996). Also, this measurement is specifically for the diaphragm and ignores extra-diaphragmatic fatigue. This may be an important limitation when measuring RM fatigue since it is known that the accessory muscles make a substantial contribution to respiration during exercise (Johnson, Aaron et al. 1996).

The effect of exercise on RM fatigue

Numerous studies have used measures of MIP, Pdimax and shifts in the frequency spectrum of the diaphragm integrated EMG activity to investigate the effects of exercise on respiratory muscle fatigue. These studies are summarized in Table 2.

Table 2: Effects of exercise on measures of respiratory muscle fatigue

Method	**Exercise type & intensity**	**Subject training**	**% Change in measure**	**Reference**
MIP	42.2 km marathon	Healthy male	-16%*	(Loke, Mahler et al. 1982)
MIP	Max incremental test	Untrained males	-10%*	(Coast, Clifford et al. 1990)
MIP	Max incremental test	Highly trained XC skiers	0%	(Coast, Clifford et al. 1990)
MIP	Multistage shuttle run	Moderately trained	-10.5%*	(McConnell, Caine et al. 1997)
MIP	17km run	6 well trained	1.9%	(Nava, Zanotti et al. 1992)
MIP	85% of VO_2max to exhaustion	Healthy	1.1%	(Perret, Pfeiffer et al. 1999)
MIP	20km TT	Well Trained cyclists	-18%*	(Romer, McConnell et al. 2002)
MIP	40km TT	Well Trained	-13%*	(Romer,

		cyclists		McConnell et al. 2002)
MIP	Max incremental tests	Trained rowers	-7%*	(Volianitis, McConnell et al. 1999)
Pdimax (Muller)	80% of MAP	Normal males	-12%*	(Bye, Esau et al. 1984)
Pdimax (Muller)	95% of MAP	Variable training (low-high)	0%	(Johnson, Babcock et al. 1993)
Pdimax (combined)	95% of MAP	Variable training (low-high)	-12%*	(Johnson, Babcock et al. 1993)
Pdimax (combined)	85% of MAP	Variable training (low-high)	-10%*	(Johnson, Babcock et al. 1993)
Pdimax (combined)	85% of MAP	Variable training (low-high)	-11%*	(Johnson, Babcock et al. 1993)
Pdimax,twitch	95% of MAP	Variable training (low-high)	-13%*	(Johnson, Babcock et al. 1993)
Pdimax,twitch	85% of MAP	Variable training (low-high)	-18%*	(Johnson, Babcock et al. 1993)
Pdimax,twitch	70-75% of MAP	Healthy subjects	-19%* in 9 of 14 subjects. 5 did not change	(Mador and Dahuja 1996)
Pdimax,twitch	80% of MAP	Healthy subjects	-17%*	(Mador, Magalang et al. 1993)
Pdimax,twitch	65%-75% of MAP	Elderly sedentary	0%	(Jeffery Mador, Kufel et al. 2000)

* $p<0.05$

The effects of exhaustive exercise on RM fatigue remain inconclusive regardless of the method that is used to estimate it. It is possible that stronger RM's are less susceptible to fatigue following exhaustive exercise as suggested by Coast et al (1990) and McConnel et al (1997). Therefore, studies that used participants with weaker RM's may have observed greater RM fatigue than studies using participants with stronger

RM's. Motivation of the subjects may be another determinant of RM fatigue. Fatigue of the RM is related to an increased sensation of dyspnea (American Thoracic Society1999). Subjects that are able to withstand increasing levels of dyspnea for a longer period of time are more likely to fatigue their RM's (Johnson, Aaron et al. 1996). The method used to measure fatigue may also play a role in identifying RM fatigue. Most of the methods used to estimate RM fatigue are volitional in nature and require the hindrance of a mouthpiece and nose clip. Possible decrements in these measures may be motivational in nature rather that actual RM fatigue (Decramer and Macklem 1995; Sheel 2002). As discussed earlier, the only objective measure of RM fatigue that has been used is bilateral phrenic nerve stimulation. Only 3 of the 5 studies showed decrements in Pdimaxtwitch while the other 2 studies did not. Additionally, these measurements did not account for extra-diaphragmatic fatigue. RM fatigue likely exists in motivated subjects following exhaustive exercise, but more studies need to be performed using objective methods to estimate RM fatigue before any generalization can be made.

Another method used to investigate the relationship between RM fatigue and performance is the voluntary induction of RM fatigue and observing its effect subsequent performance. Mador et al (1991) reported a significant decrease in the time to exhaustion when cycling at 90% of VO_2max following breathing through a pressure resistance equivalent to 80% of MIP until task failure. This result suggests that the RM's can limit exercise performance when they are voluntarily fatigued.

RM fatigue and Breathing Pattern

Several studies have shown that the induction of RM fatigue (breathing through a pressure resistance equivalent to 60-80% of MIP until task failure) in normal subjects alters breathing pattern at rest and during maximal intensity exercise by increasing *f* and either maintaining or decreasing V_T (Gallagher, Hof et al. 1985; Mador and Acevedo 1991; Yan, Sliwinski et al. 1993; Sliwinski, Yan et al. 1996). The degree of tachypnea elicited by inspiratory muscle fatigue tended to correlate with the magnitude of fall in MIP after fatigue (Mador and Acevedo 1991). Sliwinski et al (1996) attributed this altered breathing pattern to a substantially different pattern of respiratory muscle recruitment. They reported a significant increase in the tonic and phasic activities of the abdominal muscles that acted to increase end expiratory lung volume and lengthen the diaphragm putting it on a more optimal position of its length tension curve. This allowed the maintenance of the same V_T with less shortening of the inspiratory rib cage muscles and an increase in *f* by decreasing the expiratory time (Sliwinski, Yan et al. 1996).

Respiratory Muscle Efficiency and Blood Flow Demand

Recent research by Harms et al (1997, 1998, 1999, 2000a, 2000b) suggests that the limiting role of the RM's in aerobic performance may be a consequence of blood flow distribution during exercise and is likely related to RM fatigue. Loading of the RM's at maximal exercise while working at a constant power output results in a significant reduction in leg blood flow and oxygen uptake ($\dot{V}O_2$) and a parallel increase in leg vascular resistance. Conversely, unloading of the RM's results in a significant increase

in leg blood flow and VO_2, a parallel decrease in leg vascular resistance and a greater work rate for a given VO_2 (Harms, Babcock et al. 1997; Harms, Wetter et al. 1998; Harms and Dempsey 1999; Harms 2000; Harms, Wetter et al. 2000). These results demonstrate the importance of the blood flow interaction between the RM's and the peripheral muscles during maximal intensity exercise. Since cardiac output reaches a maximal value at high intensity workloads, both the locomotor muscles and the RM's compete for a portion of the cardiac output. If the RM's receive a preferential share of the cardiac output during high intensity workloads, blood flow and oxygen delivery to the locomotor muscles will decline (Dempsey, Harms et al. 1996). Shock in animals has shown a reduction in blood flow to all parts of the body except the RM's where an increase of up to 17% of the total cardiac output was observed suggesting preferential blood flow to the RM (Viires, Sillye et al. 1983; Hussain and Roussos 1985). Decreased cardiac output to the periphery could lead to increased anaerobic metabolism that disrupts the lactate and pH homeostasis in the blood and contributes to peripheral muscle fatigue (Viires, Sillye et al. 1983; Hussain and Roussos 1985; Johnson, Aaron et al. 1996). Thus, the effects the RM's have on limiting athletic performance may be strictly due to the shunting of blood away from the locomotor muscles to the RM's. This shunting of blood away from the locomotor muscles may also be augmented by RM fatigue. Recent studies have demonstrated that both diaphragmatic and expiratory muscle fatigue associated with prolonged heavy exercise or loaded breathing (inspiratory or expiratory resistance) to task failure resulted in reflexively precipitated sympathetically-mediated vasoconstriction as measured by muscle sympathetic nerve activity (Harms, Babcock et al. 1997; Sheel, Derchak et al. 2001; Dempsey, Sheel et al. 2002; Derchak, Sheel et al. 2002). This

vasoconstriction is thought to be due to a metaboreflex and can result in decreased blood flow to the locomotor muscles during exercise (Harms, Babcock et al. 1997; Sheel, Derchak et al. 2001).

The above reasoning may explain the improved performance observed following RM unloading. Aaron et al (1985) and Harms et al (2000) reported an increased exercise duration time when tested at intensities greater 90-95% of VO_{2max} following unloading of the respiratory muscles with a pressure assist device or a helium/oxygen mixture (Aaron, Seow et al. 1992; Harms, Wetter et al. 2000). However, unloading of the RM's at lower intensities did not improve performance times suggesting that the intensity may have been too low to limit blood flow to the locomotor muscles (Krishnan, Zintel et al. 1996).

Methods of RMT

Three methods have been used to train the RM's: isocapnic hyperpnoea, flow resistive breathing and pressure resistive breathing. These methods are described below.

Isocapnic Hyperpnoea (IH)

Isocapnic hyperpnoea is voluntary breathing at a predetermined target V_E (Powers, Coombes et al. 1997). Subjects breathe through a 2-way valve and measurements of V_E, f, and V_T are recorded. Carbon dioxide (CO_2) is added at a rate required to maintain the fractional concentration of CO_2 in the expired air (Fairbarn, Coutts et al. 1991; Boutellier, Buchel et al. 1992; Boutellier and Piwko 1992). As the ability to maintain the target V_E becomes easier, the overload is increased by increasing the f or V_T. Although this type of training is similar to that observed during exercise and

thus has high construct validity, it requires an elaborate and costly setup. Consequently, it is not practical for portable use (Gosselink and Decramer 1994).

Flow resistive training device (FRD)

Flow resistive training devices consist of either a unidirectional or bi-directional valve on which resistance caps of different sized holes can be attached (Hanel and Secher 1991). The principal behind this type of device is that orifices with smaller diameters will produce a greater resistance to breathing than orifices with larger diameters. This is analogous to breathing through different sized straws. A smaller diameter straw increases the resistance to air flow and makes it more difficult to breathe. Although this type of device is simple, inexpensive and portable, the airflow rate has a major effect on the magnitude of the resistance produced (Gosselink and Decramer 1994). With a given orifice size, resistance can vary from almost a negligible amount when airflow is minimal to a considerable amount when airflow is maximal. Thus appropriate training intensity is only achieved if an adequate target pressure is obtained (Belman and Shadmehr 1988). The reason this type of device has not gained much popularity in the research setting is likely because of the difficulty in controlling airflow rates in human subjects.

Pressure resistance training device (PRD)

Pressure resistance training devices consist of a spring-loaded valve that requires a minimum inspired or expired mouth pressure to enable airflow. Increasing or decreasing the tension in the spring alters the work of breathing by adjusting the minimum pressure required to enable airflow. The advantages of this device is that it is

portable, relatively flow rate independent, and inexpensive (Gosselink and Decramer 1994). The disadvantage of this device is that the *f* and V_T adopted during training can compromise the training adaptations when they are not controlled. In spite of this limitation, this device appears to be the most practical for field use.

Muscular Adaptations to Training

Muscular adaptations in skeletal muscle

Skeletal muscle is remarkable in its ability to adapt to strength and endurance training. As one may expect, specific strength and endurance training regimes will result in different physiological adaptations within the muscles. Strength training results in an initial neural adaptation followed by an increase in muscle cross sectional area (Sale 1988). The improved neuromuscular component is due to an enhanced intramuscular recruitment pattern and intermuscular coordination between the RM's as well as an increased intramusclular firing rate and synchronization of the motor units (Sale 1992). The increase in muscle cross sectional area is due to an enlargement of both type 1 and type 2 fibers with the latter changing more than the former (Kraemer, Deschenes et al. 1988). This increase occurs as a result of protein synthesis, primarily actin and myosin in the myofilaments, which produces a greater number of contractile units.

Endurance training adaptations in skeletal muscle is characterized by an improvement in their oxygen extraction ability (Docherty and Sporer 2000). This is due to an increased capillarization and mitochondrial enzyme concentration and myoglobin content in the muscle (Holloszy and Coyle 1984; Hoppeler, Howald et al. 1985). Additionally, a shift in fiber-type recruitment to type IIa fibers along with an increase in their oxidative capacity have been reported at higher levels of exercise intensity (Dudley,

Abraham et al. 1982; Staron, Karapondo et al. 1994). Increased oxygen extraction ability of the muscle can also decrease its lactate production because of the muscles decreased blood flow requirement (Powers and Howley 2001).

The adaptation to training by the skeletal muscles also depends on the type of training performed and magnitude of the training stimulus. According to the principle of specificity, training adaptations will be specific to the type of training performed (Pierce, Weltman et al. 1990). The overload principle states that the strength, endurance and hypertrophy of a muscle will increase only when the muscle performs for a given period of time at or near its maximal strength and endurance capacity (Foss and Keteyian 1998). For example, training using submaximal loads (30-40% of maximal isometric force) and high movement velocities will tend to increase the maximum velocity of muscle shortening at lower loads while training using maximal loads and slow movement velocities will tend to increase maximal muscle force production but have minimal effect on the maximum velocity of muscle shortening at smaller loads (Figure 5) (Caiozzo, Perrine et al. 1981; Behm and Sale 1993; Moritani 1993; McBride, Triplett-McBride et al. 2002)

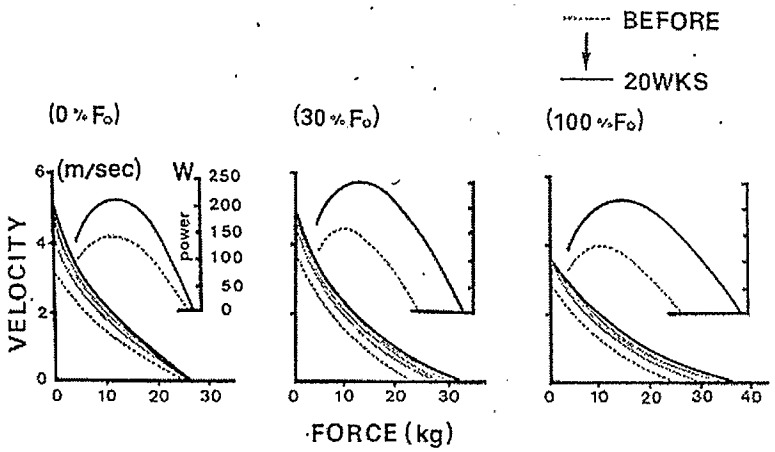


Figure 5: The time course of changes in the force-velocity (concave) and force-power (convex) relationships during muscle power training with different loads. Reprinted from Moritani (1993).

Muscular Adaptation in RM

Animal studies have demonstrated the trainability of the RM's to be similar to other skeletal muscles, although the magnitude of training induced changes is smaller (20-30% in the RM's compared to 40-80% in the locomotor muscle) (Grinton, Powers et al. 1992; Powers, Criswell et al. 1992; Powers, Criswell et al. 1992; Powers, Lawler et al. 1992; Powers, Martin et al. 1992; Powers, Criswell et al. 1994; Powers, Farkas et al. 1994). With endurance training in rats, a 12-30% increase in the number of oxidative fibers and a 20% increase in Krebs cycle enzyme activity have been observed in the diaphragm (Ianuzzo, Noble et al. 1982; Moore and Gollnick 1982). Similarly, the glycolytic enzymes LDH and hexokinase have also been shown to increase significantly in the diaphragm following such training (Ianuzzo, Noble et al. 1982; Powers, Criswell et al. 1992; Powers, Criswell et al. 1992; Powers, Lawler et al. 1992; Powers, Martin et al. 1992; Lawler, Powers et al. 1993). It should be noted that the crural and costal parts of the diaphragm respond differently to training, with a majority of the training adaptations

occurring in the costal portion (Powers and Criswell 1996). The parasternal muscles also respond to training in a similar fashion as the diaphragm. Ten weeks of endurance training has reportedly increased the Krebs cycle enzyme activity of citrate synthase and succinate dehydrogenase by 40% in this muscle (Powers, Criswell et al. 1994). Recently, Vrabas et al (1999) reported a reduction in the rate of diaphragm fatigue in the rat following treadmill training for 5days/week, 60min/day for 10wks at 70% of VO_2max (Vrabas, Dodd et al. 1999). They speculated this to be a consequence of the 10% increase in citrate synthase, 12% increase in superoxide dismutase and significant increase in Type 1 fibers in the diaphragm following this training intervention. Although diaphragm fatigue was reduced, no change diaphragm specific velocity of shortening or force production was observed.

Although animal studies are an important source for invasive information, animals nonetheless differ from human subjects and the data obtained from these studies cannot be generalized to humans. Several studies have investigated cellular changes in the diaphragm of COPD patients (Levine, Nguyen et al. 2001; Levine, Gregory et al. 2002). These studies reported transformations from type II to type I fiber types, increases in succinate dehydrogenase activity in Type 1 fibers, and higher mitochondrial oxidative capacity relative to ATP demand. Such adaptations are thought to be a result of the increased work of breathing that severe COPD patients are chronically subjected to and may make the diaphragm more resistant to fatigue by increasing its aerobic ATP generating capacity relative to ATP utilization (Levine, Gregory et al. 2002).

Since there are a limited number of studies that have investigated the cellular adaptations to training of the RM in humans, indirect measures of RM strength and

endurance have had to be used to assess training adaptations. These studies suggest that the RM's respond to training specificity and overload in a similar manner as skeletal muscles (Tzelepis, Vega et al. 1994; Tzelepis, Vega et al. 1994; Tzelepis, Kasas et al. 1999). Table 3 summarizes the changes in MIP following RMT in athletes. Significant improvements in both RM strength measured as MIP and RM endurance measured as CLRB or ITL have been observed following RMT with a pressure resistance device when using a resistance equivalent to 50% of MIP or greater (Tzelepis, Vega et al. 1994; Inbar, Weiner et al. 2000; Hart, Sylvester et al. 2001; Sonetti, Wetter et al. 2001; Volianitis, McConnell et al. 2001; Romer, McConnell et al. 2002; Williams, Wongsathikun et al. 2002). No significant improvements in velocity dependent tests such as MVV have been observed following this type of training (Inbar, Weiner et al. 2000; Hart, Sylvester et al. 2001). It should be noted that RMT training effects are insignificant below 30% of MIP and thus a minimum pressure of 30% of MIP has been used for RMT in the healthy population (O'Kroy and Coast 1993; Lisboa, Munoz et al. 1994).

Although no minimum V_E has been established for hyperpnoea training, significant improvements in both RM endurance tests such as MSVC and maximal tests such as 15 second MVV have been reported following this type of training (velocity training) (Morgan, Kohrt et al. 1987; Boutellier, Buchel et al. 1992; Boutellier and Piwko 1992; Spengler, Laube et al. 1996). No significant improvements in strength dependent tests such as MIP or endurance tests such as CLRB or ITL have been reported.

The differences in the effects between pressure resistance and hyperpnoea training are in agreement with O'Kroy et al (1993) and Tzeplepis et al (1994) who reported that high inspiratory flow training showed improvements in tests that measure flow rates

(MVV, MSVC) while high pressure loaded training showed improvements in tests that measure strength (MIP, CLRB). Furthermore, it was shown that strength gains from PRD RMT were specific to the lung volume at which training occurred and a combination of high-flow and high-pressure training increased the maximum pressure and flow generated to values achieved from isolated training (Tzelepis, Vega et al. 1994; Tzelepis, Vega et al. 1994; Tzelepis, Kasas et al. 1999).

Table 3: Change in MIP values following training with a Pressure or Flow resistive device.

Type of training	Training Load	Duration	Change in MIP (%) Experiment	Control	Reference
PRD	30% - 80% of MIP	½ hr/day, 10wks	25%**	1%	(Inbar, Weiner et al. 2000)
PRD	Max resistance which 30breaths could be completed	30 breaths, 2times/day, 4wk	45%**	5%	(Volianitis, McConnell et al. 2001)
FRD	50% of MIP	10min, 2times/day, 4wk	18%**	3.8%	(Hanel and Secher 1991)
PRD, IH	47-55% of MIP	40 breaths, 5wks	8%**	3.7%	(Sonetti, Wetter et al. 2001)
PRD	Max resistance which 30breaths could be completed	4wks (unsure of protocol)	31%*	-	(Williams, Wongsathikun et al. 2002)
PRD	Max resistance which 30breaths could be completed	30 breaths, 2times/day, 6wk	28%**	1.7%	(Romer, McConnell et al. 2002).
PRD	Max resistance which 30breaths could be completed	30 breaths, 2times/day, 6wk	12%**	1%	(Hart, Sylvester et al. 2001)

** Significantly different from control or placebo ($p<0.05$), * significant improvement from pre value ($p<0.05$) IH: isocapnic hyperpnoea, PRD: pressure resistive device, FRD: flow resistive device

Studies using FRD have reported varying results that can be explained from the protocols used. Generally low flow rates have been shown to have less of a training effect than high flow rates (Belman, Thomas et al. 1986). This can probably be attributed to the flow rate dependency of these devices

Although improvements in overall respiratory muscle strength measured as MIP have been reported following PRD RMT, few studies have investigated the specific effect of PRD RMT on diaphragm strength. Suzuki et al (1993) and Wanke et al (1994) reported a 30% improvement in Pdimax performed as a combined feedback maneuver and sniff maneuver respectively, while Hart et al (2001) did not report any change in Pdimax twitch or sniff Pdi following PRD RMT. A possible reason for these conflicting results is that Suzuki et al (1993) did not measure Pdimax in the control group and thus this improvement may have been a result of familiarization rather than an improvement in strength. Although RMT improves MIP, any specific improvement in diaphragm strength is still uncertain.

In contrast to strength and endurance tests, pulmonary function measurements of vital capacity (VC), forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV_1) have not been reported to change following RMT (Fairbarn, Coutts et al. 1991; Hanel and Secher 1991; Boutellier, Buchel et al. 1992; Boutellier and Piwko 1992; Inbar, Weiner et al. 2000).

Effects of RMT on measures of ventilatory and metabolic performance

Several studies have evaluated the effects of RMT on ventilatory and metabolic measures. Spengler et al (1996 & 1999) and Boutellier et al (1992a) reported a decrease in blood lactate levels at the end of an incremental cycle test following IH RMT while

other studies including a recent study by the same group failed to detect any between group changes in blood lactate concentration following IH and PRD RMT (Boutellier, Buchel et al. 1992; Spengler, Laube et al. 1996; Spengler, Roos et al. 1999; Sonetti, Wetter et al. 2001; Stuessi, Spengler et al. 2001; Volianitis, McConnell et al. 2001). It is possible that the significant reduction in lactate values reported in the 3 studies were a consequence of having no control group since the Volianitis et al (2001) study also reported a significant decrease in lactate values, however these changes were not significant between groups. The decrease in the perception of dyspnea at each stage of an incremental rowing ergometer test has also been reported in the experimental group following PRD RMT although this difference was not significantly different from the control group (Volianitis, McConnell et al. 2001). It was suggested by the authors that this change might be indicative of a decreased work of breathing. Although conflicting, some studies have shown an increased V_T and decreased *f* during submaximal constant load tests following PRD, FRD and IH RMT suggesting a less fatigued breathing pattern, but only one of these groups had a control group (Hanel and Secher 1991; Boutellier, Buchel et al. 1992; Boutellier and Piwko 1992; Amonette and Dupler 2001). VO_{2max}, MAP, V_{Emax} and the anaerobic threshold have not shown to differ significantly following any type of RMT (Fairbarn, Coutts et al. 1991; Boutellier, Buchel et al. 1992; Boutellier and Piwko 1992; Spengler, Laube et al. 1996; Spengler, Roos et al. 1999; Inbar, Weiner et al. 2000). Recently, Romer et al (2002) and Volianitis et al (2001) reported an attenuation of global inspiratory muscle fatigue measured by MIP following PRD RMT in trained cyclists and rowers .

Effects of RMT on athletic performance

For any training intervention, field performance is an important measure of an interventions success. Despite the numerous studies, the benefit of RMT on athletic performance is still not clear. Table 4 summarizes current studies that have examined the effect of RMT on aerobic performance. Boutellier et al (1992a & 1992b), Spengler et al (1996 & 1999), Steussi et al (2001) and Markov et al (2001) (although the last two articles appear to be from the same study) reported a prolonged cycling time following IH RMT in both sedentary and normal trained subject. Similarly, Volianitis et al (2001) reported an improvement in the 6 min and 5km performance bouts following PRD RMT in rowers and Romer et al (2002) reported an improvement of 3-4% in 20 and 40km cycle time trial performance following RMT. Other studies have failed to report such improvements in performance (Morgan, Kohrt et al. 1987; Fairbarn, Coutts et al. 1991; Hanel and Secher 1991; Sonetti, Wetter et al. 2001). Fairbarn et al (1991) and Morgan et al (1987) reported no significant changes in cycling time when performed at 90% and 95% of VO_{2max} following IH RMT, Sonetti et al (2001) reported no significant changes in 8km time trial performance following PRD and IH RMT and Hanel et al (1991) reported no significant improvement in the 12km distance run following FRD RMT .

Table 4. Summary of studies examining the relationship between RMT and aerobic performance

Reference	RMT Method	Sample size/subjects	Training frequency	Training load	*f*	Outcome measures
(Boutellier and Piwko 1992)	IH	4 sedentary subjects	½ hr/day; 5days/week; 4weeks	V_E- 76-102L/min	38 b/min	CE-↑, BE - ↑, BL – ↓ O_2 saturation – NC
(Boutellier, Buchel et al. 1992)	IH	8 healthy trained subjects	½ hr/day; 5days/week; 4weeks	V_E- 85-160L/min	38-46b/min V_T- 2.3-3.5L	CE-↑, BE, MVV - ↑, AnT - NC VO2max, BL (cycle test,V02max) –NC
(Spengler, Roos et al. 1999)	IH	19 healthy active males	½ hr/day; 5days/week; 4weeks	V_E - 123→162L/min	40-50b/min	CE – ↑ BE, MVV – ↑, VC, FEV– NC VO_2max, MAP, AnT – NC BL (cycle test,V02max) - ↓
(Markov, Spengler et al. 2001)	IH	13 E_1 15 C 9 E_2: Healthy sedentary	E_1 : ½ hr/session; 40 session within 15wks E_2 : ½ hr/session; 40 session within 15wks (endurance training)	V_E- 60% → 79% of MVV		CE- ↑, BE - ↑ MAP, VO_2max : NC
(Spengler, Laube et al. 1996)	IH	G1-10 high V_T and Low *f* G2 - 9 low V_T and high *f* Active males	½ hr/day; 5days/week; 4weeks	G1- V_E 113→150L/min G2- V_E 128→160L/min	G1– 40b/min, 2.6-3.8L G2- 38→60b/min, 2.2-3.4L	CE (AnT) – ↑ , VO_2max, Vemax – NC BE, MVV – ↑ BL- ↓ at 10-14thmin of cycle test
(Fairbarn, Coutts et al. 1991)	IH	5 E, 5 C Well-trained cyclists	3x8min sessions/day 4 days/week; 4 weeks	V_E - 155→173L/min Nothing about V_T or *f*	-	CE (90% VO2 max), VO_2max, Vemax- NC BE – ↑
(Inbar, Weiner et al. 2000)	PRD	10 E, 10 C Endurance athletes	½ hr/day; 6days/week; 10weeks	Begin at 30% and increased to 80% of MIP	-	MIP - ↑, BE - ↑ FVC, FEV, MVV - NC VO2max, VE max,O_2 sat., AnT – NC
(Volianitis, McConnell et al. 2001)	PRD	7 E, 7 C Elite rowers	E: 30 efforts/session 2 session/day C: 60 efforts/day 11wks	E: begin at 50% of MIP → max pressure could sustain 30 breaths C: 15% MIP	-	MIP - ↑ 6min, 20km performance test - ↑ CR-10 BORG scale - ↓
(Sonetti, Wetter et al. 2001)	PRD/IH	9 E, 8 C Moderately trained cyclists	PRD: 3-5min/day, IH: 30-35min/day 5days/week 5 weeks	50% MIP V_E-50-60% MVV	E: 57-68 b/min (IH)	MIP - ↑ 8km TT- NC VO2, submax test: NC
(Hanel and Secher 1991)	FRD	10 E, 10 C College students	2x10min/day; 7days/week 4 weeks	50% of MIP	6-8b/min	12min track test – NC MIP - ↑, FEV, FVC – NC VO2max, VE max – NC
(Morgan, Kohrt et al. 1987)	FRD	4 E, 5 C Moderately trained cyclists	4times/day (2min, 5min, 9min, 12min) 5 days/week; 3weeks	-	-	CE (95% VO2 max) – NC, Training load – ↑, BE, MVV_{15} – ↑ VO2 max - NC
(Romer, McConnell et al. 2002)	PRD	8E, 8C Highly trained cyclist	E: 30 efforts/session 2 session/day C: 60 efforts/day 6 wks	E: begin at 50% of MIP → max pressure could sustain 30 breaths C: 15% MIP	-	MIP - ↑ 20km, 40km performance test - ↑ MIP following test - ↓

IH: Isocapnic Hyperpnoea, PRD: pressure resistance device, FRD: flow resistance device, V_E: ventilation rate, BE: breathing endurance, CE: cycle endurance, BL: blood lactate, MIP: maximum inspiratory pressure, AnT: anaerobic threshold, MAP: maximal aerobic power, $V0_2$max: maximal oxygen uptake, V_T: tidal volume, *f*: breathing frequency, NC: no change

Limitations of Current Literature

The conflicting results of the current studies evaluating the effects of RMT on performance may be explained in part by the differences in study design.

Placebo Group

Perceiving that an intervention will improve performance can have a significant effect on the outcome itself. Clarke et al (2002) reported a 3.8% and 5.4% improvement in a simulated 40km time trial in subjects who were falsely told they were receiving carbohydrate (CHO) supplementation compared to subjects who were told they were in the placebo group and subjects who were not told which group they were in (Clark, Hopkins et al. 2000). A 3.8% and 5.4% improvement in performance due to expectation is clearly performance significant, particularly when the margin between a medal standing is as small as several percent. Although RMT studies by Boutellier et al (1992a,b), Spengler et al (1996, 1999), Stuessi (2001) and Markov et al (2001) reported significant improvements in a constant-load exercise test following IH RMT, only the study by Markov et al (2001) utilized a control group. While the inclusion of a control group improves the strength of the study design, it still does not provide the expectation of improvement. This makes it difficult to determine how much of the improvement observed in the experimental group of these studies is due to the RMT and how much is due to the expectation of improving performance. This is one of the observations made by Sonetti et al (2001), where they compared the effect of PRD and IH RMT to placebo RMT training in trained cyclists. This group reported significant improvements in constant load exercise performance, MAP and 8km time trial, however when compared between groups, these improvements were not significant. In contrast to the observations

made by Sonetti et al (2001), Romer et al (2002) and Volianitis et al (2001) both reported significant between group improvements in cycling and rowing performance respectively when using a placebo group. There are a few possible reasons for the different observations between the Sonetti study and the latter two studies.

a) The variability in the outcome measures were greater in the study by Sonetti et al (2001) compared to the studies by Romer et al (2002) and Volianitis et al (2001), thus decreasing the ability to detect a significant difference. The variability in mean improvement time for the time trials in the studies by Romer et al (2002) and Volianitis et al (2001) were between 1.0% - 1.9% while the variability in mean improvement time for the time trials in the study by Sonetti et al (2001) were between 1.2% –2.7%. The lower variability in the latter two studies is likely because of the highly trained status of their participants compared to the moderately trained status of the former study. The greater variability in the study by Sonetti et al (2001) might mask any possible improvement from RMT and may be a consequence of the subjects lower training status.

b) The baseline RM strength of the subjects varied between studies. This is another possible reason for the discrepancies observed between these studies. Although the study by Romer et al (2002) used highly trained male cyclists with a VO_2max around 4.60 L/min, their baseline MIP was only 104cmH_20. In contrast, the cyclists in the study by Sonetti et al (2001) used cyclists with a lower mean VO_2max (4.07 L/min) but a much larger MIP of 168cmH_20. Since the former study showed larger performance improvements than the latter study (3.8 and 4.6% in the former vs. 1.2% in the latter), it may be that weaker RM's play a

greater role in limiting performance and demonstrate a larger training potential than stronger RM's. This is suggested by the significantly larger increase in RM strength in the former study as compared to the latter study (28% vs. 8%)

Sample Size

One of the primary limitations in most studies examining the effect of RMT on performance is the use of small sample sizes. Every study measuring the effects of RMT on athlete performance that has used a control/placebo group except for Markov et al (2001) has used a sample size of 10 or less subjects per treatment group (Morgan, Kohrt et al. 1987; Fairbarn, Coutts et al. 1991; Hanel and Secher 1991; Boutellier, Buchel et al. 1992; Boutellier and Piwko 1992; Spengler, Laube et al. 1996; Inbar, Weiner et al. 2000; Sonetti, Wetter et al. 2001). The smaller sample size of these studies weakens the statistical ability to distinguish a significant difference between pre and post training parameters particularly when the outcome measures exhibit considerable variability. It can be estimated that a sample size of 12 subjects per group is required to detect a minimum change of 2.0% in an 8km cycle time trial (assuming a SD of 1.7%). It is likely that many of these studies may have been expecting a large effect size following RMT (around 20-30%). Expecting a large effect size is unrealistic particularly because the respiratory system is less of a limiting factor than the cardiovascular and locomotor systems (Boutellier and Piwko 1992). Therefore, any improvements following RMT are likely to be small in magnitude. Since improvements of only a few percent can be considered significant in trained athletes, it is important that studies are designed so that they can detect a small effect size before any definite conclusions can be made.

Outcome Measures

The use of inappropriate outcome measures may be another explanation for the conflicting results reported in recent studies.

Maximal Oxygen Uptake

VO_{2max} values using incremental tests have shown to be insensitive to certain training adaptations, particularly those in highly trained individuals (Daniels, Yarbrough et al. 1978; Foster, Schrager et al. 1996). It is possible that this test may also be insensitive to RMT adaptations and thus a poor outcome measure of aerobic performance in these studies. Additionally, training induced improvements in the critical power of the RM's will generate a much smaller improvement in an incremental test compared to an endurance test (Walsh 2000). Thus, a larger sample size would be required to generate the same statistical power when using an incremental test as compared to an endurance test performed at a constant work-rate. This may be a key limitation of the study by Inbar et al (1999) where the only performance measure used was an incremental VO_2max test (Inbar, Weiner et al. 2000).

Open-ended tests

Using time to exhaustion tests is another limitation of the studies by Fairbarn et al (1991) and Morgan et al (1991). These types of tests are shown to exhibit substantial individual variability when compared to time trial tests (interclass correlation coefficient (ICC) = 0.47 vs. 0.97) (McLellan, Cheung et al. 1995; Harms, Wetter et al. 2000; Sonetti, Wetter et al. 2001). Since the ability to detect a significant difference if one actually

exists from a given sample size diminishes with increasing variability, a larger sample size would be required to detect a significant difference when using these types of tests. Thus the inability of these studies to detect a significant difference in performance following RMT may be a consequence of the type of test used.

Another critique of this type of test is the unusual nature of the test itself. Rarely in real life competition is the winner of an event defined as the person who could last the longest. However, this is exactly what this type of test is measuring. The unfamiliar nature of this test and poor applicability to real life competition may help explain the substantial random variation associated with this test (Sonetti, Wetter et al. 2001).

Time trial

In contrast to open-ended tests, a fixed distance time trial test shows small random variation between trials. Hickey et al (1992) reported an intraindividual CV ranging from +/-0.95% to +/-2.43% with the shortest distance having the highest variability. Similarly, Sonetti et al (2001) reported a small CV ranging between +/- 0.9 – 1.1% for an 8km time trial. The reason for the considerably lower random variability in fixed distance time trial tests is likely due to its similarity with real life competition (Coyle, Feltner et al. 1991; Sonetti, Wetter et al. 2001).

Exercise Intensity

Another problem in deciding on an adequate outcome measure involves knowing which exercise intensities may benefit from RMT. Harms (2000) suggested that improved RM efficiency is only likely to benefit maximal or near maximal intensity

exercise where cardiac output becomes a limiting factor. This observation is supported by Volianitis et al (2001) who reported improvements in the 6 minute distance and 5km time when testing was performed on a rowing ergometer. Such activities are likely to have been performed at an intensity close to 100% of VO_2max and 85-90% of VO_2max respectively. On the other hand, the positive results reported in the literature also support the benefit of RMT at lower exercise intensity. Boutellier et al, (1992a & 1992b), Spengler et al (1996 & 1999), Markov et al (2001) and Romer et al (2002) have shown a prolonged time to fatigue when exercise is performed at or slightly below anaerobic threshold (AnT). It is difficult to definitely conclude which exercise intensities benefit from RMT but it is possible that more than one physiological adaptation may occur and thus there is a range of exercise intensities that will benefit from RMT. Examining the physiological adaptations to RMT may help clarify this discrepancy.

Breathing Frequency and Tidal Volume

Two particular important parameters of RMT regimes that may be an important determinant of the success of RMT are the f and V_T adopted during RMT. Although most of the studies using hyperpnoea to train the RM's have controlled these parameters, studies using pressure resistance devices to train the RM's have not. This may offer an explanation for the conflicting results reported between studies that have used IH or PRD. Belman et al (1986) reported that subjects who chose their own f tended to adopt a slower breathing pattern than normal. Such a pattern is better tolerated by subjects because it results in lower respiratory pressures, reduction in RM work, reduction in the sensation of breathing effort and a slower rate of RM fatigue development (Belman, Thomas et al. 1986). In fact, when using PRD RMT devices, the estimated work of breathing is

significantly lower when adopting a *f* of 15bpm as opposed to 30bpm (Belman, Botnick et al. 1994). The adaptations of the RM to training are also specific to the lung volume and speed at which the training occurs (Tzelepis, Vega et al. 1994; Tzelepis, Vega et al. 1994). Training effects may be reduced during a breathing pattern that adopts a low *f* and V_T and thus give the apparent result that RMT is not effective for improving athletic performance.

Study Characteristics

RMT Training Method

The two most common methods used to train the RM are IH and PRD RMT. As discussed earlier, IH RMT only showed improvements in flow resistive test such as MVV and MVSC while PRD RMT only showed improvement in pressure resistive tests such as MIP, CLRB and ITL. According to the recent studies by Volianitis et al (2001), Romer et al (2002), Spengler et al (1996 & 1999), Markov et al (2001) and Steussi et al (2001) both PRD and IH RMT appears to improve aerobic performance in both highly trained and normal subjects. The current literature suggests that the adaptations of the RM to IH and PRD RMT differ, yet both seem to improve performance. Cleary, the current literature has not addressed this question. This is an interesting area of RMT that may provide some further insight in to the mechanism of performance improvement from RMT and thus should be the focus of further research.

Training Load and Duration

Training durations have varied between 4 to 10 weeks while training frequencies have varied between 30 breaths, twice a day to ½ hour continuous training bouts

(Boutellier, Buchel et al. 1992; Boutellier and Piwko 1992; Spengler, Laube et al. 1996; Spengler, Roos et al. 1999; Inbar, Weiner et al. 2000). Wide variations in training loads have also been used for RMT. Training loads when using a pressure resistive training device and hyperpnoea training have ranged between 30% and 80% of MIP and between 76 and 170L/min respectively. A summary of the various training loads and durations used has been summarized in Table 4. Although a dose response relationship of the RM to training stimulus may be observed as is observed in other skeletal muscle, Volianitis et al (2001) reported a 40% improvement in MIP at the 4 week mark of RMT but very slight further increase of 5% in MIP following 6 further weeks of RMT. This results may suggest that similar to other skeletal muscle, the initial rapid improvement in RM strength is due to a neural adaptation (improved coordination and learning) and any further increase in strength will occur at a slower rate that results from adaptations within the muscle (Sale 1988). Although the ideal training load and duration has yet to be established, preliminary research suggests that PRD RMT should performed for at least 4weeks at 50% of MIP consisting of 2 sets of 30 breaths/day (Volianitis, McConnell et al. 2001; Romer, McConnell et al. 2002). The RMT regime for IH should be performed ½ hour a day for 4 weeks at around 60-85% of MVV (Spengler, Laube et al. 1996; Spengler, Roos et al. 1999).

Subject Characteristics

Subject characteristics between studies vary considerably. Boutellier et al, (1992b) used sedentary individuals, Sonetti et al (2001) used moderately trained cyclists and Inbar et al (1999), Fairbarn et al (1991), Romer et al (2002), and Volianitis et al

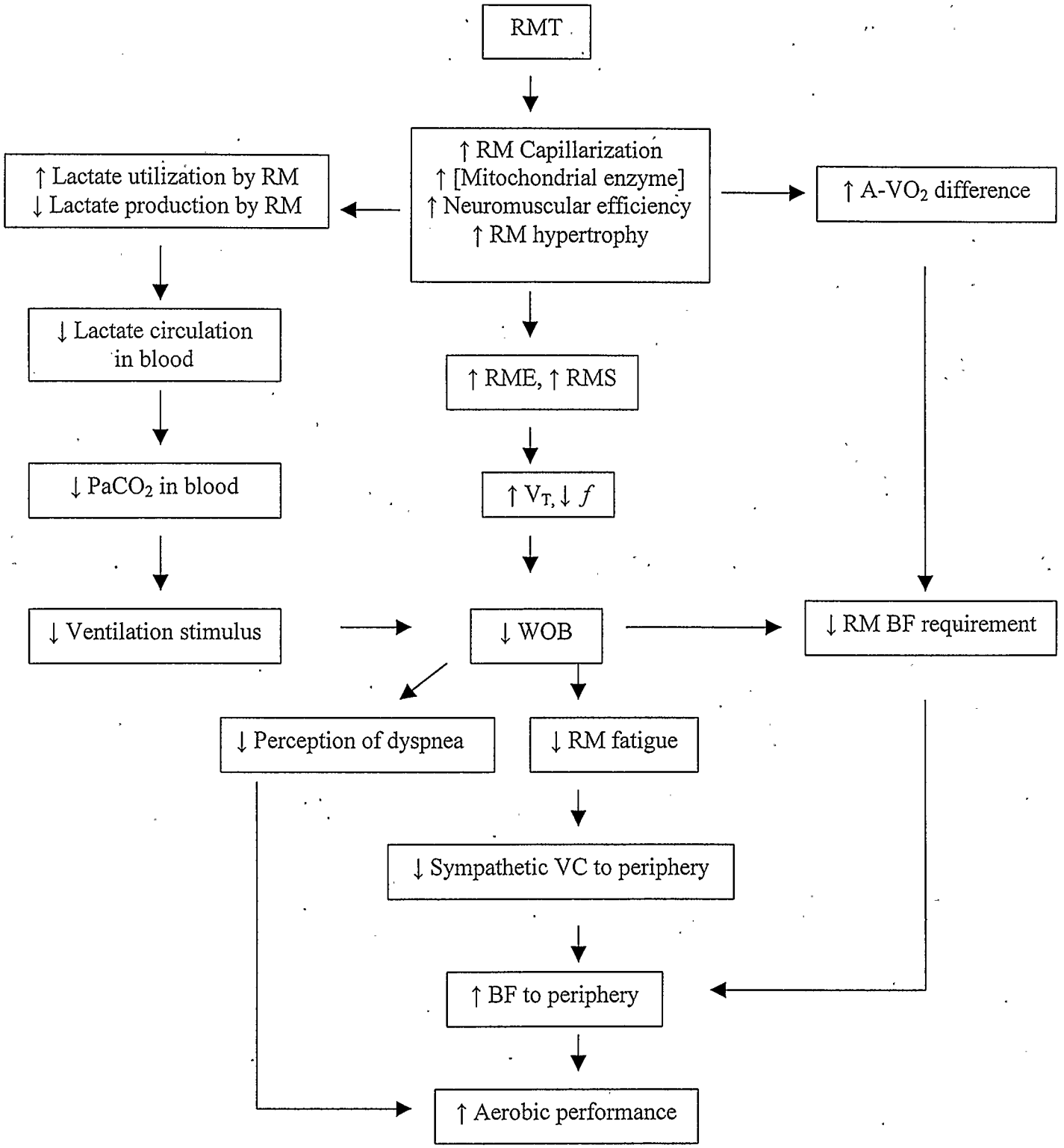
(2001) used highly trained endurance runners, cyclists and rowers. It is difficult to determine from the current literature whether training status is a confounding factor in RMT. One suggestion is that individuals with high RM baseline strength may not benefit as much as individuals with lower RM baseline strength because higher RM baseline strength has shown to incur less fatigue following exhaustive exercise (McConnell, Caine et al. 1997). Furthermore, these individuals may have already reached or are close to their upper limit for adaptation to RMT. The high baseline strength of the subjects in the study by Sonetti et al (2001) may explain the smaller improvements observed in MIP following RMT and the non-significant improvement in performance. This is contrast to the studies by Romer et al (2002) and Volianitis et al (2001) that used subjects with lower baseline RM strength and did show significant improvements in performance.

Another suggestion is that less trained subjects are more variable than highly trained individuals in tests measuring performance. Highly trained individual are less likely to be affected by motivation, familiarization and acute physiological changes. Any improvement from RMT may be masked by the variability in less trained individuals giving the apparent notion that RMT is not effective in improving performance.

Proposed model for the benefits of RMT on athletic performance

The respiratory muscles can utilize up to 15-16% of the cardiac output during maximal intensity exercise in trained individuals (Aaron, Seow et al. 1992; Harms, Babcock et al. 1997). The author proposes that RMT may decrease the RM's blood flow requirement during maximal exercise and enable a greater proportion of the cardiac output to be redirected to the working locomotor muscles (Figure 6).

Figure 6: Proposed model of the effects of RMT on aerobic performance



A-VO_2 difference: arterial-venous oxygen difference, *f*: breathing frequency, BF: blood flow, RM: respiratory muscle, RME: respiratory muscle endurance, RMT: respiratory muscle training, RMS: respiratory muscle strength, VC: vasoconstriction, V_T: tidal volume, PaCO2: arterial CO2 pressure, WOB: work of breathing

This model is based on the adaptations to training that have been reported in animal studies (discussed in the previous section). These may in part be extended to human. An increase in RM strength and endurance may result in a decreased work of breathing as a result of more efficient muscles (Volianitis, McConnell et al. 2001; Romer, McConnell et al. 2002). Consequently, RM fatigue is reduced or delayed (less tachypneic breathing pattern) and could in turn decrease sympathetically mediated vasoconstriction of blood flow to the locomotor muscles (Volianitis, McConnell et al. 2001; Romer, McConnell et al. 2002). In a parallel fashion, decreasing the lactate concentration in the blood would also decrease blood $PaCO_2$. This would reduce its known stimulatory effect on V_E, which in turn would suppress the work of breathing and possibly reduce the perception of dyspnea (Johnson, Aaron et al. 1996; Wasserman, Hansen et al. 1999). All these effects would increase the proportion of blood flow available to the working locomotor muscles and could theoretically augment performance. Thus RMT may be beneficial during high intensity exercise bouts where the cardiac output is limited.

CHAPTER THREE

METHODOLOGY

Overview of Methodology

Prior to commencement of training, all subjects underwent baseline testing of the performance variables. Subjects assigned to the experimental group underwent pressure resistive RMT consisting of 3 sets/day, 6 days/week for four weeks at 50% of their maximal inspiratory pressure (MIP) while subjects assigned to the control group followed a similar protocol except that the training load was set at 10% of their MIP. At the completion of training (4weeks), post-experimental testing was conducted on the same variables as in the pre testing sessions.

Testing Sites

Pulmonary Function Lab, Rockyview General Hospital, Calgary, Alberta and Human Performance Laboratory, Faculty of Kinesiology, University of Calgary, Calgary, Alberta.

Subjects

Sample Size/Subject Recruitment

26 competitive male road cyclists, mountain bikers, triathletes and speed skaters, aged 18 to 40 years old were recruited for the study.

The sample size for each group was determined using a web-based sample size calculator for independent groups with unequal variances (http://ebook.stat.ucla.edu/calculators/powercalc/normal/n-2-unequal/n-2-uneq-var-

samp.php). The calculation was based on the 8km time trial (primary outcome) using cycling data from past research (Sonetti, Wetter et al. 2001; Volianitis, McConnell et al. 2001; Romer, McConnell et al. 2002). Variability in repeated time trials have ranged from 1.0-2.7% and performance improvements from RMT have ranged from 2.0-4.6%. The probability of committing a type I error (alpha value) was set at 0.05. The probability of committing a type II error (beta value) was set at 0.20, thus giving a power of 0.80.

To measure an effect size of 2.0% between the experimental and control group using a standard deviation of 1.7%, a sample size of 12 participants per groups was required. Assuming 1 dropout per group, 13 subjects per group was the intended sample size.

Inclusion criteria

The inclusion criteria were:

1. Training for a minimum of 1 year with a club or competed in at least 3 competitions in the previous year
2. VO_2max greater than 3.85 L/min or 55 ml/kg/min (this cutoff helped to identify participants with a moderate to high level of endurance training background)
3. No chronic respiratory diseases
4. Willing to perform the tests and training program required for this study

Subjects were recruited by the primary investigator through advertisements in local bicycle shops; triathlon, mountain bike and road bike clubs; and postings on the Alberta Bicycle Association and Pink Bike.com websites (Appendix A). Recruitment

began in December 2001 and was ongoing until June 2002. This study was performed in 3 batches for logistic reasons (between 6-9subjects per batch): batch 1 from January – March, batch 2 from February to April, batch 3 from May to July. All potential participants that showed interest in the study initially completed a phone interview questionnaire (Appendix B). Those candidates that passed the criteria of the phone interview were invited to participate in the study and were booked for baseline testing. Each participant completed a physical activity readiness questionnaire and a practice 8km time trial prior to any physical testing sessions (Appendix C). The purpose of this initial 8km time trial ride was to increase the participant's familiarity with the test. Also, all participants were fully informed about the aim and potential risks of the investigation before giving written consent although the exact nature of the 2 groups was not revealed (Appendix D). The Medical Bioethics Committee at the University of Calgary approved the experimental protocol. During all testing sessions, subjects were told to:

- Abstain from vigorous exercise 2 full days prior to each testing session
- NO caffeine beverages at least 6 hours before each test or training session
- NO alcoholic drinks 6 hours before each test or training session
- NO smoking 2 hours before each test or training session.

All tests were performed according to the ACSM Guidelines for Exercise Testing and Prescription (1995). This included the termination of a test if:

- Onset of angina or angina like symptoms
- Signs of poor perfusion: lightheadedness, confusion, ataxia, pallor, cyanosis, nausea or cold and clammy skin

- Failure of heart rate to increase with increased exercise intensity
- Subject requests to stop
- Physical or verbal manifestations of severe fatigue
- Failure of the testing equipment

Outcome Measures

These were the outcome measures for the study:

1. Cycle time trial performance
2. Maximal incremental exercise test
3. Constant load submaximal test
4. RM strength and endurance tests

As well as assessing performance from a laboratory perspective, these measures also extend the applicability of the results to a practical setting. A sport physiologist or strength coach may then be able to determine if the results of this study could be directly used as part of their athletes training plan.

Maximum intensity 8 km cycle time trial.

This outcome measure has low random variability (CV: +/- 0.9-1.1% and ICC of 0.97), is used for training purposes, shares many similarities with competition, and is a distance that would be minimally affected by motivation (Sonetti et al, 2001). Additionally, it is performed at an intensity equivalent to about 95% of VO_2 max, which is an intensity that may benefit from RMT. During the time trial, heart rate was measured using an electronic heart rate monitor (Polar). The time trials were performed in the Human Performance Lab on a set of Kreitler rollers (Kreitler Rollers Inc., Ottawa, KS) in

the "killer headwind" mode with subjects using their own bicycles. The rollers had the front fork fixed for these time trials so that subjects with little on no rollers experience could perform the trials with minimal learning. Each time trial began with a warm-up of 5-10 minutes and subjects received real-time information on their speed only. Other feedback was not provided to minimize additional motivational effects in subsequent trials. Tire pressure was maintained at the same pressure for each time trial and subjects did not use their tire for any other riding. This was to minimize any impact that tire pressure and wear may have had on the reproducibility of the trials. Subjects performed a practice time trial before the start of the study to become familiar with the test.

Maximal incremental cycle test (VO_2max test).

A graded cycle, maximal oxygen consumption test was performed to identify individual fitness levels and provide a power output for the submaximal constant load test. This test was performed on an electronically braked ergometrics 800S cycle ergometer (Sensor Medics, Yorba Linda, California). Subjects began with a 5-10 minute warm-up at a workload of their choice followed by an incremental increase in the workload of 30W every 2 minutes beginning at 150W until the second ventilatory threshold (VT2), as described by (McLellan 1985), was observed. Thereafter, the workload was increased by 15 W every minute until subjects fell below a cadence of 50rpms. V_E, rate of carbon dioxide expiration (VCO_2), rate of oxygen uptake (VO_2), ventilatory equivalents for carbon dioxide and oxygen (V_E/VCO_2, V_E/VO_2), V_T and f was measured breath-by-breath using a Sensormedics Vmax 2200 metabolic cart (MMC Horizons, Yorba Linda, California). Heart rate was also recorded every 30 seconds. The

metabolic cart and the cycle ergometer were appropriately calibrated before every test. In order to minimize the effects of different seat heights on the performance measures, the seat height was kept consistent between tests for the second half of the subjects (Nordeen-Snyder 1977).

Submaximal constant load cycle test.

This purpose of this test was to provide information on ventilatory and metabolic parameters at a constant workload equivalent to AT + 50%Δ where AT is the work rate corresponding to the pre-training 2nd ventilatory threshold and Δ is the difference between the work rate corresponding to the 2nd ventilatory threshold and MAP for that subject. Subjects began with 4 minutes of cycling at 50W followed by a maximum of 10 minutes of exercise at the pre-determined workload. If subjects were unable to complete the full 10 minutes of exercise (cadence fell below 50rpms), the termination time was recorded and the subject exercised for the same time period in the post training exercise test. V_E, VCO_2, VO_2, HR, V_T, T_I/T_{TOT} and *f* were measured using the same method as the incremental cycle test. In addition, blood lactate was assessed at pretest, 5 minutes into the test, test termination, and 5 minutes posttest. Blood was sampled from the fingertip and analyzed for its lactate concentration using the Lactate Pro LT-1710 Portable Lactate Analyzer (Arkray Factory Inc.). The lactate pro analyzer has a correlation greater than 0.98 and a mean difference that varies less that 0.52mM with YSI 2300 and the ABL 700 series acid-base analyzer through a physiological range of 1.0-18.0mM (Pyne, Boston et al. 2000). The perception of breathlessness and muscular exertion by the legs was assessed using a modified CR-10 Borg scale at baseline, 3 and 6 min during the test, upon

test termination and 5 min post test. Both these measures have been shown to be valid and reliable when used under these conditions (Borg 1998).

Respiratory muscle strength

Global inspiratory and expiratory muscle pressures were assessed as the peak values for MIP, sniff Pes and MEP while the diaphragm's contribution to maximal inspiratory pressure was assessed as the peak value for Pdimax and sniff Pdimax. A round disposable mouthpiece was used with a 1.5mm hole inserted to minimize pressure generation by the facial muscles. Pes was measured with the insertion of a balloon, 6cm long, 1.05cm in diameter, and containing 0.4ml of air attached to polyethylene tubing (100 cm in length, 1.7mm ID) positioned in the midesophagus about 45cm from the anterior nares. Pga was measured using a second balloon and tubing of the same dimensions but containing 10.0 ml of air, positioned in the stomach 65cm from the anterior nares. This volume was equivalent to about 200 cmH20. Each balloon catheter system recorded a zero pressure change over a range of balloon volumes from 0.4-4.0ml. The 10ml inserted into the gastric balloon showed a linear relationship between an external pressure of 0 –150cm H20 (Appendix D). The detailed constructions of the balloon-catheter systems are provided by Mead et al (1955). Both balloons were passed through the same nostril after topical anesthesia of the nasal mucosa (lidocaine 2%) and were swallowed by drinking cold water through a straw. Once in their respective location, the balloons were emptied by subjects performing a valsalva maneuver with the catheters open to atmosphere and then filled with either 0.4ml or 10.0ml of air. The mouthpiece and balloon catheter systems were connected to Validyne MP 45-1 pressure

transducers (Validyne, Northridge, CA), range +/- 200cmH20 that had the signal demodulated and amplified by a HP 8802A medium gain carrier amplifier and analyzed using Datasponge© 2000 computer program (sampled at 100Hz). Pdimax was obtained from a separate pressure transducer that had the Pga catheter attached to the positive end and the Pes catheter attached to the negative end (Pdi = Pga – Pes).

The protocol for the assessment of MIP and MEP was similar to that outlined by Clanton and Diaz (1995), Black and Hyatt (1969) and Wen et al (1997).

- Nose clip was attached to the subject's nose to prevent leakage.
- Subjects were asked to take 1 or more deep breaths and then exhale to RV. The subjects then inhaled maximally and were asked to maintain inspiratory efforts for greater than 1 second.
- The procedure was repeated a maximum of 20 times. The recorded value of MIP was taken as the average of the three largest peak values obtained .
- Testing of MEP utilized the same procedure as MIP except that maximum expiratory effort was produced at TLC and with the cheeks supported by the hands.

Pdimax was measured using two different protocols. The first protocol was the same one used to perform MIP (Muller maneuver). This protocol is similar to that outlined by Miller et al (1985). The second protocol used to measure Pdimax involved subjects performing maximal sniffs (short sharp sniffs as hard as possible and such that peak Pdi is not sustained) while seated with at least 2 quiet breaths between each sniff.

Subjects were provided with visual feedback of sniff Pdi. Maximal sniffs were performed until a plateau value was reached and was followed by a further 8-10 maximal sniffs to ensure no further increase (Miller et al, 1985). Sniff Pes was determined from the sniff Pdimax maneuver. The value immediately preceding the maneuver was assigned an arbitrary value of zero and changes in pressures swings were determined for Pdi and Pes (Ford, Whitelaw et al. 1983; Wanke, Formanek et al. 1994; Wanke, Toifl et al. 1994). The tests were administered with the subject sitting in the erect position. The position of the catheters between pre and post training trials remained consistent to minimize any additional variability that may be introduced through positioning and the volume of air in the balloons was checked periodically.

Respiratory muscle endurance

Respiratory muscle endurance was assessed with constant load resistive breathing (CLRB). CLRB was measured by breathing against a pressure resistance equivalent to 50% of MIP until exhaustion. This is similar to the protocol used by Perret et al (1999) but the resistance was 30% smaller. During the CLRB test, subjects ventilated at a V_T of 2 L and at a *f* of 35 bpm. From preliminary testing, we found 50% of MIP to be the maximal resistance that subjects could ventilate at while maintaining a *f* of 35 bpm and a V_T of 2L. Feedback on these parameters was provided on a breath-by-breath basis using the Sensormedics Vmax 220 metabolic carts. The test was terminated when the *f* dropped below 30bpm or tidal volume fell below 1.70 L for 3 continuous breaths. The test was administered with the subject sitting in the erect position.

Protocol

Table 5 outlines the time line for this study.

Table 5: Time line for study

Baseline Measures				**Training Period**	**Post-training Evaluation**			
Wk# 1-2				Wk# 3-6	Wk# 7-8			
Day 1	Day 5	Day 10	Day 15	Day 21-49	Day 50	Day 53	Day 55	Day 60
PFT VO_2max	Submax Test MIP/MEP/ Pdi/CLRB	8km TT	8km TT	MIP re-evaluated on the first day of each week	PFT VO_2max	Submax Test MIP/MEP/ Pdi/ CLRB	8km TT	8km TT

All recruited subjects began with a VO_2max test on day one that included pre and post spirometry (flow-volume loops). The purpose of the pre and post spirometry (PFT) was to identify subjects with chronic respiratory disorders. Any subjects that had abnormal spirometry results according to American Thoracic Society guidelines (1995) or a VO_2max less than 55 ml/kg/min or 3.85 L/min were excluded from the study. In the second session of testing (following a minimum 2 full day rest period), esophageal and gastric balloon catheters were inserted transnasaly and positioned at their respective sites. Participants were first assessed for MIP, MEP and Pdimax and then performed the submaximal constant load exercise test. Pes, Pga, Pdi and ventilatory parameters were monitored continuously throughout the submaximal test while the subjects perception of muscular exertion by the legs and breathlessness was assessed at baseline, at three and six minutes during the test, at test termination and 5 min post test. Immediately following this test, participants were reassessed for MIP, MEP and Pdimax. These reassessments were completed within 5 minutes posttest with 3 maneuvers performed for the MIP and MEP measurements. The average of the 3 values were reported as the post exercise

measures of RM strength. Following a 15-minute resting period, subjects were assessed for CLRB

In the third and fourth testing session, following a minimum of 2 full days rest period between each test, subjects performed two 8km maximum effort time trials. The purpose of these trials was to establish reliability of the baseline time trial information and allow familiarization.

Subjects then underwent sequential randomization into one of two groups: RMT or C.

Training Intervention

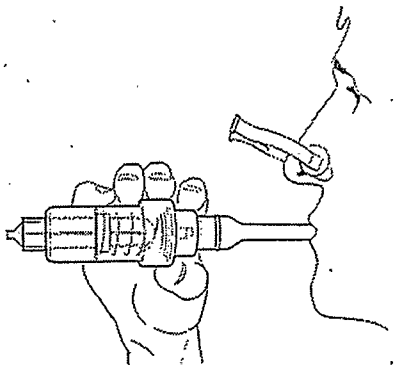


Figure 7: THRESHOLD Inspiratory Muscle Trainer (Respironics Inc., NJ)

Subjects assigned to the RMT group performed RMT for a period of 4 weeks with the THRESHOLD Inspiratory Muscle Trainer (Respironics Inc., New Jersey)(Figure 7). This PR RMT device had been modified by the manufacturers to elicit an adjustable resistance between 15 –95cmH20 and has been shown to be reliable and accurate with respect to the resistance required to initiate airflow (Gosselink, Wagenaar et al. 1996;

Johnson, Cowley et al. 1996). Additionally, flow rates have minimal effect on this resistance (Gosselink, Wagenaar.et al. 1996; Johnson, Cowley et al. 1996). During the 4 week training period, subjects trained 6-days/week, 3 sets/day, 30 breaths/set on their own time with a 1-minute rest between sets. Subjects were instructed to breathe from RV to TLC at a *f* of 35 breaths per minute, a frequency that has shown improvements in athletic performance (Spengler et al, 1996). The inspiratory resistance on the RMT device was set to 50% of the subjects current MIP by a lab technician. This resistance was similar to that used in a recent study by Volianitis et al (2001) in which an improvement in rowing performance was observed. A piece of tamper resistant tape was used to prevent tampering with the device (changing the resistance) and to maintain the subjects naivety regarding the resistance of the device. The resistance set on the device was re-adjusted in accordance to results from repeat MIP tests that occurred at the beginning of each week of the 4-week training period. During each training session, the subjects were instructed to train in the seated position in order to simulate cycling position. The first training session was performed at the University of Calgary Human Performance Laboratory to ensure proper technique.

Subjects in the C group performed sham RMT using a similar protocol as the experimental groups. Sham RMT consisted of a training resistance set to 10% of the subjects MIP (did not change if MIP increased), a *f* set at 35 breaths per minute and a V_T from RV to TLC. Such a resistance is known to elicit insignificant training adaptations (Larson et al, 1988).

Subjects were told that the purpose of the study was to investigate the effects of two different RMT protocols. To maintain subject naivety and expectation of performance improvement, subjects were blinded to their group assignment.

Subjects were asked to keep a detailed training log of their regular exercise training sessions (Appendix F) beginning 2 weeks prior to the start of testing, and a training log of their RMT sessions (Appendix G). Subjects with significant changes in their regular exercise training sessions or with RMT compliance less than 85% were not included in any of the study calculations. Additionally, in order to standardize pre-testing protocols, subjects were asked to abstain from vigorous exercise and consume a normal diet 2 full days prior to each testing sessions. Following the 4 weeks of RMT, the subjects were re-evaluated for the VO_2max test, submaximal test, MIP, MEP, CLRB Pdimax and 8 km cycle time trial. The workloads for the submaximal test and CLRB were the same as the pre-training values. Subjects continued to train with the device at the last adjusted resistance during the post-testing period however, they did not train one day prior to each testing day. The purpose of this training was to maintain any training adaptations over the post-testing duration. Subjects were given a maximum of 7 days to recover from any sickness.

Subjects were required to commit to 11 evaluation sessions during their study involvement. The spirometry, VO_2max tests and submaximal tests took place at the Pulmonary function lab, Rockyview General Hospital. All other tests took place at the Human Performance Laboratory, University of Calgary.

Statistical Analysis

Normality of data was assessed with the Shapiro-Wilk W test for normality. For P-values greater than 0.05, an independent sample t-test was performed between experimental and placebo groups to test for between group differences and dependent sample t-test was performed to determine within group differences. When the assumption of normality was not met, the Wilkoxon Signed-ranks test was used to test for within group differences and the Mann-Whitney test was used to test for between group differences. The variables of interests were:

a. 8km time trial performance
b. VO_2max
c. MAP
d. MIP
e. MEP
f. Sniff Pes
g. Pdimax
h. Sniff Pdimax
i. *f* (last 30sec of submax test)
j. V_T (last 30sec of submax test)
k. Ti/Tot (last 30sec of submax test)
l. CLRB
m. 5min post lactate concentration
n. Perception of breathlessness and end of test
o. Perception of muscular exertion at end of test

CHAPTER FOUR

RESULTS

Subjects

Twenty-six male competitive cyclists (8 triathletes, 8 road cyclists, 9 mountain bikers and 1 speed skater) were initially recruited to participate in this study. One subject was identified as having exercise-induced asthma (EIA) by a fall in FEV1 greater than 15% following the maximal incremental test. However, he was kept in the study because he was unaware that he had such a syndrome and the symptoms did not seem to bother him. Three subjects in the control group did not complete the study (hemorrhoids, pulled groin/nerve problems and lack of interest). Therefore, 23 subjects (13 RMT and 10 placebo) completed the study and were included in the results except where stated. One subject in the RMT group did not perform the post RMT time trial due to a lung infection that lasted about 3 weeks and another subject in the RMT group had trouble performing the MIP maneuvers and was excluded from all the MIP and Pdimax measurements.

Only the CLRB measurement violated the assumption of normality and thus non-parametric statistics was used to analyze this variable. All other variables met the assumption of normality and as such, parametric statistics was used.

The baseline characteristics of the participants that completed the study are presented in Table 6. The placebo and RMT group did not differ in age, height, weight, FEV1 and FVC at baseline measurements. Although in absolute values (L/min), the placebo group had a higher VO_2max than the RMT group (4.50 vs. 4.03), the relative values (ml/kg/min) did not differ significantly (58.2 vs. 55.64) and the baseline time trial

values did not differ significantly between groups. The baseline MIP values for the placebo group and RMT group were 148.73 (20.44) and 125 (22.72) respectively.

Table 6. Baseline Characteristics of the study subjects (mean +/- SD)

	Placebo Group	**RMT Group**
Total Number	10	13
Anthropometry		
Mean Age (years)	30.20 (4.59)	29.61 (6.71)
Subject Weight (kg)	77.85 (7.09)	72.97 (7.90)
Subject Height (cm)	178.20 (8.05)	177.00 (4.65)
Maximal Incremental Exercise		
VO_2max (ml/kg/min)	58.02 (4.34)	55.64 (5.57)
VO_2max (L/min)	4.50 (0.31)	4.03 (0.29)
MAP (W)	339.00 (33.31)	320.77 (21.68)
Resting Pulmonary Function		
FVC (L)	6.49 (1.55)	5.74 (1.05)
FEV1 (L)	4.97 (1.21)	4.65 (0.90)
MIP (cmH20)	148.73 (20.44)	125.00 (22.72) [a]
MEP (cmH20)	182.16(34.81)	185.00 (42.07)
CLRB (s)	75.60 (164.05)	90.00 (98.36)
Time Trial Performance		
8km (s)	854.30 (61.10)	850.58 (42.43) [a]

[a] **n = 12**

RMT Compliance and duration of testing

Compliance to the RMT protocol was excellent in both the experimental and placebo groups. On average, both the RMT and placebo group completed 23 of the 24 sessions. Also, the time to complete the post testing sessions after the 4 weeks of training was 17 (6) days for the placebo groups and 18 (5) days for the RMT group. The resistance of the training device for the placebo group was consistent at 15.8 (0.94) cmH_20 and for the experimental group increased significantly from 65.2 (12.0) cmH_20 to 75.6 (15.7) cmH_20 ($P < 0.001$) by the end of the 4 weeks. Participant's physical activity

during the 4-week training period of the study as measured by number of training hours did not differ significantly between the RMT and placebo (31.83 (12.44) vs. 32.64 (15.28)).

Respiratory Muscle and Pulmonary Function

The RMT group saw a significant increase in MIP, sniff Pes, and CLRB that exceeded that of the placebo group (P = 0.036, 0.031, 0.025 respectively Figure 8, Table 7). No change in MEP, Sniff Pdimax and Muller Pdimax, FVC and FEV1 was observed in either group although sniff Pdi for the placebo group did decrease significantly following training. Repeat measurements of MIP without the Pes and Pga balloon catheters revealed a slight but non-significant increase in the baseline value of both groups (Placebo: 5%, RMT: 4%). There was a significant correlation between MIP and sniff Pes ($r = 0.49$, $P = 0.02$); MIP and sniff Pdi ($r = 0.43$, $P = 0.05$); MIP and Pdimax Muller ($r = 0.49$, $P = 0.02$); sniff Pes and sniff Pdi ($r = 0.45$, $P = 0.03$). The correlation between sniff Pdi and Pdimax Muller was not significant ($r = 0.4$, $P = 0.07$). There was no significant correlation between MIP and height, weight or age ($P>0.05$). A tracing of the MIP, MEP, Pdimax Muller, Sniff Pdi and Sniff Pes can be seen in Appendix G.

Table 7: Measurements of respiratory muscle and pulmonary function before and after 4 weeks of RMT for the placebo (pre (C) and post (C) respectively) and RMT group (pre (E) and post (E) respectively) (mean +/- SD).

	Pre (C)	**Post (C)**	**Pre (E)**	**Post (E)**
MIP (cmH20)	148.73 (20.44)	160.13(27.39)	125 (22.72)	157.02(32.89) [a]*!
MEP (cmH20)	182.16(34.81)	201.26(44.52)	185 (42.07)	178.61 (50.76)
CLRB (s)	75.6(164.06)	94.6(112.40)	90(98.37)[a]	189.25(183.24)[a]*!
FVC (L)	6.49(1.55)	6.21(1.41)	5.74 (1.05)	5.53 (.71)
FEV1 (L)	4.97 (1.21)	4.73 (.89)	4.65 (.90)	4.40 (0.71)
Pdimax (cmH20)	133.5 (31.57)	137.2 (47.05)	118.82 (36.98)	120.74 29.65
Sniff Pdi (cmH20)	128.33 (38.49)	117.3(38.70)*	115.25 (27.34)	112.97 (22.66)
Sniff Pes (cmH20)	108.6 (29.98)	105.6 (27.19)	93.17 (24)	103.92 (29)*!

[a] **n = 12** **(*) p<0.05, (!) delta C vs. delta E p <0.05**

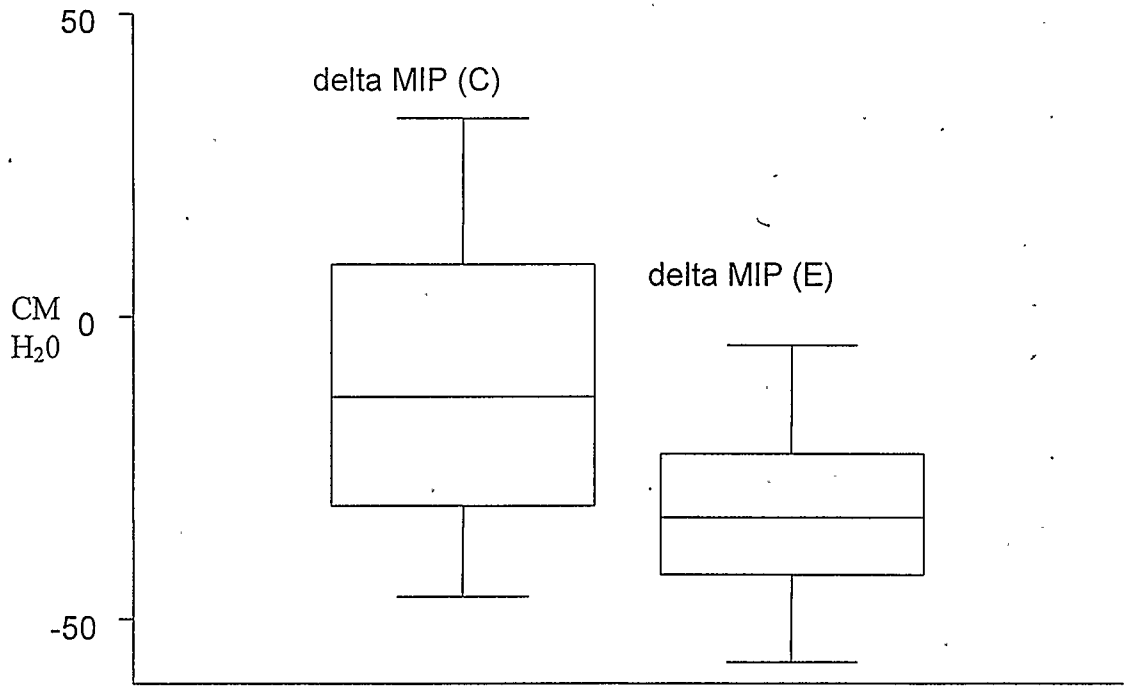


Figure 8. Changes in MIP measurement following the 4-week training period (negative value refers to an increase in strength) delta MIP(C): placebo group, delta MIP(E): RMT group, y-axis: cmH_20

Maximal Incremental Test

Neither the placebo group nor RMT group showed a significant change in VO_2max following the training period (Table 8). The RMT group showed a significant increase in MAP (P = 0.008) but this did not exceed that of the placebo group (P = 0.32).

Table 8: Measurements of the maximal incremental test before and after 4 weeks of RMT for the placebo (pre (C) and post (C) respectively) and RMT group (pre (E) and post (E) respectively) (mean +/- SD).

	Pre (C)	Post (C)	Pre (E)	Post (E)
VO_2max (L/min)	4.50 (0.31)	4.47 (0.37)	4.03 (0.29)	4.19(0.33)
VO_2max (ml/kg/min)	58.02(4.34)	58.50(5.72)	55.64(5.57)	58.50(6.61)
MAP (W)	339.00 (10.54)	339.00 (7.48)	320.77(6.01)	327.69(5.33)*

(*) p<0.05,

Time trial

The second 8 km TT time of the baseline measure for all subjects was slightly faster that the first (864s vs. 855s) but these times did not significantly differ from each other and ICC was 0.88 suggesting good reliability between trials. The placebo groups' time trial performance following the training period decreased significantly (-2.4 +/- 2.0% or – 20.7 +/- 17.0s, P = 0.004, range 0.94% to - 5.0%; Table 9, Figure 9). The RMT group time trial performance remained unchanged from baseline following the training period (-0.4% +/- 2.1% or – 3.6 +/ 18.2s, P = 0.5), range to 3.3% - 4.8%). The improvement in the placebo group exceeded that of the RMT group (P = 0.03). By the end of the 4 weeks, 8 of the 10 placebo subjects improved performance while 7 out of the 12 RMT subjects improved performance (Figure 10A,B).

Table 9: Measurements of the 8km time trial test before and after 4 weeks of RMT for the placebo (pre (C) and post (C) respectively) and RMT group (pre (E) and post (E) respectively) (mean +/- SD).

	Pre (C)	Post (C)	Pre (E)	Post (E)
TT (s)	854.30 (61.10)	833.60 (52.70)*!	850.58 (42.43) [a]	847.00(38.55) [a]

[a] **n = 12 (*) p<0.05, (!) delta C vs. delta E p <0.05**

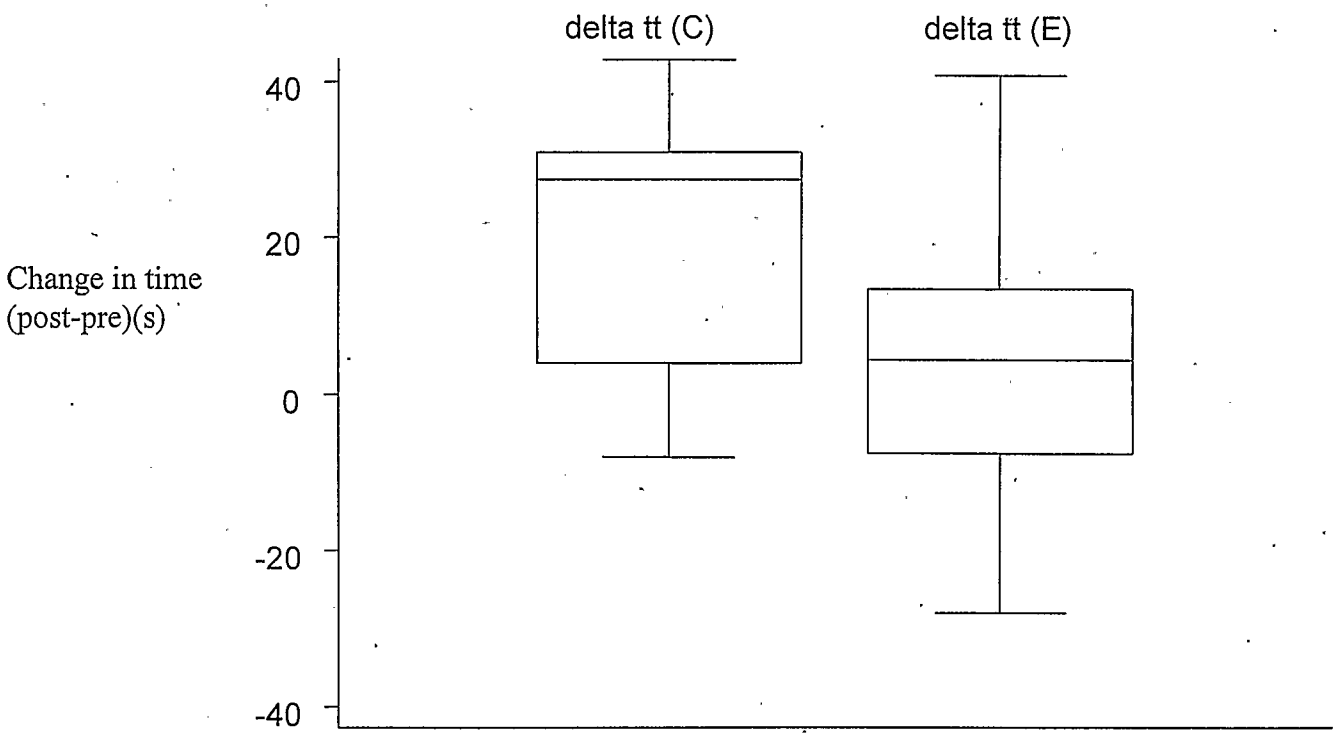
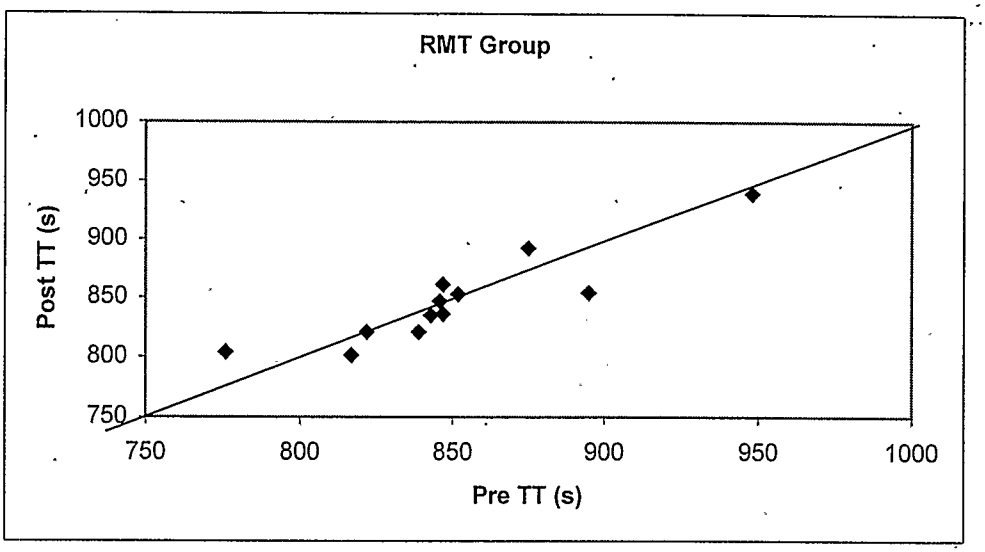


Figure 9. Comparison of time trial performance: Placebo (delta tt (C)) vs. RMT (delta tt (E))

A



B

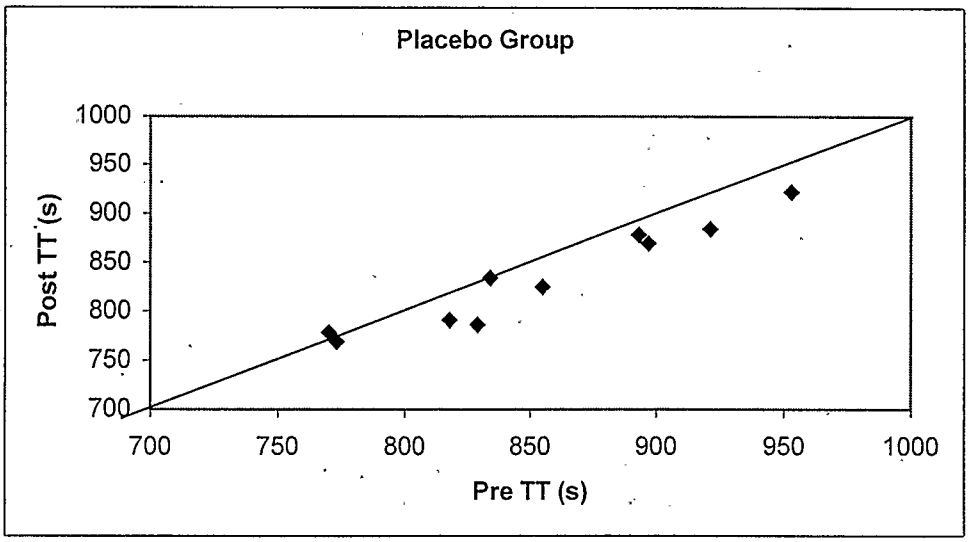


Figure 10. Effects of 4 weeks of RMT (A) or placebo (B) on 8km time trial performance. Points lying below the line of identity (diagonal line) indicate an improvement from initial test.

Response during Submaximal Test

Baseline values for the submaximal constant load test duration and test intensity were not significantly different between the placebo and RMT group (Table 10). Following training, the placebo group saw a reduction in *f* that exceeded that of the RMT group (P = 0.04). Both groups saw a significant reduction in blood lactate and increase in V_T but these values were not significantly different between groups. No change in V_E, Ti/Tot, VO_2, breathlessness or perceived exertion of the legs was observed in either group. Two RMT subjects and 1 placebo subject did not reach their initial submaximal test duration. Two measurements of breathlessness were not obtained in the placebo group and 1 measurement of breathlessness was not obtained in the RMT group.

Table 10: Measurements of variables during the last 30 seconds of the submaximal constant load test before and after 4 weeks of RMT for the placebo (pre (C) and post (C) respectively) and RMT group (pre (E) and post (E) respectively) (mean +/- SD) (Lactate taken 5min post test, Borg measures taken immediately at test termination)

	Pre (C)	Post (C)	Pre (E)	Post (E)
Test duration (s)	406.3(46.42)		462.15(35.61)	
Test intensity (W)	303.5(42.10)		291.53(18.18)	
Ti/Tot (%)	49.27(2.65)	50.68(1.91)	49.10(0.94)	48.53(1.66)
V_E (L/min)	150.05(40.15)	147.95(33.45)	153.56(25.22)	154.42(6.27)
V_T (L)	3.11(0.27)	3.43(0.16)*	3.01(0.12)	3.10(0.12)*
***f* (b/min)**	49.18(3.79)	43.59(3.36)*!	51.60(2.36)	50.19(1.99)
Lactate (5minpost)	11.2(1.03)	9.11(1.03)*	12.68(0.81)	10.92(0.68)*
Borg Breathlessness	4.75(1.28) [b]	4.50(1.92) [b]	6.83(1.99) [a]	6.58(0.65) [a]
Borg Leg	6.62(2.06) [b]	5.5(1.41) [b]	7.75(2.17) [a]	6.91(1.92) [a]
VO_2	4.10(0.75)	4.12(0.61)	3.96(0.25)	4.05(0.27)

(*) P<0.05, (!) delta C vs. delta E p <0.05, [b]:n= 8, [a]:n=12

CHAPTER 5

DISCUSSION

In an attempt to help gain a better understanding of the effects of RMT on aerobic performance, 4 weeks of PRD RMT on competitive male cyclists was conducted using an experimental design that included: a true placebo group, measurements of global RM and diaphragm strength, control of V_T and *f* during the training program. The present study found that RMT improved global RM strength and endurance but failed to improve aerobic performance in moderately trained cyclists. The secondary outcome measures will be discussed before the primary outcome measure (8 km time trial) because the secondary outcome measures provide some of the background for the discussion of the primary outcome measure.

Respiratory Muscle Strength and Endurance

MIP and sniff Pes were used to estimate global inspiratory muscle strength in this study. The RMT group showed significant improvements in MIP and sniff Pes that exceeded that of the placebo group. These results support previous literature showing that 4 weeks of RMT with a pressure resistance device at a resistance equivalent to 50% of MIP increases maximal global inspiratory muscle pressure (Hart, Sylvester et al. 2001; Sonetti, Wetter et al. 2001; Volianitis, McConnell et al. 2001; Romer, McConnell et al. 2002).

Although the MIP maneuver is a simple, non-invasive method of estimating overall RM strength, it is volitional in nature, can be difficult to coordinate in certain individuals and may require a learning period to obtain reliable maximal values (Laroche, Mier et al. 1988; Wen, Woo et al. 1997; Maillard, Burdet et al. 1998; Pacia and Aldrich

1998; McConnell and Copestake 1999; Volianitis, McConnell et al. 1999). Repeat measurements performed on all subjects before and after the training period without the distraction of the balloon catheters revealed a slight but nonsignificant increase in baseline values in both groups (C: 5%, E: 4%). Furthermore, the RMT group exhibited an increase in MIP that was significantly greater than the placebo group supporting a true improvement in strength. In agreement with Wen et al (1997), the results from this study suggest that the use of 20 maneuvers to measure MIP minimizes the influence of learning.

A majority of the subjects did not appear to have difficulty coordinating the MIP maneuver. However, one RMT subject exhibited high between and within testing session variability (delta MIP value fell well outside the 95% CI). This exemplifies the possible difficulties experienced by certain individuals when performing this maneuver.

Sniff Pes is a more familiar maneuver used to estimate global inspiratory muscle strength particularly in patients with RM weakness (Laroche, Mier et al. 1988). The measurement of sniff Pes in the participants of this study exhibited much less variability than the measurement of MIP (about half the SD). This agrees with previous studies (Laroche, Mier et al. 1988; Koulouris, Mulvey et al. 1989). However, the absolute values of sniff Pes were much lower than the values obtained by MIP measurements, which is in contrast to that reported by Laroche et al (1988) and Hart et al (2001). Theoretically, subjects that are able to coordinate the MIP maneuver properly and maximally should obtain a higher value than that provided by the sniff Pes maneuver because the former is an quasi-isometric maneuver (less shortening of the inspiratory muscles) while the latter is a dynamic maneuver (Miller, Moxham et al. 1985). The higher values obtained by the

MIP measurement in the moderately trained subjects used in this study may be a result of their ability to coordinate the maneuver properly and maximally.

Following the training intervention, the RMT group showed a 25% improvement in MIP (19% using the repeat measurement) and an 11.5% improvement in sniff Pes. The smaller increase in MIP in the repeat measurement is due predominately to the higher baseline value (125-130cmH20) and was likely caused by a small learning effect. This increase in MIP lies within the range of the 8-47% increase reported in previous studies investigating the effect of RMT on RM strength (Leith and Bradley 1976; Hanel and Secher 1991; Inbar, Weiner et al. 2000; Hart, Sylvester et al. 2001; Sonetti, Wetter et al. 2001; Volianitis, McConnell et al. 2001; Romer, McConnell et al. 2002). This suggests that the RMT regime used in this study is effective in improving RM strength and comparable to other studies. Few studies have used sniff Pes to estimate global inspiratory muscle strength following a RMT program. The 11.5% improvement in sniff Pes is in agreement with Wanke et al (1994a,b) but in contrast to Hart et al (2001) where no significant improvement was reported. It is possible that the small sample size used in the latter study (6 subjects/group) increased their chances of incurring a Type II error and missing the detection of a difference if it existed.

Since the RM's adapt in a similar manner to the skeletal muscles, the improvement in RM strength in this study is likely due to an improved neural and intramuscular adaptation. Neural adaptations to training are predominately responsible for the initial improvements in limb muscle strength following training although concomitant intrasmucular adaptations have also been observed as early as 2 weeks (Sale 1988; Staron, Karapondo et al. 1994). The proposed neural adaptations have been

described in detail in the introduction, but briefly they include: more efficient recruitment, increased neural activation, motor unit synchronization, and excitability of the alpha motor neurons and/or motor end plates and decreased Golgi tendon organ inhibition (Staron, Karapondo et al. 1994). The acute intramuscular adaptation likely involves an increase in the percentage of type IIb fibers and conversion of fast fiber types (type IIb → IIa) (Staron, Karapondo et al. 1994).

The results of this study suggest that 4 weeks of PRD RMT can increase global inspiratory muscle strength as measured by both MIP and sniff Pes in moderately trained cyclists. The smaller variability observed in the sniff Pes might make it more sensitive to changes over time and the familiarity of the maneuver might make it less susceptible to a familiarization effect.

MEP did not increase following the 4-week RMT period. The RMT device used in this study only had an inspiratory resistance and the only external resistance was that of the mouthpiece itself (~210mm^2). The mouthpiece may add some additional resistance particularly when breathing at higher flow rates, but this additional resistance is likely to be small. Since MEP was unchanged following RMT, the external resistance imposed by the RMT device mouthpiece is not substantial enough to increase expiratory muscle strength.

The non-significant change in the Pdimax of the RMT group when measured using a Muller or sniff maneuver suggests than RMT does not increase the force produced by the diaphragm during a maximal quasi-isometric or dynamic maneuver. This result is in agreement with Hart et al (2001) where no significant improvements in twitch Pdi or sniff Pdi were observed but is in contrast to Suzuki et al (1996) and Wanke

et al (1994) where a significant improvement in Pdimax measured using a combined maneuver and sniff maneuver respectively was observed. There are two possible reasons for these conflicting results.

First, a weakness of the study by Suzuki et al (1996) was that Pdimax was not measured in the control group, so it is unknown whether this improvement was due to a learning effect or an improvement in diaphragm strength. Since the combined maneuver is difficult to perform in naïve individuals, there is a greater likelihood for a learning effect than the other maneuvers used to measure Pdimax (Chan, Cheong et al. 1996).

Second, a major limitation of using the Muller or sniff maneuver to measure Pdimax is that the weaker rib cage may limit the maximal pressures generated by the stronger diaphragm (Hershenson, Kikuchi et al. 1988). Rather than measuring the maximal force generating capacity of the diaphragm, these maneuvers are measuring the force produced by the diaphragm during a maximal inspiratory effort, which is generally less than the maximal force generating capacity of the diaphragm (Laporta and Grassino 1985; Hershenson, Kikuchi et al. 1988). If indeed the weaker rib cage muscles limit the maximal force production of the diaphragm, increased diaphragm force would be expected with stronger rib cage muscles. This is a possible explanation for the increased sniff Pdimax observed by Wanke et al (1994) where their increase in sniff Pes was much larger that what was reported in this study (38% vs. 11%). The sample of COPD patients used in their study may have had RM weakness particularly at RV and this is supported by low baseline values of sniff Pes and sniff Pdi (sniff Pes 60cmH20 and sniff Pdi 80-90) (Wanke, Formanek et al. 1994). This may have predisposed them to a larger window of improvement in rib cage muscle strength and diaphragm strength than the moderately

trained cyclists used in this study. In contrast, the 11.5% increase in sniff Pes in this study may have not been large enough to increase the diaphragm's activation during the maneuver.

The significant decrease in the placebo group's sniff Pdi following RMT was unexpected. It is unlikely that the RMT in this group impaired the diaphragm's force production during maximal inspiratory pressures especially since Pdimax Muller did not follow a similar pattern. A more likely explanation for this result is the inherent variability in Pga observed during a sniff maneuver. Verin et al (2002) reported a significant variation in sniff Pdi during diaphragmatic, extradiaphragmatic and natural sniff maneuvers. This variability was a result of changes in Pga since the Pes remained consistent during these different maneuvers and was concluded by the authors to be attributed to varying degrees of diaphragm and abdominal muscle activation. The results of this study exemplify the limitation of using sniff Pdi to measure diaphragm strength during a maximal inspiratory maneuver.

In summary, RMT increases the rib cage muscles ability to generate larger maximal inspiratory pressures but does not alter the diaphragm's contribution.

The significant correlation between MIP and sniff Pes in this study provides support for the theoretical relationships expected between these variables and is in agreement with the results from Koulouris et al (1989). During a Muller maneuver against an occluded mouthpiece, the change in mouth pressure will be close to Ppl when there is little or no airflow during the effort (Pacia and Aldrich 1998). Since this assumption is invalid if the glottis closes or if there is significant suction by the cheek and pharyngeal muscles, a small leak is introduced into the mouthpiece to prevent the

development of spuriously strongly negative airway pressure (Pacia and Aldrich 1998). The measure of Pes provides a more direct estimate of Ppl because there is no influence from glottis closure or cheek muscles on the values of Pes obtained. Possible reasons for the variance MIP not explained by sniff Pes may be attributed to technique issues such as difficulties in coordinating the MIP maneuver, the volitional nature of the tests and the hindrance of a nose clip. Additionally, the variable effect of the cheek muscles on pressure development may also add to the unexplained variance. A significant correlation would also be theoretically expected between sniff Pdi and Pdimax Muller because they are both measuring the force produced by the diaphragm even though a slightly different maneuver is used. In contrast to theoretical expectation, this relationship was not significant (P= 0.07) in this study. This might be explained by the same factors that contribute to the unexplained variance between the MIP and sniff Pes maneuver. Additionally, in contrast to Pes and MIP, abdominal contraction will have a substantial effect on Pga and Pdi and contributes to the substantial variability reported in this measurement (Laporta and Grassino 1985; Miller, Moxham et al. 1985; Verin, Delafosse et al. 2001).

The RMT group demonstrated a significant improvement in CLRB that exceeded that of the placebo group suggesting an increased ability to breath against a constant resistance equivalent to 50% of MIP following RMT. This is in agreement with Inbar et al (1999) who reported significant increases in ITL following RMT. This improvement is probably due to neuromuscular adaptation received from RMT that is known to be

responsible for the initial increase in strength and endurance following training (Sale 1988).

Metabolic and Ventilatory Parameters

In agreement with previous literature, no significant between group improvements in VO_2max, MAP, FVC and FEV_1 was observed following RMT (Inbar, Weiner et al. 2000; Sonetti, Wetter et al. 2001; Volianitis, McConnell et al. 2001; Romer, McConnell et al. 2002). This finding in not unexpected since VO_2max is insensitive to small changes in performance improvements (Daniels, Yarbrough et al. 1978; Foster, Schrager et al. 1996).

Both the RMT and placebo group exhibited significant reductions in their blood lactate concentrations, but these concentrations were not significantly different between groups. This is in agreement with previous literature that have shown reductions in blood lactate that have not exceeded that of the control/placebo group (Spengler, Roos et al. 1999; Markov, Spengler et al. 2001; Sonetti, Wetter et al. 2001; Stuessi, Spengler et al. 2001; Volianitis, McConnell et al. 2001; Romer, McConnell et al. 2002; Williams, Wongsathikun et al. 2002). These reported reductions in blood lactate observed in this study and in other studies might be partly due to improved overall physical training and not just from RMT training per se. Alternatively, any reduction in blood lactate from RMT is likely to be small in magnitude and much larger sample sizes would be required to detect a between group difference in this measure if it really exists.

The perceived exertion by the legs and the perception of dyspnea were not significantly different following RMT in either group although the mean values for these

measures were slightly lower in both groups after training. The results of this study are in agreement with previous literature (Suzuki, Yoshiike et al. 1993; Sonetti, Wetter et al. 2001; Volianitis, McConnell et al. 2001; Romer, McConnell et al. 2002). The theoretical basis for expecting a reduction in the perception of dyspnea is that an improvement in the force-generating capacity of the RM following RMT would decrease their relative tension for a given level of ventilation and delay RM fatigue (Romer, McConnell et al. 2002). Reducing the work of the RM and delaying their fatigue may redirect greater blood flow to the periphery. Improved blood flow to the exercising muscles may be accompanied with increased oxygen delivery, reduced lactate production and reduced periphery effort sensation (Harms, Wetter et al. 1998; Harms 2000; Romer, McConnell et al. 2002). Similar to blood lactate, the slight reductions in mean values of these measures following RMT may be due to overall physical training rather than RMT.

Time trial performance

The primary outcome measure of this study was the 8km cycle time trial. There was a significant 2.4% improvement in the placebo group time trial performance as compared to a non-significant change of 0.4% in the RMT group time trial performance ($P = 0.03$) following 4 weeks of PRD RMT. The improvement in the placebo group exceeded that of the RMT group ($P = 0.03$).

Only three other studies have investigated the effects of RMT on performance using true placebo groups (Sonetti, Wetter et al. 2001; Volianitis, McConnell et al. 2001; Romer, McConnell et al. 2002). Two of these studies have reported significant between group improvements in the RMT group while the third study did not detect any between

group improvement. This study is the fourth study in this group and fails to show any benefit from RMT on performance. Studies that have not used true placebo groups in their experimental design have been criticized for having weak internal validity (Sonetti, Wetter et al. 2001; Romer, McConnell et al. 2002). As reported earlier, expectation of improvement alone can account for a 3-5% improvement in time trial performance. Studies reporting improvements in performance following RMT that have not used a true placebo group may be important for hypothesis generating, but are not adequate for making any meaningful conclusions. The placebo group in this study satisfied the criteria established by Ojaunen (1994) which are: 1) the placebo needs to be inert; 2) it should generate expectations, involvement, subject utility, and be meaningful to the subjects. The placebo group trained with the same device using the exact same protocol as the experimental group except that the resistance was set at 10% of MIP. To maintain subject naivety of the resistance, the device was completely covered with a 3M™ tamper resistant security tape such that subjects in neither group knew what resistance they were training at and both groups had equal contact time with the technician. The only information given to the subject was that they were randomized to two groups, but the nature of the groups was not revealed until the completion of the study.

Of the 3 previous studies utilizing a RCT design with a true placebo group and time trial performance outcome measure, the 2 studies that reported a significant improvement used subjects that were highly trained and homogenous in characteristics (trained national team caliber rowers, trained cyclists VO_2max ~64ml/kg/min) (Sonetti, Wetter et al. 2001; Volianitis, McConnell et al. 2001; Romer, McConnell et al. 2002). These types of subjects are likely to have much less variability from internal and external

sources (motivation, training, familiarization) and thus any improvement from RMT is less likely to be masked. This study however, used moderately trained subjects (VO_2max ~55-58ml/kg/min) that had a similar training status as the subjects used by Sonetti et al (2001) (VO_2max ~55ml/kg/min). These tests had more variability than the tests performed by highly trained subjects (1.8-2.7% vs. 1.0-1.9%) and failed to show any improvement in time trial performance following RMT. Therefore, the level of training is likely a significant factor in determining the success of RMT on improving time trial performance. Highly trained athletes are more likely to demonstrate an improvement in performance following RMT than moderately or untrained athletes because they are less susceptible to internal and external sources of variability.

A distinct difference between the current studies is that the subjects used by Romer et al (2002) and Volianitis et al (2001) had a much lower initial RM strength than the subjects used in this study or in the study by Sonetti et al (2001) (100cm H20 vs. 129-168cmH20). It is possible that subjects with weaker RM's had larger room for improvement in RM strength and experienced greater RM fatigue than individuals with stronger RM's. This might make these subjects more likely to benefit from RMT and explain the larger improvements in RM strength reported by these studies (28%& 45% vs. 8% & 25) (Coast, Clifford et al. 1990; McConnell, Caine et al. 1997).

A possible explanation for the time trial results obtained in this study is that the placebo group in this study underwent RMT adaptations that differed from those of the experimental group and had a greater effect on performance. Although several studies have demonstrated that PRD RMT below 15% of MIP does not improve RM strength or

endurance, the breathing pattern adopted during these training regimes are generally slow and protracted (Sonetti, Wetter et al. 2001; Volianitis, McConnell et al. 2001; Romer, McConnell et al. 2002). In contrast, this study had the placebo group train using a dynamic breathing pattern, similar to that used during IH RMT. The only major difference initially expected between the placebo and RMT group was the resistance at which training occurred. However, it is also likely that the two groups differed in the V_T at which they trained such that the placebo group adopted a larger V_T than the RMT group because they had a smaller resistance to overcome. A larger V_T would have resulted in a V_E that was closer to that used during IH RMT. This study raises the possibility that the *f* (speed of the inspiratory contraction) and V_T adopted during RMT may be more important to improving RM performance than the resistance at which training occurs. This has been investigated in skeletal muscle where a greater improvement in movement velocity capabilities were observed in subjects that trained at 30% compared 80% of their 1 repetition maximum in the squat (McBride, Triplett-McBride et al. 2002). Additionally, the improvements in the velocity of shortening of skeletal muscles are specific to the training workload (Moritani 1993). For example, training with no resistance increased the velocity of shortening at no resistance while training at a resistance equivalent to 50% of maximum increased the velocity of shortening at this workload with smaller improvements at other workloads. These studies suggest the importance of the specificity of the movement used during RMT and provides theoretical support for the improvements in performance following IH RMT. During exercise, the ability for the RM's to develop forces/pressures at high shortening velocities is important for increasing V_E. This is particularly important at higher lung volumes

where the force generating capacity of the RM is reduced. The better adapted the RM's are to generating forces at high shortening velocities when utilizing larger lung volumes, the less likely they are to become fatigued and impair performance. Given the evidence of the benefits of velocity specific training, the placebo group may have received a training adaptation that exceeded that of the RMT group. This added adaptation might have resulted in a larger improvement in the velocity of shortening and power output over a larger range of lung volumes. In contrast, the higher training loads used by RMT group contrasts the principle of specificity because the RM's never have to achieve such high pressures during exercise and as such this training might have comprised an improvement in the velocity of shortening. If this group did use a smaller V_T to maintain the set *f*, adaptations at larger lung volume would have also been compromised. Adaptations at higher lung volumes would appear to benefit the most from RMT because these muscles are used less frequently at these lung volumes. The significant decrease in *f* and increase in V_T observed in the placebo group provides support for a possible training adaptation in the placebo group that exceeded that of the RMT group. A reduced *f* and increased V_T would suggest a decreased work of breathing that may utilize a smaller proportion of the overall blood flow and produce smaller amount of lactic acid (Volianitis, McConnell et al. 2001; Romer, McConnell et al. 2002). The altered breathing pattern observed in the placebo group is in agreement with the studies by Romer et al (2002) and Volianitis et al (2001) where a trend towards a less tachypneic breathing pattern was observed following RMT.

Another possible explanation for the results of this study is that they may be because of chance. Although statistical methods are useful for decision making, when alpha is set at 5%, there is a 0.05 probability of rejecting the null hypothesis when it is true. Therefore, although small, there is a possibility that our samples fell on the extremes (tails) of the normal distribution curve.

The above discussion has centered on various reasons why the RMT group did not improve their 8km performance time trial. To provide a comprehensive discussion, one other possible reason for the results obtained in this study is that RMT does not improve aerobic performance. From a theoretical perspective, an improvement in aerobic performance caused by RMT could be from a decreased work of breathing and an attenuation of RM fatigue. The existence of RM fatigue following aerobic exercise is still conflicting and is likely influenced by fitness level, motivation exercise duration and exercise intensity (Loke, Mahler et al. 1982; Bye, Esau et al. 1984; Coast, Clifford et al. 1990; Nava, Zanotti et al. 1992; Johnson, Babcock et al. 1993; Mador, Magalang et al. 1993; McConnell, Caine et al. 1997; Perret, Pfeiffer et al. 1999; Volianitis, McConnell et al. 2001; Romer, McConnell et al. 2002). These studies suggest the possibility that the RM's do not fatigue following exercise and as a result, there is no subsequent vasoconstriction of blood flow to the periphery. Since V_E, VO_2, blood lactate, perceived exertion by the legs and the perception of breathlessness did not change following RMT in this study, it might also be suggested that the reported adaptations of the RM's to whole body endurance training are sufficient enough to overcome any limiting effect they might have on performance (Robinson and Kjeldgaard 1982; Coast, Clifford et al. 1990).

Thus, any subsequent improvement in RM strength and endurance following RMT will not provide any additional benefits in performance.

Although subjects were randomly assigned to either a placebo or RMT group, it is possible that the groups differed in their regular physical activity during the study and the improvement in the placebo group was a result of increased physical activity. However, this is unlikely because physical activity logs during the study period were consistent between groups with respect to the number of training hours. The intensity level or modality of the workouts were not quantified leaving the possibility that these parameter may have differed between the groups and thus accounted for improvements in performance. Future studies should quantify the influence of training intensity and modality in the training logs to minimize the confounding effects of these parameters on performance improvement.

A potential critique of the study methods is that the training duration was not long enough to induce training adaptations. However, the improvements in RM strength measured by MIP in this study were similar in magnitude to other studies that have shown improvements in performance. Furthermore, although the study by Volianitis et al (2001) used a training program that lasted 11 weeks in duration, an improvement in RM strength and rowing performance was observed within 4 weeks of training, and 7 additional weeks of training did not result in any further increase in RM strength or time trial performance.

Another possible criticism of this study is whether an 8km time trial is an adequate outcome that would benefit from RMT. Studies showing improvements in performance that have used a placebo group have been conducted at a variety of intensities that ranged from 72% of MAP to 100%of VO_2max and have shown evidence of RM fatigue when measured by MIP. The 8km time trial used in this study was performed at an intensity equivalent to about 95% of VO_2max and thus within the range of these previous studies. Thus, the intensity of exercise used in this study has the potential to be improved by RMT and is likely not the reason for contrasting results.

Limitations of study

As mentioned previously, the training status of the study participants may be an important determinant of the effectiveness of RMT on performance. The two studies that have shown performance improvements when using a RCT design with a true placebo group used highly trained rowers and cyclists. Highly trained athletes are likely to exhibit less variability in performance outcome measures for several reasons including: 1) familiarity with performance test of interest (i.e. time trial) so there is less of a learning effect; 2) are highly motivated; 3) reached the flatter part of the training curve such that there is less influence from physical activity on performance. It is possible that the variability from these factors masked any influence of RMT on performance in the subjects used in this study. Clearly, the between subject variability in time trial performance and MIP measurements in this study were larger that that reported by the other 2 studies.

The measurements of diaphragm strength used in this study were limited to measuring the muscle's force generation during maximal inspiratory pressure. It has been suggested that the diaphragm is not maximally activated during such a measurement and is possibly limited by the strength of the ribcage muscles (Hershenson, Kikuchi et al. 1988; Nava, Ambrosino et al. 1993). Consequently, it was not possible to discern whether maximal diaphragm strength was improved by RMT. The combined maneuver with feedback and bilateral phrenic nerve stimulation are two other methods that can be used to provide information maximal diaphragm strength. The combined maneuver described in the introduction provides a more accurate estimation of Pdimax (Laporta and Grassino 1985) but is still volitional in nature, requires the hindrance of a mouthpiece and is difficult to coordinate in novice subjects. Bilateral phrenic nerve stimulation is a more objective method to measure Pdimax because it does not rely on subject motivation and/or coordination and permits the measure of maximal diaphragm activation. Unfortunately, there is no similar objective measurement of rib cage muscle activation because they are innervated by several nerves. Therefore, MIP and sniff Pes are still the best methods to assess RM strength although they both have several limitations that have been discussed previously.

An improvement in RM performance from the increased *f* that was used during the RM training regime was indiscernible. One possible adaptation to such a breathing pattern would be an increased velocity of shortening of the muscles. It is well accepted that skeletal muscle decreases its ability to generate force with increasing velocity of contraction. With ventilation rates exceeding 200L/min in highly trained subjects at maximal exercise, increasing the RM's velocity of shortening for a given pressure

generation would enable one to maintain higher V_T during increased *f*. Recently, Romer et al (2002) was the first study to measure inspiratory flow rate at 30% of MIP. This study reported a significant improvement in this measure following RMT that was not observed in the placebo. It is likely that the subjects in this study also improved their velocity of shortening during an inspiratory effort but whether the breathing pattern used during the training in this study results in greater improvement has yet to be investigated.

The three participants that dropped out of the study were in the placebo group. An argument can be made that these subjects dropped out because they thought they were in the placebo group or did not believe they were receiving any training adaptations and thus the validity of this study may be comprised. It is unlikely that these dropouts were a result of these concerns. First, one subject dropped out before he was even told what group he was randomized to because he was unhappy with his VO_2max result. Another subject dropped out half way during the training intervention because his doctor attributed his development of hemorrhoids to the training device. The third subject incurred a serious groin injury just prior to his follow-up testing that prevented him from performing any sort of physical activity for just over 4 weeks.

Conclusions

The results of the 8km time trial suggests that 4 weeks of PR RMT does not improve aerobic performance in moderately trained cyclists. This is in agreement with current research findings by Sonetti et al, (2001). While, this type of RMT does improve global RM strength and endurance it fails to increase the diaphragm's contribution to

maximum inspiratory efforts. This is also in agreement with current research findings (Inbar, Weiner et al. 2000; Sonetti, Wetter et al. 2001; Volianitis, McConnell et al. 2001; Romer, McConnell et al. 2002).

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Appendix A: Recruitment Flyer

Subjects are needed for a study examining the effects of **RESPIRATORY MUSCLE TRAINING** on **ATHLETIC PERFORMANCE**.

Criteria:

- Males 18 to 39 years old
- Training with a club (cycling, triathlon, speedskating) for a minimum of 1 year prior to the beginning of the study and/or competition experience
- No chronic respiratory diseases (asthma, COPD, cystic fibrosis, etc.).

Study Involvement:

- Recruited subjects will undergo VO_2max testing, submaximal constant load testing, lactate testing, time trial testing
- Respiratory muscle training for 4 weeks
- Minimal impact on your current training program

At no cost to subject:

- Physiological testing with a detailed analysis of your current training status (VO2max, Anaerobic threshold, max heart rate, maximum aerobic power etc.)
- Identifying areas of weakness for improvement

Please contact **Sidd Thakore at 220-8949** or email at **sthakore@ucalgary.ca** if you have any question regarding this study or if you would like to participate in this study.

Appendix B: Phone Interview Questionnaire

Research Study Phone interview Question:

- How did you hear about the study?
- What type of training background do you have? (road or mountain biking)
- What club have you trained with? (how long, at what level)
- Have you competed at all? (What type of competitions)
- What is your age?
- Do you have any chronic respiratory diseases that you are aware off (asthma…)?
- What type of bike do you own (road or mountain bike)
- For the duration of the study, you will not be allowed to use your rear tire for any other activities. You will be given $15 towards the purchase of a tire for the study.
- If mountain bike, required to use slicks for testing
- Have you ever been on rollers before? (If not, would you be willing to come in an do a practice trial)
- Have you ever ridden 8km before? (If not, would you be willing to come in and do a practice trial)

If so far OK

- Will be performing a VO2max test on day 1, if your VO2max is below 55ml/kg/min, you will be excluded?
- If are identified to have exercise induced asthma, you will be excluded

Outline of Study:

- It is a training intervention, thus you will be required to commit about 2months to this study. (Yes or No)
- 4wks will consist of RMT intervention, using a portable device on your own time, ~5min/day (yes or no), 1/week for 4wks of training.
- Random assignment
- Required to perform VO2max test (Yes or No)
- Required to perform breathing tests to assess respiratory muscle strength (tiny catheters to esophagus)
- Required to perform a submaximal constant load test.
- Required to do 8km time trials on rollers, front fork fixed.
- Required to modify training schedule such that there is no maximal exercise (including weights) performed within 48 hours of test time.
- Required to keep a training log workouts and RMT training during the study duration and 2 week prior to study
- Required to maintain a consistent training program during the study time period and 2 weeks before.

Book Times with me.

- Book all 4 days at once.
- Book Block Times

Appendix C: Physical Activity Readiness Questionnaire

Revised 1994 **Physical Activity Readiness Questionnaire (PAR-Q)**

YES NO

_____ _____ 1. Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?

_____ _____ 2. Do you feel pain in your chest when you do physical activity?

_____ _____ 3. In the past month, have you had chest pain when you were not doing physical activity?

_____ _____ 4. Do you lose your balance because of dizziness or do you ever lose consciousness?

_____ _____ 5. Do you have a bone or joint problem that could be made worse by a change in your physical activity?

_____ _____ 6. Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?

_____ _____ 7. Do you know of any other reason why you should not do physical activity?

NOTE: 1. This questionnaire applies only to those 15 to 69 years of age.

2. If you have temporary illness, such as a fever or cold, or are not feeling well at this time, you may wish to postpone the proposed activity.

3. If you are pregnant, you are advised to discuss the "PARmed-X for Pregnancy" form with your physician before exercising.

4. If your health changes so that you then answer YES to any of the above questions, tell your fitness or health professional. Ask whether you should change your physical activity plan.

I have read, understood and completed this questionnaire.

SIGNATURE_____DATE_____

SIGNATURE OF PARENT_____
OR GUARDIAN (for participants under the age of majority)

Witness_____Date_____

Informed use of the PAR-Q: The Canadian Society for Exercise Physiology, Health Canada, and their agents assume no liability for persons who undertake physical activity, and if in doubt after completing this questionnaire, consult your doctor prior to physical activity.

Appendix D: Subject Consent Form

UNIVERSITY OF CALGARY

FACULTY OF KINESIOLOGY
SPORT MEDICINE CENTRE

Consent Form

Research Project Title: The effects of respiratory muscle training on athletic performance in trained cyclists.

Investigators: Dr. Victor Lun, Sidd Thakore, Dr. Stephen Norris, Dr. Gordon Ford
Sponsor: Respironics Inc.; Sport Science Association of Alberta; University of Calgary Thesis Research Grant.
This consent form, a copy of which has been given to you, is only part of the process of informed consent. It should give you the basic idea of what the research is about and what your participation will involve. If you would like more detail about something mentioned here, or information not included here, please feel free to contact **Sidd Thakore at 220-8949**. Please take the time to read this carefully and to understand any accompanying information.

Purpose of the research: This research is intended to determine the effects of respiratory muscle training on athletic performance in trained cyclists.

Measurement and Procedures: Male volunteers between the ages of 18 - 39 from local triathlete and cycle clubs who have been training for a minimum of 1year, have no chronic respiratory diseases and are willing to perform the tests and training program described below will qualify for the study. Subjects will be required to fill out a ParQ form. Subjects with any positive response on the ParQ form must be evaluated by a physician for clearance to participate in the study.
Additionally, your voluntary participation in this study will require the following:

1. Abstain from vigorous exercise 2 full days prior to each testing session
2. NO caffeine beverages at least 6 hours before each test session
3. NO alcoholic drinks 6 hours before each test session
4. NO smoking 2 hours before each test session.
5. NO food 2 hour before each test session
6. Two incremental cycle ergometer tests to exhaustion to determine maximal aerobic power/VO_2max; four 8km time trials to measure performance; two submaximal constant load exercise tests; two pulmonary function tests including maximal flow-volume loops; seven assessments of inspiratory muscle strength; two assessments of diaphragm strength; two assessments of inspiratory muscle endurance; four assessments of expiratory muscle strength; four weeks of respiratory muscle training.

7. Detailed training log of your regular exercise training sessions beginning 2 weeks prior to the start of testing, with no drastic changes in your training regime during this time
8. Training log of your RMT sessions

Maximal aerobic power/ VO_2max will be determined via an incremental test performed on an electronically braked cycle ergometer starting at 150 watts and increasing by 30 watts every three minutes until the technician observes the anaerobic threshold. Thereafter, the increase will be 15 watts every minute until exhaustion. Throughout the test, your expired air will be collected and analyzed for oxygen and carbon dioxide content. This procedure requires that you breathe room air through a mouthpiece, which is connected to a hose that collects your expired air. Subjects with a VO_2max measurement less than 55ml/kg/min will be excluded from the study. Both before the VO_{2max} test and 5-15 minutes after the VO_{2max} test, subjects will have spirometry assessed (maximal flow-volume loop).

8km cycle time trial will be performed in the Human Performance Lab, University of Calgary on Kreitler rollers with subjects using their own cycles. Heart rate will be monitored every minute with an electronic heart rate monitor. Subjects must not use their rear tire for any riding other than the four time trials over the duration of the study.

Submaximal constant load exercise test will be performed in the PFT lab at the Rockyview General Hospital on an electronically braked cycle ergometer. Subjects will warm up for 4min and then exercise at a predetermined workload equivalent to AT + 50%Δ where AT is the work rate corresponding to the pre-training ventilatory threshold and Δ is the difference between the work rates corresponding to the ventilatory threshold and VO_2max for that subject for 10minutes. In addition, fingertip blood samples will be collected at baseline, 5min, 10min and 5min post test. Each time a sample is drawn, approximately 5 μl of blood will be collected from your fingertip. Blood samples will be analyzed for lactate concentrations. Perceived leg exertion and shortness of breath will be measured at baseline, every 3min during test and at end of test using a modified CR-10 Borg scale. You will identify your perceived level of exertion on a scale from 0 – 10.

Inspiratory and expiratory muscle strength will be assessed as the maximal inspiratory and expiratory pressure (MIP & MEP) respectively measured at the mouth. Additionally, you will be required to swallow two balloon catheters in order to measure diaphragmatic strength. Inspiratory muscle endurance will be assessed by constant load breathing against a resistance equivalent to 50% of MIP until fatigue (CLRB). Termination of this test will be determined by the inability to maintain a pre-set breathing frequency. Diaphragmatic strength will be measured both during and before and after the submaximal test.

Inspiratory muscle training will occur over 4 weeks: 6-days/week, 3 sets/day, 30 breathes/set at a predetermined breathing frequency with 1-minute rest between sets. The first training session will occur in the lab so subject technique can be monitored. All the other training sessions will occur during the subject's own time at home.

Your time commitment to this study will result in about 8 hours in the laboratory over an eight week period (each visit ranging from 10 to 60 minutes in duration) and about 20-25 minutes of RMT on your own time. The VO_2max tests and submaximal constant load test will take place at the Pulmonary Function Lab, Rockyview General Hospital. All other tests will take place at the Human Performance Laboratory, University of Calgary. In addition, you will be asked to keep a detailed training log of your regular exercise training sessions beginning 2 weeks prior to the start of testing, as well as a training log of your inspiratory muscle training sessions. Therefore, your total time commitment to this study will be approximately 8.5 hours.

Your participation in this study also requires that you have fingertip blood samples taken on during submaximal constant load tests. On each occasion, you will provide 4 blood samples but no more than 400 µl (0.400ml) of blood will be collected. The first set of samples will be taken in the 1st week and the second set of samples will be taken in the 8th week. It is possible that there may be some tenderness or light bruising surrounding the area where the needle prick occurs. However any discoloration should clear within a few days of completing the study and subsequent needle pricks will not occur in areas where light bruising is present.

Risks: As with any maximal effort testing, you may experience some degree of muscle fatigue, nausea and light-headedness during the cycle testing sessions. During the insertion of the balloon catheters there is a possible risk of a perforated esophagus, sinus damage, and obstructed larynx, although the risk is minimal. Additionally there is the possibility of infection and minor residual pain after the removal of the catheter. If you experience any abnormal symptoms or any of the symptoms listed here, please report these to the technician administering the tests. However, this study should not leave you with any long-term adverse effects. You will be asked to remain in the lab for 5 to 20 minutes after the test is complete to be sure you have recovered appropriately.

Upon completion of the investigation, you will receive a detailed analysis of your personal results, which will identify you current fitness level, as well as your physiological strengths and weakness. Additionally, you may keep the respiratory muscle-training device if you wish.

Your personal results will be maintained in strict confidence, and will be revealed only to you and to the investigators involved in this study. All computer data will be password protected and paper documents kept in a filing cabinet in a locked office. All subjects will be assigned a code for data analysis. Once data is entered, all references to a particular patient's data will be by code number only. Participation in this project is voluntary and you reserve the right to withdraw at any time without prejudice. Data will be kept in locked storage for a period of 5 years and then destroyed. During storage, only the investigators and laboratory staff will have access to data.
In the event that you suffer injury as a result of participating in this research, no compensation will be provided to you by the sponsor, the investigator or the University of Calgary. The technician administering the tests is trained in emergency procedures,

and has appropriate qualification to be conducting the tests. You still have all your legal rights. Nothing said here about treatment or compensation in any way alters your right to recover damages.

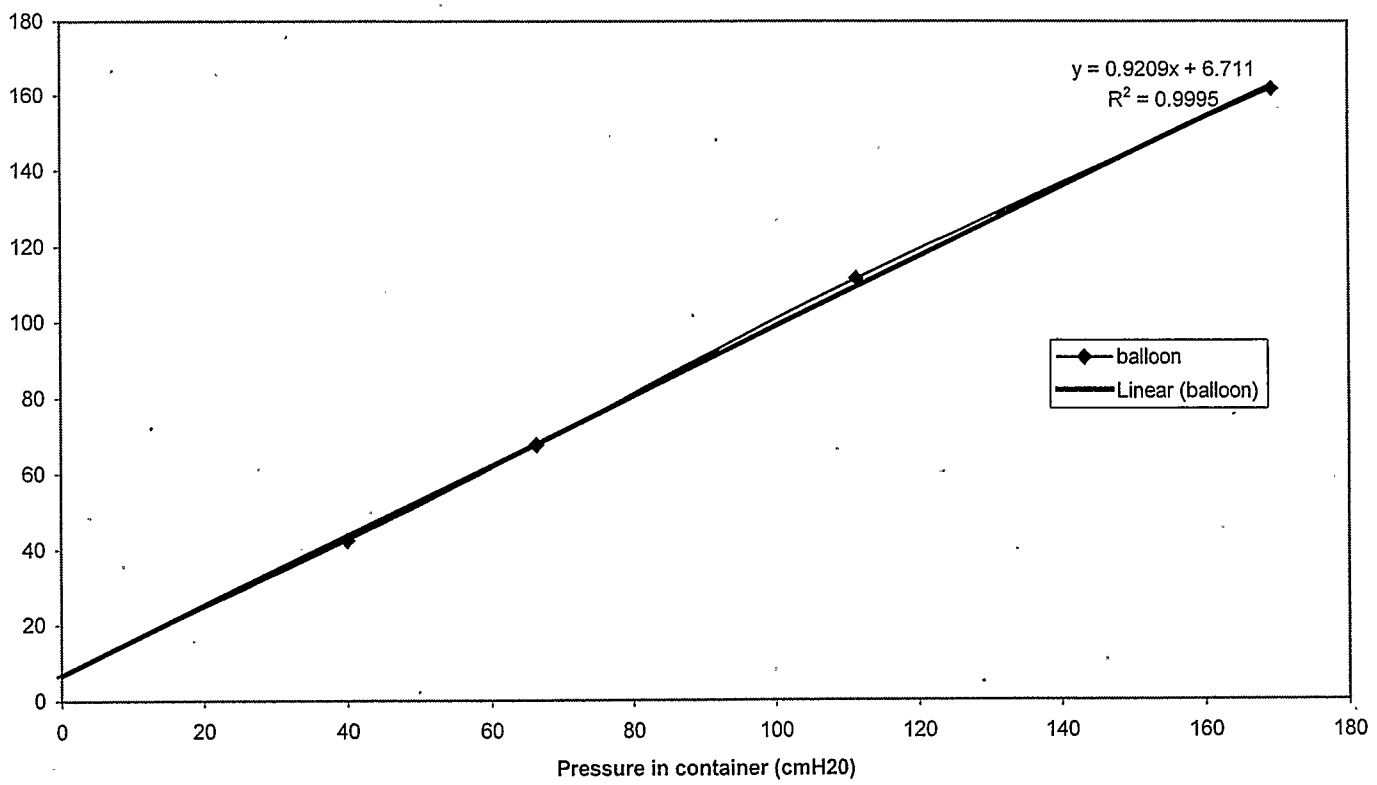
Your signature on this form indicates that you have understood to your satisfaction the information regarding participation in the research project and agree to participate as a subject. In no way does this waive your legal rights nor release the investigators, sponsors, or involved institutions from their legal and professional responsibilities. You are free to withdraw from the study at any time. Your continued participation should be as informed as your initial consent, so you should feel free to ask for clarification or new information throughout your participation. If you have further questions concerning matters related to this research, please contact: Dr. Victor Lun (supervisor) at: 220-8956 or Sidd Thakore (student researcher) at: 220-8949.

If you have any questions concerning your rights as a possible participant in this research, please contact Pat Evans, Associate Director, Internal Awards, Research Services, University of Calgary, at 220-3782."

Participant's Signature	Date
Investigators and/or Delegate's Signature	Date
Witness' Signature	Date

A copy of this form has been given to you for your personal files.

Appendix E: Pressure Volume Curve for Balloon Catheter system with 10ml air inserted into balloon.



Appendix F: Regular physical activity training log

RMT STUDY EXERCISE TRAINING JOURNAL

LAST NAME:_____ FIRST NAME:_____ SUBJECT NO:_____

MONTH OF ENROLLMENT (Please circle): 1 2 3 4 5 6 7 8 9 10 11 12 DATE OF DAY 1: _____

Please indicate the type of training performed under training modality (ie. cycling, swimming). Please indicate the total training duration (if you did more than one training modality, indicate total training duration). Please indicate intensity level of training (Heart Rate if known, and low/medium/high level)
Date of day one refers to the day you begin recording information (two weeks prior to first testing day ie. Feb 1)

Day	Training Modality	Training Duration	Training Intensity (HR)&(Low/Med/High)	Day	Training Modality	Training Duration	Training Intensity (HR)&(Low/Med/High)
1				17			
2				18			
3				19			
4				20			
5				21			
6				22			
7				23			
8				24			
9				25			
10				26			
11				27			
12				28			
13				29			
14				30			
15				31			
16							

If you have any questions, please call Sidd at 220-8949 or email at sthakore@ucalgary.ca.

Appendix G: RMT Log

RMT STUDY SUBJECT JOURNAL

LAST NAME:_____ FIRST NAME:_____ SUBJECT NO:_____

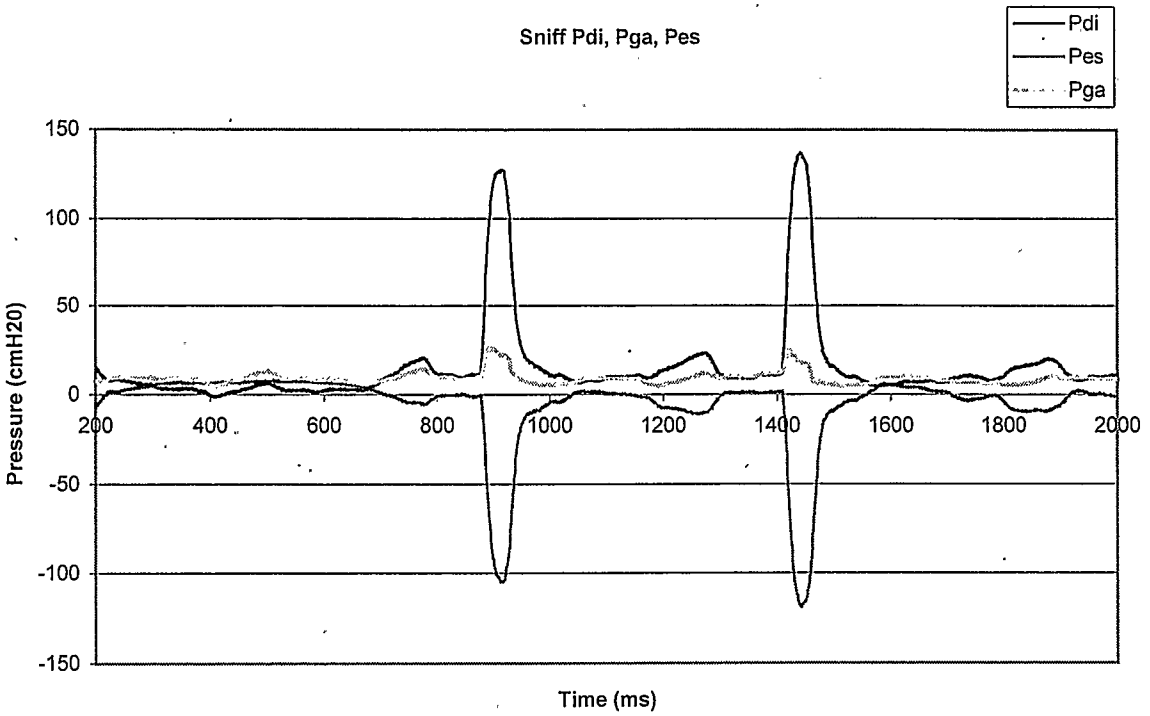
MONTH OF ENROLLMENT (Please circle): 1 2 3 4 5 6 7 8 9 10 11 12 DATE OF DAY 1: _____

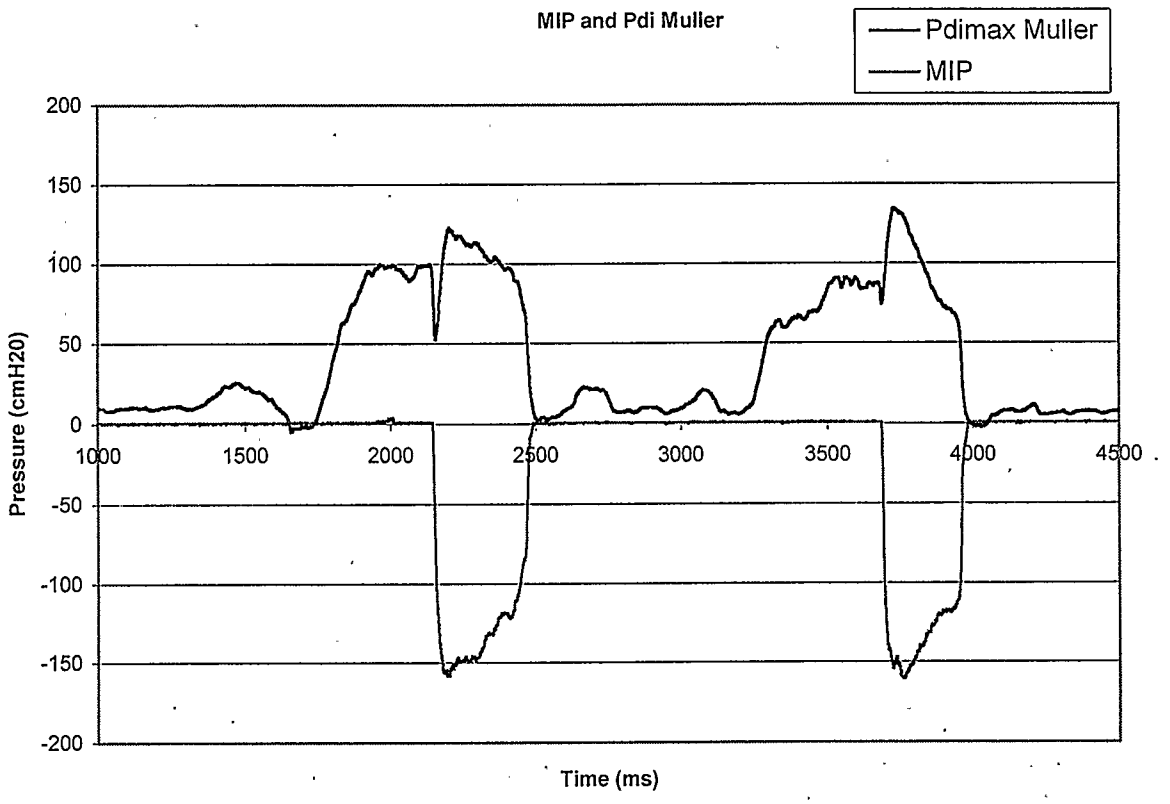
Please indicate with a check the days that you did your training. Also note if you encountered any problems during training.

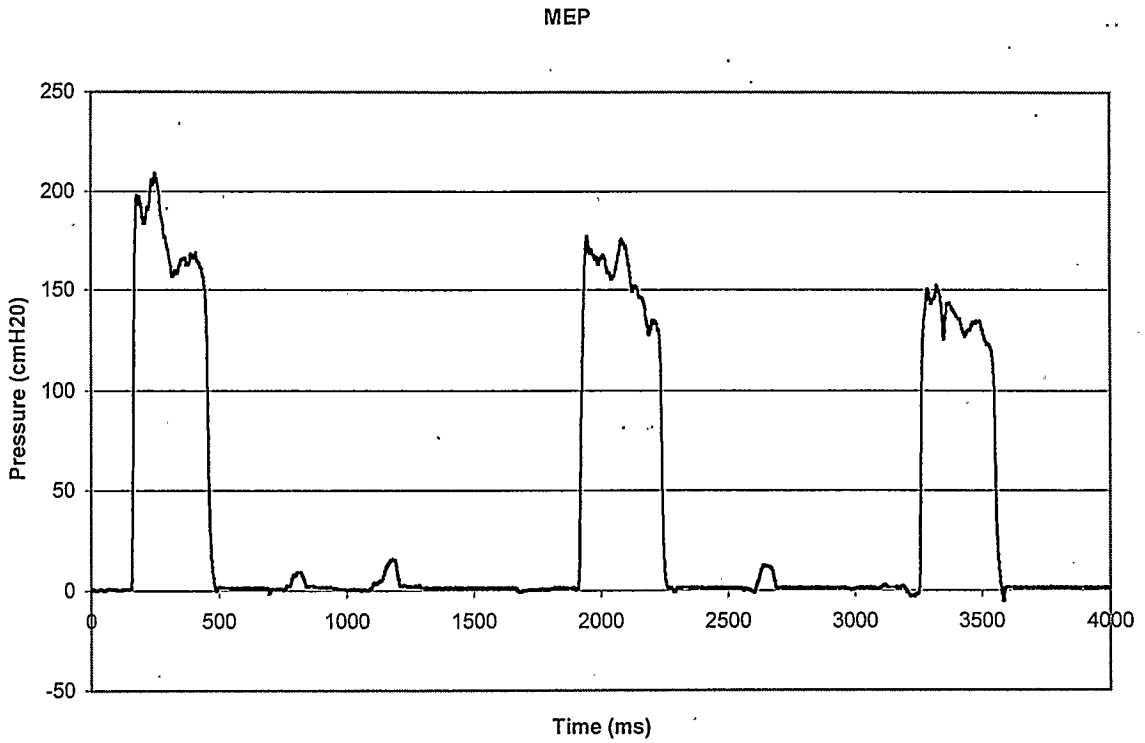
Day	Training	Complications	Day	Training	Complications
1			17		
2			18		
3			19		
4			20		
5			21		
6			22		
7			23		
8			24		
9			25		
10			26		
11			27		
12			28		
13			29		
14			30		
15			31		
16					

If you have any questions, please call Sidd at 220-8949 or email at sthakore@ucalgary.ca.
Training 6 days/week, 3 sets of 30 reps/ day, each set is performed at a breathing frequency of 35bpm using your entire lung volume. Therefore each set should take just under 1minute

Appendix H: Tracing of Subject # 11's MIP, MEP, Pdimax, Sniff Pdi and Sniff Pes






MEP
250
200
150
100
50
0
-50
Pressure (cmH20)
0
500
1000
1500
2000
2500
3000
3500
4000
Time (ms)